City of Milwaukee

City Hall
200 East Wells Street
Milwaukee, WI 53202

Meeting Agenda

STEERING & RULES COMMITTEE

ALD. WILLIE L. HINES, Jr., CHAIR
Ald. Michael J. Murphy, Vice-Chair
Ald. Joe Davis, Sr., Ald. Ashanti Hamilton, Ald. James Bohl, Ald. Robert Bauman, Ald. Terry Witkowski, and Ald. T. Anthony Zielinski
Staff Assistant, Tobie Black, 286-2231; Fax: 286-3456, tblack@milwaukee.gov
Legislative Liaison, Richard Watt, 286-2253, rwatt@milwaukee.gov

Thursday, May 31, 2012 | **1:30 PM** | **Room 301-B, City Hall**

1. 120187 Resolution ordering the immediate cessation and prohibition of the introduction of sodium silicofluoride or any fluoride compound into water distributed by the Milwaukee Water Works.

Sponsors: Ald. Bohl

2. 120188 Resolution authorizing the transfer and expenditure of funds within the Milwaukee Water Works to create an oral health program for medically-underserved children.

Sponsors: Ald. Bohl

This meeting will be webcast live at www.milwaukee.gov/channel25.

Common Council members who are not members of this committee may attend this meeting to participate or to gather information. This meeting may constitute a meeting of the Common Council or any of its standing committees although no formal action will be taken at this meeting.

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Legislation Details (With Text)

File #:	120187 **Version:** 0		
Type:	Resolution	**Status:**	In Committee
File created:	5/22/2012	**In control:**	STEERING & RULES COMMITTEE
On agenda:		**Final action:**	
Effective date:			
Title:	Resolution ordering the immediate cessation and prohibition of the introduction of sodium silicofluoride or any fluoride compound into water distributed by the Milwaukee Water Works.		
Sponsors:	ALD. BOHL		
Indexes:			
Attachments:	Community Flouride Study Part 1, Community Flouride Study Part 2, Community Flouride Study Part 3, Community Flouride Study Part 4, Letter from Wisconsin Dental Association, Tooth Decay Trends in Flouridated Vs Unflouridated Countries, Excitotoxicity: A Possible Central Mechanism in Flouride Neurotoxicity, Building a Database of Developmental Neurotoxicants, Association of Vascular Flouride Uptake with Vascular Calcification and Coronary Artery Disease, Caries and Flurosis Prevalence in Communities with Different Concentrations of Fluoride in the Water, Dental Flourosis Decline After Changes to Supplement and Toothpaste Regimes, Hearing Notice List, Flouride Supplementation for Children- Interim Policy Recommendations, May 27 2012 Press Release from Ald Bohl, May 22 Press Statement from Ald Bohl, National Institute of Health Flouride Study Abstract, Hearing Notice List		

Date	Ver.	Action By	Action	Result	Tally
5/22/2012	0	COMMON COUNCIL	ASSIGNED TO		
5/29/2012	0	STEERING & RULES COMMITTEE	HEARING NOTICES SENT		
5/29/2012	0	STEERING & RULES COMMITTEE	HEARING NOTICES SENT		
5/29/2012	0	STEERING & RULES COMMITTEE	HEARING NOTICES SENT		

Number
120187
Version
ORIGINAL
Reference

Sponsor
ALD. BOHL
Title
Resolution ordering the immediate cessation and prohibition of the introduction of sodium silicofluoride or any fluoride compound into water distributed by the Milwaukee Water Works.
Analysis
This resolution orders the Milwaukee Water Works to terminate and prohibit the introduction of sodium silicofluoride, or any fluoride compound, into water distributed by the Milwaukee Water Works.
Body
Whereas, The Centers for Disease Control and Prevention has now acknowledged the findings of many leading dental researchers that the mechanism of fluoride's main benefits are derived from

surface application to teeth and not from ingestion; and

Whereas, Despite being prescribed by doctors for over 50 years, the U.S. Food and Drug Administration (FDA) has never approved any fluoride designed for ingestion as safe and effective; and

Whereas, Drug products on the market prior to 1938 were presumed safe by the FDA and grandfathered to be sold without testing; and

Whereas, Fluoride's grandfathered federal status came as a result of its use in the market as a rat poison; and

Whereas, Fluoride is a cumulative poison, and 50% of the fluoride ingested each day is excreted through the kidneys and the remainder accumulates in bones, pineal gland and other tissues; and

Whereas, If the kidney is damaged, fluoride accumulation in a body will increase; and

Whereas, Growing evidence links fluoridated water with increased lead uptake; and

Whereas, Fluorosilicic acids bind with lead, leaching high levels of soluble lead from lead water pipes, delivering a compounded dose of toxic lead with toxic fluoride to Milwaukee water drinkers; and

Whereas, Once fluoride is put in the water supply it is impossible to control each dose an individual receives as fluoride is found in sources other than the water supply; and

Whereas, Other sources of fluoride include food and beverages processed with fluoridated water, fluoridated dental products, mechanically deboned meat, teas and pesticide residue on food; and

Whereas, A comparison of results from 24 studies of un-fluoridated districts in 8 countries revealed the reduction in dental caries are just as great in non-fluoridated areas as fluoridated; and

Whereas, The chemicals used to fluoridate water in the United States are not pharmaceutical-grade but instead come from wet scrubbing systems of the super-phosphate fertilizer industry; and

Whereas, These chemicals are classified as hazardous wastes contaminated with various impurities as recent testing by the National Sanitation Foundation suggested that the levels of arsenic in these chemicals are relatively high; and

Whereas, In 1953, the Common Council voted to add fluoride to the water supply at a time when Milwaukee had a serious problem of tooth decay and fluoridated tooth pastes, gels or mouthwash were not readily available, as they are today; and

Whereas, Some of the earliest opponents of fluoridation were biochemists, and at least 14 Nobel Prize winners are among numerous scientists who have expressed their reservations about the practice of fluoridation; and

Whereas, The American Dental Association and a number of dental researchers recommend that children under 12 months of age should not consume fluoridated water and that babies under 6

months of age should not receive any fluoride drops or pills due to an increased risk of dental fluorosis, a sign of overexposure to fluoride, and a lack of demonstrable benefit from ingesting fluoride before teeth erupt; and

Whereas, In January 2011, the U.S. Department of Health and Human Services reported that 41% of adolescents in the United States have dental fluorosis; and

Whereas, Children are being over-exposed to fluoride with the highest doses going to bottle-fed babies as infant formula is used with fluoridated tap water; and

Whereas, Minorities are more likely to use infant formula with fluoridated tap water, resulting in minority children ingesting significantly more fluorides and having higher rates of dental fluorisis; and

Whereas, Andrew Young, Alveda and Bernice King, Rev. W. Owens of the Coalition of African American Pastors, and League of United Latin American Citizens (LULAC) are among the growing number of fluoride opponents; and

Whereas, Fluoridation is unethical as individuals are not asked for their informed consent prior to medication, as is standard practice for the administration of all other medications; and

Whereas, Only through the total removal of all fluoridation from Milwaukee's public water supply can all residents be protected from the possible adverse health effects; now, therefore, be it

Resolved, By the Common Council of the City of Milwaukee, in its continuing effort to promote the health, safety and welfare of all, orders the immediate cessation and prohibition of the introduction of sodium silicofluoride, or any fluoride compound, into water distributed by the Milwaukee Water Works.

Requestor

Drafter
LRB137347-1
Amy E. Hefter
2/23/2012

Fluoride:
Poison in Our Water

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CITY HISTORY AND LEGISLATION

Town of Lake, State of Wisconsin, and that, as provided in Chapter 275 of the Laws of Wisconsin, 1931, and the amendments thereto, a plan of said proposed improvement be prepared, together with a map and description of the property proposed to be taken or used or that may be benefited by said improvement, with an estimate of the total cost of completing said improvement, and with an estimate of the total benefits, if any, that may be assessed against the property benefited, if any, by the improvement and that a report thereof be made to the Common Council and be it

Further Resolved, That the City Real Estate Agent is hereby directed not to acquire any property involved in this project until such time as the report and plan of the board of assessment is adopted by the Common Council.

Referred to the Committee on Buildings-Grounds-Harbors and Board of Public Land Commissioners.

By ALD. MORTIER, KUJAWA, SULKOWSKI, HANSEN, ZILLMAN, HOFFMANN, SCHMIDT, KEPPLER, FASS, FROEMMING and TOMCZYK—

FILE NUMBER 52-1221

Resolution relative to commencing the introduction of sodium silicofluoride into the city water system.

Whereas, Pursuant to Resolution File No. 48-1922-a, adopted October 24, 1950, the Milwaukee Water Works has taken all steps necessary to introduce fluorine into the water it distributes, has a stock of the necessary chemicals on hand, and is prepared to commence fluoridation; now, therefore, be it

Resolved, By the Common Council of the City of Milwaukee, that the proper city officers be and they hereby are directed to commence the introduction of sodium silicofluoride into water distributed by the Milwaukee Water Works in sufficient quantities to bring the fluorine content of the water to a concentration of approximately one part per million.

Referred to the Joint Committee on Public Utilities and Health-Traffic.

By ALD. HEIDEN—

FILE NUMBER 52-1222

Resolution to set up funds in 1953 budget to remedy inadequate sewer conditions on W. Center Street from N. 87th Street to N. Menomonee River Parkway.

Whereas, The area from approximately N. 87th Street to N. Menomonee River Parkway on W. Center Street is now without the proper sewer facilities to carry off water due to even a slight rainfall, causing basements to be flooded and great inconvenience to the residents of that area; therefore be it

Resolved, By the Common Council of the City of Milwaukee, that the Commissioner of Public Works be and hereby is instructed to see that sufficient funds are set up in the 1953 budget so that the work can be started in 1953 to remedy the inadequacy of the sewer facilities.

Referred to the Committee on Streets-Expressways.

By unanimous consent, ALD. HEIDEN (By request) at this time presented the following:

FILE NUMBER 52-1223

Petition of Edward J. Russell, et al., for vacation of north and south alley between W. Concordia and W. Auer Avenues, N. 94th and N. 95th Streets, in the 26th Ward of the City of Milwaukee.

By ALD. HEIDEN (By request)

FILE NUMBER 52-1223

Resolution to vacate the north and south alley in block bounded by W. Concordia Avenue, N. 94th Street, W. Auer Avenue and N. 95th Street, in the 26th Ward.

Whereas, A petition was presented to the Common Council of the City of Milwaukee by Edward J. Russell and others, on the 15th day of July, 1952, for the vacation of a portion of alley.

Resolved, By the Common Council of the City of Milwaukee that said petition be and the same hereby is granted and that the following described portion of alley to-wit:

"Commencing at a point in the northwest corner of Lot one (1) in Block sixteen (16), Colonial Highlands being a subdivision of a part of the south one-half (S. ½) of Section eight (8), Township seven (7) north, Range twenty-one (21) east; running thence south along the west line of Lots one (1) to twelve (12) both inclusive in Block sixteen (16) of said subdivision, six hundred and no one-hundredths (600.00) feet to a point in the southwest corner of said Lot twelve (12), said point also lying in the north line of West Auer Avenue; thence west along the north line of West Auer Avenue extended twenty and no one-hundredths (20.00) feet to a point in the southeast corner of Lot thirteen (13) in said Block sixteen (16); thence north along the east line of Lots thirteen (13) to twenty-two (22) both inclusive in Block sixteen (16) aforesaid six hundred and no one-hundredth (600.00) feet to a point in the northeast corner of said Lot twenty-two (22), said point also lying in the south line of West Concordia Avenue; thence east along the south line of West Concordia Avenue extended twenty and no one-hundredths (20.00) feet to the point of commencement."

FILE NUMBER 53-498

To improve W. Hopkins Street from N. 17th Street to N. 27th Street and parts of abutting streets in the 20th Ward of the City of Milwaukee.

(As printed in full in proceedings of May 19, 1953, pages 345-346.) *Adopted.*

FILE NUMBER 53-499

To improve W. Idaho St. from S. 61st St. to W. Manitoba St. in the 24th ward of the City of Milwaukee.

(As printed in full in proceedings of May 19, 1953, page 346.) *Adopted.*

FILE NUMBER 53-500

To improve Pedestrian Way betw. S. 62nd St. & S. 65th St., W. Bennett Ave. to 110′ ± North in the 24th Ward of the City of Milwaukee.

(As printed in full in proceedings of May 19, 1953, page 346.) *Adopted.*

FILE NUMBER 53-501

To improve S. 55th St. from a Pt. South of W. Euclid Ave. to W. Oklahoma Ave. in the 24th Ward of the City of Milwaukee.

(As printed in full in proceedings of May 19, 1953, pages 346-347.) *Adopted.*

FILE NUMBER 53-502

To improve S. 56th St. from a Pt. South of W. Euclid Ave. to W. Oklahoma Ave. in the 24th Ward of the City of Milwaukee.

(As printed in full in proceedings of May 19, 1953, page 347.) *Adopted.*

FILE NUMBER 53-503

To improve S. 60th Street from a point south of W. Warnimont Avenue to W. Euclid Avenue (Extd.) in the 24th Ward of the City of Milwaukee.

(As printed in full in proceedings of May 19, 1953, pages 347-348.) *Adopted.*

FILE NUMBER 53-504

To improve S. 61st Street from W. Bennett Avenue to W. Idaho Street in the 24th Ward of the City of Milwaukee.

(As printed in full in proceedings of May 19, 1953, page 348.) *Adopted.*

FILE NUMBER 53-505

To improve S. 62nd Street & W. Manitoba Street from W. Bennett Avenue to W. Idaho Street in the 24th Ward of the City of Milwaukee.

(As printed in full in proceedings of May 19, 1953, page 348.) *Adopted.*

FILE NUMBER 53-506

To improve W. Concordia Avenue from N. 92nd Street to N. 96th Street in the 26th Ward of the City of Milwaukee.

(As printed in full in proceedings of May 19, 1953, pages 348-349.) *Adopted.*

FILE NUMBER 53-507

To improve N. 51st Boulevard from W. Fond du Lac Avenue to W. Congress Street in the 26th Ward of the City of Milwaukee.

(As printed in full in proceedings of May 19, 1953, page 349.) .. *Adopted.*

FILE NUMBER 53-508

To improve N. 93rd Street from W. Burleigh Street to W. Townsend Street in the 26th Ward of the City of Milwaukee.

(As printed in full in proceedings of May 19, 1953, page 349.) *Adopted.*

FILE NUMBER 53-767

All the foregoing reports of committees and resolutions favorably reported upon, on which no separate action had been demanded, and to which no objection had been made and designated by the word "adopted" were adopted by the following vote:

Ayes:—Ald. Choinski, Collins, Fass, Fleming, Froemming, Gromacki, Hansen, Hass, Heiden, Hoffmann, Jendusa, Kelly, Keppler, Kroenke, Kujawa, LaBelle, Meyers, Mortier, Quirk, Schimenz, Schmidt, Schreiber, Sulkowski, Tomczyk, Whittow, Zillman and the *President*—27. *Noes:—0*

ALD. MORTIER moved that

FILE NUMBER 52-1221

Resolution relative to commencing the introduction of sodium silico-fluoride into the city water system.

be recalled from the Joint Committee on Public Utilities and Health-Traffic and brought before the Council for action at this time.

The motion prevailed by the following vote:

Ayes:—Ald. Collins, Fass, Fleming, Froemming, Hansen, Hoffmann, Jendusa, Keppler, Kujawa, Mortier, Schmidt, Schreiber, Sulkowski, Tomczyk, Whittow and Zillman—16.

Noes:—Ald. Choinski, Gromacki, Hass, Heiden, Kelly, Kroenke, LaBelle, Meyers, Quirk, Schimenz and the President—11.

ALD. MORTIER moved that the foregoing resolution be adopted.

The motion prevailed by the following vote:

Ayes:—Ald. Collins, Fass, Froemming, Hansen, Heiden, Hoffmann, Jendusa, Keppler, Kujawa, Mortier, Quirk, Schmidt, Schreiber, Sulkowski, Tomczyk, Whittow and Zillman—17.

Noes:—Ald. Choinski, Fleming, Gromacki, Hass, Kelly, Kroenke, LaBelle, Meyers, Schimenz and the President—10.

ALD. QUIRK gave notice at this time that at the next meeting of the Common Council he would move a reconsideration of the vote by which the foregoing resolution was adopted.

The Clerk was instructed to spread said notice upon the Journal.

ALD. QUIRK at this time presented the following:

By ALD. QUIRK—

FILE NUMBER 52-1221-d

Resolution requesting the President of the Common Council to appoint a special committee of five or more to investigate fluoridation of water in other communities.

Resolved, By the Common Council of the City of Milwaukee, that the President of the Common Council be and hereby is requested to appoint a special committee of five or more, to investigate the fluoridation of water in other communities and that sufficient funds be allocated for this purpose.

Referred to the Committee on Finance-Printing.

COMMUNICATIONS FROM CITY OFFICERS

THE CHAIR presented the following communications:

FROM THE OFFICE OF THE MAYOR

FILE NUMBER 52-3603

Milwaukee, June 9, 1953.

To the Honorable, the Common Council:

Gentlemen: I am herewith returning with my veto Common Council Resolution Number 52-3603 concerning a request for a driveway for the International Trading Company on N. 27th Street between W. Center Street and W. Silver Spring Drive.

The original request was for 100 feet and the substitute resolution has reduced this to 60 feet. In the opinion of the Commissioner of Public Works 30 feet would be adequate. The public works department has received many requests for driveways larger than 30 feet. In my opinion the pedestrian safety is not served by these larger driveways and I therefore respectfully request you to reconsider this resolution.

This resolution must also be considered in connection with Resolution Number 52-3604, which is a resolution to install a 51 foot driveway on property adjacent to that covered by Resolution Number 52-3603. A substitute introduced by the Common Council would permit a 40 foot driveway. Together, the two driveways, in Resolution Number 52-3603 and Resolution Number 52-3604, would produce a 100 foot driveway which, in my opinion, is excessive. If both requests were reduced to 30 foot driveways and were placed together they would still result in 60 feet of driveway. This, in my opinion, is ample and will not establish a bad precedent.

Respectfully yours,

FRANK P. ZEIDLER, Mayor.

ALD. FASS moved that the further action on the foregoing matter be deferred until the next regular meeting.

The motion prevailed unanimously.

FILE NUMBER 52-3604

Milwaukee, June 9, 1953.

To the Honorable, the Common Council:

Gentlemen: I am herewith returning Resolution File Number 52-3604 with my veto. The reasons for this veto are stated in a message which I am sending with Resolution Number 52-3603 in returning that resolution also with a veto. The message is appended herewith.

Respectfully yours,

FRANK P. ZEIDLER, Mayor.

ALD. FASS moved that the further action on the foregoing matter be deferred until the next regular meeting.

The motion prevailed unanimously.

FILE NUMBER 52-3664

Milwaukee, June 8, 1953.

To the Honorable, the Common Council:

Gentlemen: I am returning herewith, without my signature, Common Council Ordinance, File Number 52-3664, which permits 40-foot buses to operate on city streets.

In not affixing my signature to this ordinance, I am supporting the view that these buses will do excessive damage to the streets, and that they will tend to obstruct two lanes of traffic at stopping places.

I do not regard bigger buses as necessarily reflecting progress in public transit. Without our ex-

COMMON COUNCIL

CITY OF MILWAUKEE

JOURNAL OF PROCEEDINGS

Regular Meeting, Tuesday, June 30, 1953, 2:00 o'clock P.M.

FILE NUMBER 53-966

Common Council Regular Meeting, Tuesday, June 30, 1953 (2:00 P.M.).

PRESIDENT McGUIRE IN THE CHAIR

Present:—Ald. Choinski, Collins, Fass, Fleming, Froemming, Gromacki, Hansen, Hass, Heiden, Hoffmann, Jendusa, Kelly, Keppler, Kroenke, Kujawa, LaBelle, Meyers, Mortier, Quirk, Schimenz, Schmidt, Schreiber, Sulkowski, Tomczyk, Whittow, Zillman and the President—27.

By unanimous consent, ALD. LaBELLE at this time presented the following:

FILE NUMBER 53-967

Whereas, Miss Daisie E. Allen, Common Council Committee Clerk, is retiring after 46 years of service with the City of Milwaukee; and,

Whereas, Miss Allen started as a stenographer and clerk on February 19, 1907, in the Health Department; in 1912 the Commissioner of Health, Dr. Gerhard A. Bading, was elected mayor and she moved with him to the mayor's office; she became executive secretary to the mayor in 1915; in 1916 she went to work in the Inspector of Buildings office; and, on March 1, 1917, she transferred to the City Clerk's office and has been there ever since; and,

Whereas, Miss Allen has been a strong "right arm" for various city clerks and scores of aldermen, department heads went to her for advice on how to put into proper form documents that had to be prepared for introduction into the Common Council, and she has written thousands of ordinances and resolutions; and,

Whereas, Members of this Honorable Body wish to express their feelings on this memorable occasion to this lady of gentle manner with a whimsical sense of humor and twinkling eyes and an acute mind; therefore, be it

Resolved, By the Common Council of the City of Milwaukee, that it hereby congratulates Miss Daisie E. Allen, Common Council Committee Clerk, on her retirement after 46 years of service and hopes her plans for the future will materialize; and be it

Further Resolved, That this resolution be spread upon the permanent records of this Council and a suitably engrossed copy be forwarded to Miss Allen.

Upon motion the rules were suspended and the resolution adopted.

CORRECTIONS OF THE JOURNAL

ALD. SCHIMENZ moved to correct the Journal of the Proceedings of the regular meeting held June 15, 1953, at page 560, first column, by striking out "File Number 58653" in the second printed line from the top of said page, in said column, and inserting "File Number 58653-a;" further at page 656, first column, by striking out "File Number 53-1873" in the tenth printed line from the top of said page, in said column, and inserting "File Number 53-873."

The motion prevailed.

The Journal of the Proceedings of the regular meeting held June 16, 1953, as corrected, and of the special meeting held June 23, 1953, were thereupon approved.

UNFINISHED BUSINESS

Pursuant to notice given at the last regular meeting of the Common Council, ALD. QUIRK at this time moved a reconsideration of the vote by which

FILE NUMBER 52-1221

Resolution relative to commencing the introduction of sodium silicofluoride into the city water system was adopted.

The motion was lost by the following vote:

Ayes:—Ald. Choinski, Fleming, Gromacki, Hass, Heiden, Kelly, Kroenke, LaBelle, Meyers, Quirk, Schimenz, Whittow and the President—13.

Noes:—Ald. Collins, Fass, Froemming, Hansen, Hoffmann, Jendusa, Keppler, Kujawa, Mortier, Schmidt, Schreiber, Sulkowski, Tomczyk, and Zillman—14.

By unanimous consent, ALD. KELLY at this time presented the following:

FILE NUMBER 52-1221-e

Resolution to restrain the Superintendent of the Water Department from introducing fluorides into the city water until Milwaukee County Medical Society reports on merits of the same.

Resolved, By the Common Council of the City of Milwaukee, that the Superintendent of Water Department be restrained from introducing fluorides into the city water until we have the answer of the

Milwaukee County Medical Society on the merits of the issue, the same to be taken up in executive session by the Milwaukee County Medical Society.

ALD. KELLY moved that all rules interfering with the adoption of the foregoing resolution at this time be suspended.

The motion was lost by the following vote (a two-thirds vote being required):

Ayes:—14. Noes:—13.

Ald. Choinski, Fleming, Froemming, Gromacki, Hass, Heiden, Jendusa, Kelly, Kroenke, LaBelle, Meyers, Quirk, Schimenz and the President voting aye.

ALD. QUIRK moved that the foregoing resolution be referred to the Committee on Public Utilities.

The motion prevailed.

FILE NUMBER 52-3603

Veto of His Honor the Mayor of File Number 52-3603, being a resolution authorizing granting of permit to International Trading Co. for two 100 ft. driveways, one on N. 27th Street between W. Custer Avenue and W. Silver Spring Drive and one on W. Custer Avenue between N. Teutonia Avenue and N. 27th Street (as substituted and printed in the Proceedings of June 2, 1953, at page 452)

laid over from the last regular meeting, was taken up.

THE CHAIR put the question: "Shall the foregoing resolution, as substituted, be adopted notwithstanding the objections of His Honor the Mayor?"

The foregoing resolution, as substituted, thereupon failed of adoption by the following vote:

Ayes:—0.

Noes:—Ald. Choinski, Collins, Fass, Fleming, Froemming, Gromacki, Hansen, Hass, Heiden, Hoffmann, Jendusa, Kelly, Keppler, Kroenke, Kujawa, LaBelle, Meyers, Mortier, Quirk, Schimenz, Schmidt, Schreiber, Sulkowski, Tomczyk, Whittow, Zillman and the President—27.

FILE NUMBER 52-3604

Veto of His Honor the Mayor of File Number 52-3604, being a resolution authorizing granting of permit to Rubin Lakam to install 51 foot driveway on N. 27th Street between W. Custer Avenue and W. Silver Spring Drive (as substituted and printed in the Proceedings of June 2, 1953, at page 452)

laid over from the last regular meeting, was taken up.

THE CHAIR put the question: "Shall the foregoing resolution, as substituted, be adopted notwithstanding the objections of His Honor the Mayor?"

The foregoing resolution, as substituted, thereupon failed of adoption by the following vote:

Ayes:—0.

Noes:—Ald. Choinski, Collins, Fass, Fleming, Froemming, Gromacki, Hansen, Hass, Heiden, Hoffmann, Jendusa, Kelly, Keppler, Kroenke, Kujawa, LaBelle, Meyers, Mortier, Quirk, Schimenz, Schmidt, Schreiber, Sulkowski, Tomczyk, Whittow, Zillman and the President—27.

FILE NUMBER 53-461

The matter of the revocation of Class "B" intoxicating liquor license of John Handzlik, laid over from the last regular meeting, was taken up.

The city being represented by Alan H. Steinmetz, Assistant City Attorney.

The city presented the complaint in the above matter, together with the summons and return thereon of Police Sergeant Paul Reardon.

Deputy Inspector Miller, being first duly sworn, testified in behalf of the city.

THE CHAIR presented a resolution to revoke the Class "B" intoxicating liquor license of John Handzlik.

ALD. GROMACKI moved that the foregoing resolution be placed on file. *The motion prevailed.*

PRESENTATION OF ORDINANCES

By ALD. HASS—

FILE NUMBER 52-1483-c

AN ORDINANCE

To amend Section 105-20.5 of the Milwaukee Code of Ordinances relating to ball playing.

The Mayor and Common Council of the City of Milwaukee do ordain as follows:

Part 1. Section 105-20.5 of the Milwaukee Code relating to prohibition of ball playing in certain areas is hereby amended by deleting therefrom the following:

Under the caption, "The playing of hard baseball and softball shall be prohibited on the play areas and practice fields of the following secondary schools, excepting only as the respective pupils at such schools may play such games during school hours on school days, and on Saturdays and vacation days, but only under the direction of a teacher or some other person designated by the principals of the respective schools:", delete "Girls' Junior Trade, 414 W. Garfield Avenue."

Under the caption, "The playing of twelve inch softball shall be permitted on the following play fields, playgrounds and other named premises:", the

FILE NUMBER 53-1133

Request of Milwaukee Downtown Y's Men's Club to rent Borchert Field for use by the Mills Bros. Circus.

by recommending that they be placed on file.

Reports adopted and matters ordered on file.

ALD. FLEMING—

From the Committee on Public Utilities reported upon:

FILE NUMBER 52-1221-e

Resolution to restrain the Superintendent of the Water Department from introducing fluorides into the city water until Milwaukee County Medical Society reports on merits of the same. (Page 683.)

by recommending the adoption of the following substitute resolution:

FILE NUMBER 52-1221-e

Resolved, By the Common Council of the City of Milwaukee, that the Superintendent of Water Department be restrained from introducing fluorides into the city water until we have the answer of the Milwaukee County Medical Society on the merits of the issue.

ALD. MORTIER moved separate action on the foregoing matter. *The motion prevailed.*

ALD. MORTIER moved that the foregoing matter be re-referred to the Committee.

The motion lost by the following vote:

Ayes:—Ald. Collins, Fass, Hansen, Hoffmann, Keppler, Mortier, Schmidt, Schreiber, Sulkowski, Tomcyzk and Zillman—11.

Noes—Ald. Choinski, Fleming, Gromacki, Hass, Heiden, Jendusa, Kelly, Kroenke, Kujawa, LaBelle, Meyers, Quirk, Schimenz, Whittow and the President—15.

ALD. MORTIER moved that the foregoing matter be laid over to the next regular meeting.

The motion prevailed by the following vote (six votes only being necessary):

Ayes: Ald. Collins, Fass, Hansen, Hoffmann, Keppler, Mortier, Schmidt, Sulkowski, Tomczyk and Zillman—10.

Noes:—Ald. Choinski, Fleming, Gromacki, Hass, Heiden, Jendusa, Kelly, Kroenke, Kujawa, LaBelle, Meyers, Quirk, Schimenz, Schreiber, Whittow and the President—16.

ALD. KROENKE moved that the Superintendent of Water Works be requested not to fluoridate the city water supply for the next two weeks or until information is received from the Milwaukee County Medical Society in accordance with the foregoing resolution.

The motion prevailed by the following vote:

Ayes:—Ald. Choinski, Fleming, Gromacki, Hass, Heiden, Jendusa, Kelly, Kroenke, Kujawa, LaBelle, Meyers, Quirk, Schimenz, Whittow and the President —15.

Noes:—Ald. Collins, Hass, Hansen, Hoffmann, Keppler, Mortier, Schmidt, Schreiber, Sulkowski, Tomcyzk and Zillman—11.

ALD. FLEMING—

From the Committee on Public Utilities reported upon:

FILE NUMBER 53-1083

Resolution directing a survey as to medical and dental aspect of fluoridation to the City of Milwaukee. (Page 791.)

by recommending the adoption of the following substitute resolution:

FILE NUMBER 53-1083

Resolved, By the Common Council of the City of Milwaukee, that the Municipal Reference Librarian of the City of Milwaukee be and hereby is instructed to secure the official membership list of the Milwaukee County Medical Association; and be

Further Resolved, That the Municipal Reference Librarian is hereby instructed to prepare a questionnaire to be submitted to this membership to read as follows:

1. Does sufficient research exist to warrant the belief that the introduction of sodium silico fluoride into drinking water will benefit the teeth of children from six to nine?
2. Does sufficient research exist to prove that fluorides do not have a deleterious effect on the soft tissues of the body and on bone structure?
3. Does sufficient research exist to prove that fluorides do not do harm to the sick and aged?
4. Does sufficient research exist to show whether fluorides accumulate in the body?
5. In your opinion does the fluoridation constitute medication?
6. Will fluorides of drinking water added in the amount of one part per million cause mottling of teeth?
7. Will drinking water to which fluoride compounds have been added have any effects different from water containing fluorine naturally?

(Regular Meeting, July 28, 1953, and Special Meeting, July 30, 1953)

COMMON COUNCIL

CITY OF MILWAUKEE

JOURNAL OF PROCEEDINGS

Regular Meeting, Tuesday, July 28, 1953, 2:00 o'clock P.M.

FILE NUMBER 53-1328

Common Council Regular Meeting, Tuesday, July 28, 1953 (2:00 P.M.)

PRESIDENT McGUIRE IN THE CHAIR

Present:—Ald. Choinski, Collins, Fass, Fleming, Froemming, Gromacki, Hansen, Hass, Heiden, Hoffmann, Jendusa, Kelly, Keppler, Kroenke, Kujawa, LaBelle, Meyers, Mortier, Quirk, Schimenz, Schmidt, Schreiber, Sulkowski, Tomczyk, Whittow, Zillman and the President—27.

The Journal of the Proceedings of the regular meeting held July 14, 1953, was approved.

FILE NUMBER 53-1015

The matter of the revocation of Class "D" intoxicating liquor license of Gordon Lee Schroeder was taken up at this time.

The City being represented by John F. Cook, Assistant City Attorney, and the defendant appearing in person.

The City presented in evidence the complaint in the above matter, together with the summons and return thereon of Police Sergeant Louis Rozman.

Deputy Inspector of Police Rudolph Miller and police officer William Ericksson, being first severally duly sworn, testified in behalf of the City.

Gordon Lee Schroeder, being first duly sworn, testified in his own behalf.

George Mader, employer of Mr. Schroeder, requested the Common Council to grant Mr. Schroeder another chance.

THE CHAIR presented a resolution to revoke the Class "D" intoxicating liquor license of Gordon Lee Schroeder.

ALD. FLEMING moved that the foregoing resolution be placed on file.

The motion prevailed by the following vote:

Ayes:—Ald. Fass, Fleming, Froemming, Gromacki, Hass, Heiden, Hoffmann, Kelly, Keppler, Kroenke, LaBelle, Meyers, Quirk, Schimenz, Schmidt, Sulkowski, Tomczyk, Whittow and the President—19. Noes:—Ald. Choinski, Hansen, Jendusa, Mortier, Schreiber and Zillman—6. Not voting:—Ald. Collins and Kujawa—2.

UNFINISHED BUSINESS

FILE NUMBER 52-1221-e

A report of the Committee on Public Utilities recommending the adoption of a substitute resolution (Page 853) to restrain the Superintendent of the Water Department from introducing fluorides into the City water until Milwaukee County Medical Society reports on merits of the same, laid over from the last regular meeting was taken up at this time.

The report of the Committee was thereupon accepted and the resolution, as substituted, adopted by the following vote:

Ayes:—Ald. Choinski, Fleming, Gromacki, Hass, Heiden, Jendusa, Kelly, Kroenke, Kujawa, LaBelle, Meyers, Quirk, Schimenz, Whittow and the President—15. Noes:—Ald. Collins, Fass, Froemming, Hansen, Hoffmann, Keppler, Mortier, Schmidt, Schreiber, Sulkowski, Tomczyk and Zillman—12.

COMMUNICATIONS FROM CITY OFFICERS

THE CHAIR presented the following communications:

FROM THE OFFICE OF THE MAYOR

FILE NUMBER 52-1221-e

Milwaukee, July 31, 1953.

To the Honorable, the Common Council:

Gentlemen: I am herewith vetoing Resolution File Number 52-1221-e which restrains the superintendent of the water department from introducing fluorides into the city water until the Milwaukee County Medical Society has spoken on the merits of the issue.

I am submitting herewith a copy of a statement released in connection with my signing of Resolution Number 52-1221 in which the Common Council ordered placing trace amounts of fluorine in the Milwaukee water system. My reasons for signing Resolution Number 52-1221 and vetoing Resolution Number 52-1221-e are substantially the same: namely, a mandate from the electorate, as expressed in referendum on April 7, 1953, favored the introduction of fluorine.

To summarize the situation, conditions appear to be as follows: Milwaukee has a serious problem of tooth decay according to the health commissioner. Upon his recommendation it was proposed to introduce fluorine in the Milwaukee water system—approximately one part of fluorine to one million parts of water. This recommendation was based on widespread experience throughout the country where the presence of fluorine in these trace amounts seemed to have a direct effect in reducing tooth decay in a very substantial measure.

One principal opposition to fluorine appears to be based on the fact that while fluorine in trace amounts does prevent tooth decay, nevertheless, there is no degree of certainty as to whether or not it has ill effects on a cumulative basis.

The second source of opposition comes from indidividuals who have religious or psychological reasons for opposing changing the composition of the water supply. For these latter people there is no answer of a positive character which can be given them except one which might result from a test case to determine if their constitutional rights are being invaded. This legal remedy they will apparently have to seek, in view of the referendum vote.

Respectfully yours,

FRANK P. ZEIDLER, Mayor.

PRESIDENT McGUIRE IN THE CHAIR

THE CHAIR put the question: "Shall the foregoing resolution be adopted notwithstanding the objection of His Honor the Mayor?"

The foregoing resolution failed of adoption by the following vote:

Ayes:—Ald. Choinski, Fleming, Gromacki, Hass, Kroenke, Meyers, Quirk and the President—8.

Noes:—Ald. Fass, Froemming, Hansen, Heiden, Hoffmann, Jendusa, Kelly, Keppler, Kujawa, LaBelle, Mortier, Schimenz, Schmidt, Schreiber, Sulkowski, Tomczyk, Whittow and Zillman—18.

Not voting:—Ald Collins—1.

FILE NUMBER 53-1292

Milwaukee, July 31, 1953.

To the Honorable, the Common Council:

Gentlemen: I am herewith vetoing Resolution File Number 53-1292 which grants permission to the Wiviott Agency, on behalf of the Pacific Mutual Life Insurance Company, to provide poliomyelitis insurance at group rates to city employes.

I believe your Honorable Body will want to give further consideration to this resolution, as to the extent to which competitive agencies might want similar opportunities.

I am convinced that the agency making the original request had the best intentions of conforming to your wishes, but I believe that your policy reflected here needs further study.

Yours truly,

FRANK P. ZEIDLER, Mayor.

By unanimous consent, THE CHAIR at this time, presented the following:

FILE NUMBER 53-1292-a

Milwaukee, August 10, 1953.

To the Honorable, the Common Council:

Gentlemen:—On July 14, 1953, the Pacific Mutual Life Insurance Company, on behalf of the Wiviott Agency, submitted a proposal to provide poliomyelitis insurance at group rates to city employes.

I hereby withdraw the above-mentioned request.

Sincerely yours,

S. I. WIVIOTT,

Manager of Polio Insurance Dept.

Ordered on file.

your attention for the purpose of proceeding with the revocation of such license.

Respectfully,

STANLEY J. WITKOWSKI,

City Clerk.

(Communication and complaint appended.)

Referred to the Committee on Licenses-Rules-Engrossed Ordinances.

FILE NUMBER 53-2046

Milwaukee, October 20, 1953.

To the Honorable, the Common Council:

Gentlemen: I have the honor to inform you that I have been served with the following notices of injury, etc., viz.:

Notice of injury—Marie Janik vs. City of Milwaukee.

Notice of injury—Hildegarde Ryczek vs. City of Milwaukee.

Notice of injury—Irene Gray by her attorney, Samuel P. Murray vs. City of Milwaukee.

Notice of injury—Arthur J. Gray for injuries to his wife, Irene Gray, by his attorney Samuel P. Murray vs. City of Milwaukee.

Notice of injury—Mrs. Jeanette Senkel vs. City of Milwaukee.

Notice of injury and claim for damages—Harry Worland by his attorney N. Paley Phillips vs. City of Milwaukee.

Notice of injury and claim for damages—George Schrameyer by his attorney N. Paley Phillips vs. City of Milwaukee.

The same have been forwarded to the City Attorney.

Respectfully,

STANLEY J. WITKOWSKI,

City Clerk.

(Receipts of City Attorney appended.)

Ordered on file.

FROM THE CITY ATTORNEY

FILE NUMBER 52-1221-f

Milwaukee, October 14, 1953.

To the Honorable, the Common Council:

Gentlemen: This constitutes information to the Common Council that an action has been commenced in the Circuit Court of Milwaukee County, challenging the use of "certain inorganic fluoride chemicals" in the water system of the city, the plaintiffs and defendants being as follows:

Edward J. Froncek, Frank Grow, Alfred W. Kobiske, Myrtle Kobiske, Vernon Biddle, John Parker, and Florence Schroeder,

Plaintiffs,

—vs—

The City of Milwaukee, a Municipal Corporation, Walter M. Swietlik, as Commissioner of Public Works of the City of Milwaukee, Edward F. Tanghe, as Superintendent of the Water Works and Water Purification of the City of Milwaukee, Jos. J. Krueger, as Treasurer of the City of Milwaukee, and Virgil H. Hurless, as Comptroller of the City of Milwaukee,

Defendants.

The action raises, in addition to other issues, constitutional questions.

An order to show cause, returnable on October 23, 1953, at 9:30 a. m., has been issued by the Honorable Otto H. Breidenbach, Circuit Court Judge, directing the defendants in the action to show cause why each of them, their officers and agents, should not be restrained and enjoined from "enforcing in any manner, or carrying out, directly or indirectly, the Resolutions of the Common Council of the City of Milwaukee, set forth in the annexed complaint, from mixing any further sodium silicofluoride or other fluoride salt or compound in the water sold by the City of Milwaukee, from advertising for bids and from entering into or signing on behalf of the City of Milwaukee any contracts pursuant to said Resolutions, and from expending any funds in connection with the fluoridation of the Milwaukee water supply, pending the trial of this action on its merits or the further order of this court."

You are advised that the City Attorney will oppose the granting of the temporary restraining order being applied for, in accordance with the resolution adopted by the Common Council directing the proper city officers to "commence the introduction of sodium silicofluoride into water distributed by the Milwaukee Water Works."

Very truly yours,

HARRY G. SLATER,

First Assistant City Attorney.

Ordered on file.

FILE NUMBER 76342-b

Milwaukee, October 20, 1953

To the Honorable, the Common Council:

Gentlemen: In re: Stanley A. Kaminski v. City of Milwaukee—Appeal from Board of Assessment award of damages in the sum of $1,850.00 in connection with the opening, widening and extending of East and West Howard Avenue.

..Number

..Version
ORIGINAL
..Reference

..Sponsor
ALD. BOHL
..Title
Resolution ordering the immediate cessation and prohibition of the introduction of sodium silicofluoride or any fluoride compound into water distributed by the Milwaukee Water Works.
..Analysis
This resolution orders the Milwaukee Water Works to terminate and prohibit the introduction of sodium silicofluoride, or any fluoride compound, into water distributed by the Milwaukee Water Works.
..Body
Whereas, The Centers for Disease Control and Prevention has now acknowledged the findings of many leading dental researchers that the mechanism of fluoride's main benefits are derived from surface application to teeth and not from ingestion; and

Whereas, Despite being prescribed by doctors for over 50 years, the U.S. Food and Drug Administration has never approved any fluoride designed for ingestion as safe and effective; and

Whereas, Fluoride was not used for medical reasons but as a rat poison prior to 1938; and

Whereas, Fluoride is a cumulative poison, and 50% of the fluoride ingested each day is excreted through the kidneys and the remainder accumulates in bones, pineal gland and other tissues; and

Whereas, If the kidney is damaged, fluoride accumulation in a body will increase; and

Whereas, Growing evidence links fluoridated water with increased lead uptake; and

Whereas, Fluorosilicic acids bind with lead, leaching high levels of soluble lead from lead water pipes, delivering a compounded dose of toxic lead with toxic fluoride to Milwaukee water drinkers; and

Whereas, Once fluoride is put in the water supply it is impossible to control each dose an individual receives as fluoride is found in sources other than the water supply; and

Whereas, Other sources of fluoride include food and beverages processed with fluoridated water, fluoridated dental products, mechanically deboned meat, teas and pesticide residue on food; and

Whereas, A comparison of results from 24 studies of un-fluoridated districts in 8 countries revealed the reduction in dental caries are just as great in non-fluoridated areas as fluoridated; and

Whereas, The chemicals used to fluoridate water in the United States are not pharmaceutical-grade but instead come from wet scrubbing systems of the super-phosphate fertilizer industry; and

Whereas, These chemicals are classified as hazardous wastes contaminated with various impurities as recent testing by the National Sanitation Foundation suggested that the levels of arsenic in these chemicals are relatively high; and

Whereas, In 1953, the Common Council voted to add fluoride to the water supply at a time when Milwaukee had a serious problem of tooth decay and fluoridated tooth pastes, gels or mouthwash were not readily available, as they are today; and

Whereas, Some of the earliest opponents of fluoridation were biochemists, and at least 14 Nobel Prize winners are among numerous scientists who have expressed their reservations about the practice of fluoridation; and

Whereas, The American Dental Association and a number of dental researchers recommend that children under 12 months of age should not consume fluoridated water and that babies under 6 months of age should not receive any fluoride drops or pills due to an increased risk of dental fluorosis, a sign of overexposure to fluoride, and a lack of demonstrable benefit from ingesting fluoride before teeth erupt; and

Whereas, In January 2011, the U.S. Department of Health and Human Services reported that 41% of adolescents in the United States have dental fluorosis; and

Whereas, Children are being over-exposed to fluoride with the highest doses going to bottle-fed babies as infant formula is used with fluoridated tap water; and

Whereas, Minorities are more likely to use infant formula with fluoridated tap water, resulting in minority children ingesting significantly more fluorides and having higher rates of dental fluorisis; and

Whereas, Andrew Young, Alveda and Bernice King, Rev. W. Owens of the Coalition of African American Pastors, and League of United Latin American Citizens (LULAC) are among the growing number of fluoride opponents; and

Whereas, Fluoridation is unethical as individuals are not asked for their informed consent prior to medication, as is standard practice for the administration of all other medications; and

Whereas, Only through the total removal of all fluoridation from Milwaukee's public water supply can all residents be protected from the possible adverse health effects; now, therefore, be it

Resolved, By the Common Council of the City of Milwaukee, in its continuing effort to promote the health, safety and welfare of all, orders the immediate cessation and prohibition of the introduction of sodium silicofluoride, or any fluoride compound, into water distributed by the Milwaukee Water Works.

..Requestor

..Drafter
LRB137347-1
Amy E. Hefter
2/23/2012

WATER FLUORIDATION (GENERAL INFO)

Bohl, James

From: Pfaff, Richard
Sent: Tuesday, January 31, 2012 3:40 PM
To: Bohl, James
Subject: FW: Source of Fluoride, City of Milwaukee

From: Lewis, Carrie
Sent: Tuesday, January 31, 2012 2:59 PM
To: Pfaff, Richard
Subject: RE: Source of Fluoride, City of Milwaukee

Hi Richard,

We use choice #1 for fluoride.

Primary disinfectant is ozone. This is followed by free chlorine in the treatment plant, and chloramines in the distribution system.

Carrie
X2801

From: Pfaff, Richard
Sent: Tuesday, January 31, 2012 2:06 PM
To: Lewis, Carrie
Subject: Source of Fluoride, City of Milwaukee

Would you please steer me in the right direction relating to identifying whom to contact for answers to the 2 questions provided below?

Which of the following does the City use as a source of fluoride for its water fluoridation program?

1. Fluorosilicic Acid (aka Fluosilicic Acid or Hydrofluosilicic Acid).
2. Sodium Fluorosilicate (aka Sodium Silicofluoride).
3. Sodium Fluoride.

Also, which does the City use as a disinfectant, chlorine or chloramines?

Thanks.

Richard G. Pfaff, Manager
Legislative Reference Bureau
City of Milwaukee
Room 307, City Hall
200 E. Wells St
Milwaukee, WI 53202
(414) 286-2267 phone
(414) 286-0256 fax
www.milwaukee.gov/lrb

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RE: fluoride dosage

Bohl, James

sent: Wednesday, May 11, 2011 3:44 PM

To: Lewis, Carrie;

Thank you. I'll have to look at the state document provided by Dick Withers to me. I distinctly recall the state recommendation as being 1.0 milligrams per liter. Will let you know if my recollection is correct or not. jb

---

From: Lewis, Carrie
Sent: Wed 5/11/2011 3:18 PM
To: Bohl, James
Subject: fluoride dosage

After our conversation this afternoon, I did a little research on why fluoride is added to reach 1.1 mg/L.

Wisconsin Administrative Code NR 809.74 states:

"(1) PUBLIC WATER SYSTEMS WHICH ADD FLUORIDE. (a) The water supplier for a community water system artificially fluoridating the water shall establish a monitoring program in order to maintain the fluoride concentration within the range of 1.0 to 1.5 milligrams per liter as recommended by the dental health section of the department of health services for optimum dental benefits."

This shows that the level is recommended by DOHS and enforced by DNR. The requirement to fluoridate at all comes from a Milwaukee Common Council resolution.

MWW has a target of 1.1 milligram per liter because there is a slight amount of variability in the fluoride addition, and by aiming for 1.1 we do not violate the lower limit of 1.0 milligram per liter.

I hope this information is helpful.

Respectfully,

Carrie

The Case Against Fluoride

Posted by Paul Connett on November 10, 2010

[disinfo ed.'s note: The following is an excerpt from *The Case Against Fluoride: How Hazardous Waste Ended Up in Our Drinking Water and the Bad Science and Powerful Politics That Keep It There* by Paul Connett, James Beck, Spedding Micklem, courtesy of Chelsea Green Publishing]

At a public meeting held on October 17, 2009, in Yellow Springs, Ohio, a community that was considering halting its fluoridation program, Paul Connett gave a twenty-minute presentation on the scientific arguments against the practice. After a county health commissioner and local dentist responded, a woman in the audience said, "Whether this practice is safe or not, or beneficial or not, I want freedom of choice. It is my right to choose what substances I put into my body, not some governmental agency's."

This woman echoed what many opponents of fluoridation have believed and articulated for over sixty years: Government has no right to force anyone to take a medicine. Thus, while in the effort to end this practice worldwide it is helpful to provide scientific evidence that the program is neither effective nor safe, this commonsense position remains the crux of the argument against fluoridation.

The Need for Informed Consent

Every doctor knows, or should know, that he or she cannot force an individual to take medicine without that patient's informed consent. Doctors must tell their patients the benefits of any medicine prescribed and warn of any possible side effects. After they have done this, it is the patient—and only the patient—who should make the final decision as to whether to take the medicine.

This is what the American Medical Association (AMA) has to say about informed consent:

Informed consent is more than simply getting a patient to sign a written consent form. It is a process of communication between a patient and physician that results in the patient's authorization or agreement to undergo a specific medical intervention.

In the communications process, you, as the physician providing or performing the treatment and/or procedure (not a delegated representative), should disclose and discuss with your patient:

- the patient's diagnosis, if known;
- the nature and purpose of a proposed treatment or procedure;
- the risks and benefits of a proposed treatment or procedure;
- alternatives (regardless of their cost or the extent to which the treatment options are covered by health insurance);
- the risks and benefits of the alternative treatment or procedure; and
- the risks and benefits of not receiving or undergoing a treatment or procedure.

CDC 32% of American children have Dental Fluorosis

In turn, your patient should have an opportunity to ask questions to elicit a better understanding of the treatment or procedure, so that he or she can make an informed decision to proceed or to refuse a particular course of medical intervention.

This communications process, or a variation thereof, is both an ethical obligation and a legal requirement spelled out in statutes and case law in all fifty states of the United States.1

By violating the individual patient's right to informed consent, fluoridation allows decision makers, without medical qualifications, to do to the whole community what an individual doctor is not allowed to do to his or her individual patients.

Counterargument 1: It Is Unethical Not to Fluoridate

Proponents respond to this ethical argument by turning it upside down. They argue that it is unethical to deprive children of a benefit that might reduce pain and help them lead healthier lives, especially children from low-income families.

However, by not putting fluoride in the water, you are not depriving anyone of access to fluoride: It is available in tablet form and in fluoridated toothpaste. (For a discussion about topical versus systemic benefits, see chapters 2 and 6.)

From an economic perspective, avoiding fluoride in water is an expensive business, whether it involves purchasing bottled water for cooking and drinking or the use of distillation equipment or reverse osmosis systems. Thus, low-income families are disproportionately burdened by fluoridation since by and large they cannot afford avoidance measures.

In the United States, dental decay is concentrated in poor and minority families. Fifty-five years after fluoridation began, the U.S. surgeon general stated in his 2000 report, Oral Health in America: "There are profound and consequential disparities in the oral health of our citizens. Indeed, what amounts to a 'silent epidemic' of dental and oral diseases is affecting some population groups. Those who suffer the worst oral health are found among the poor of all ages, with poor children and poor older Americans particularly vulnerable. Members of racial and ethnic minority groups also experience a disproportionate level of oral health problems."2

The motivation for targeting poor children for extra help is highly laudable, but adding fluoride to the drinking water to do so is misguided. In fact, it makes an inequitable situation even worse. This is because in Western countries the children most likely to suffer from poor nutrition come from low-income families, and we will see in chapter 13 that people with inadequate diets are those most vulnerable to fluoride's toxic effects. In our view, children from low-income families are the very last children who should be exposed to ingested fluoride.

Counterargument 2: No One Is "Forced" to Drink the Water

Proponents of fluoridation further counter the notion that fluoridation in the public water system violates the individual's right to informed consent to medication by arguing that fluoridated water is only delivered to the tap and no one is actually forced to drink it.

This argument certainly does not apply to low-income families. Their economic circumstances do force them to drink the water coming out of the tap. Thus, a program that is billed as equitable is actually inequitable, since families of low income are trapped by a practice that may cause them harm (see chapters 11, 13–19).

Moreover, even for families with the means to buy bottled water for drinking and cooking, or equipment to remove the fluoride at the tap, it is very difficult to avoid fluoride once it has been put in the community's water supply. It will be in every glass of water and cup of coffee or tea consumed in town—at work and in friends' homes. It will also be in the water that is used to water the garden and in the shower and bath water.

Counterargument 3: Fluoride Is a Nutrient, Not a Drug

Proponents have tried to muddy the waters in the argument of violation of informed consent and unacceptability of "mass medication" by insisting that fluoride is not a medicine or drug, but a nutrient. We examine the evidence for their claims.

Is Fluoride an Essential Nutrient?

There is little or no evidence that fluoride is an essential nutrient. To demonstrate that a substance is an essential nutrient one has to demonstrate that some disease results from depriving an animal or a human of this substance. This has never been done for fluoride (see chapter 12).

In a 1998 letter by Bruce Alberts, president of the National Academy of Sciences, and Kenneth Shine, president of the Institute of Medicine, to Professor Albert Burgstahler, editor of the journal Fluoride and several other scientists, in response to their complaint to the National Academy about the Institute of Medicine's inclusion of fluoride in the list of nutrients in its report Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride,3 the following quote appeared:

First, let us reassure you with regard to one concern. Nowhere in the report is it stated that fluoride is an essential nutrient. If any speaker or panel member at the September 23rd workshop referred to fluoride as such, they misspoke. As was stated in Recommended Dietary Allowances 10th Edition, which we published in 1989: "These contradictory results do not justify a classification of fluoride as an essential element, according to accepted standards. Nonetheless, because of its valuable effects on dental health, fluoride is a beneficial element for humans."4

What Alberts and Shine do not discuss here is whether the supposed benefits of this "beneficial element" are obtained from some internal biological process or via some nonbiological interaction of the fluoride with the surface of the tooth enamel. This is a crucial difference when considering water fluoridation, since the former would necessitate swallowing fluoride and the latter would not (see chapter 2).

While there is no solid scientific evidence supporting the notion that fluoride is a nutrient, strenuous attempts have been made by a number of proponents throughout the history of fluoridation to try to establish this notion in the public mind. In chapter 26 we examine these

efforts, in particular the effort by Harvard researcher Dr. Frederick Stare and the aid given to him by the sugar and food lobbies.

Is Fluoride a Drug?

In a letter sent in December 2000 to Congressman Kenneth Calvert, chairman of the Subcommittee on Energy and the Environment, of the Committee on Science, the U.S. Food and Drug Administration (FDA) stated, "Fluoride, when used in the diagnosis, cure, mitigation, treatment, or prevention of disease in man or animal, is a drug that is subject to Food and Drug Administration regulation."5 The National Association of Pharmacy Regulatory Authorities in Canada lists "sodium fluoride" and "fluoride and its salts" as drugs.6

According to Cheng et al. in an article appearing in the British Medical Journal, "The legal definition of a medicinal product in the European Union (Codified Pharmaceutical Directive 2004/27/EC, Article 1.2) is any substance or combination of substances 'presented as having properties for treating or preventing disease in human beings.'"7

Both the Centers for Disease Control and Prevention (CDC)8 and the American Dental Association (ADA),9 the main proponents of fluoridation in the United States, describe dental caries (tooth decay) as a "chronic infectious disease" and recommend fluoride to prevent the disease.

If fluoride is a drug or medicinal product, fluoridation is medication delivered on a massive scale.

An Unapproved Drug

In a June 3, 1993, letter to FDA commissioner Dr. David Kessler, former New Jersey assemblyman John V. Kelly wrote, "The Food and Drug Administration Office of Prescription Drug Compliance has confirmed, to my surprise, that there are no studies to demonstrate either the safety or effectiveness of these drugs [fluorides], which FDA classified as unapproved new drugs."10

It goes without saying that it would be highly questionable to deliver any drug via the public water system—let alone fluoride, which the FDA calls an unapproved drug. The designation "unapproved drug" means that it has not gone through rigorous trials to establish either its effectiveness or its safety. This designation also puts into question the ethics and legality of school nurses and teachers administering fluoride pills and/or rinses to students in U.S. schools located in non-fluoridated areas.

Other Arguments

Violating the modern medical ethic of informed consent is not the only feature of fluoridation that makes it a poor medical practice. In a recent videotaped interview, Earl Baldwin, a member of the British House of Lords and one of the advisory board members for the York Review, the UK-sponsored review of fluoridation,11 explained why he thought fluoridation was a bad idea:

"What physician do you know, who in his or her right mind, would treat someone he does not know and has never met, with a substance that's meant to do change in their bodies, with the advice: 'Take as much, or as little, as you like, but take it for a lifetime because it may help someone's teeth'?"12

Independent observers have been saying similar things since the inception of fluoridation, but these arguments have fallen largely on deaf ears. This is not because the reasoning lacks merit, but because those who promote fluoridation have the power to ignore both common sense and scientific argument. We examine the strategies and tactics used in the promotion of fluoridation in chapter 23. In the following sections we examine some of the commonsense arguments of opponents such as Earl Baldwin in more detail.

No Control over Who Gets the Medicine

For those who promote fluoridation, one of its attractions is that it delivers fluoride to everyone indiscriminately. But for opponents this is one of its greatest weaknesses. When fluoride is added to the water supply, it goes to everyone, including those most vulnerable to fluoride's known toxic effects. These include above-average water consumers; the very young; the very old; those with diabetes; those with low thyroid function or kidney disorder; and those with an inadequate diet, including those suffering from outright or borderline iodine deficiency (see chapter 16). Also, as we indicated above, it goes to families of low income who cannot afford avoidance measures.

No Control of Dose

A critical problem with delivering a medicine via the water supply is that there is no control over the dose. Dr. Arvid Carlsson discussed this issue in a letter he wrote in February 2009:

Fluoridation is an obsolete practice. It goes against all principles of modern pharmacology. The use of the public drinking water supply to administer the same dose of fluoride to everyone, from the infant to those who consume copious amounts of water (such as diabetics), goes against all principles of science because individuals respond very differently to one and the same dose and there are huge variations in the consumption of this drug.13

Concentration versus Dose (from water and other sources)

Proponents of fluoridation stress how well engineers can control and monitor the concentration of the fluoridating agent added to the water supply. However, controlling concentration, measured in the case of fluoride in milligrams per liter (mg/liter), is not the same as controlling dose, which is measured in milligrams consumed per day (mg/day).

If someone drinks 1 liter of water containing fluoride at 1 mg/liter (i.e., 1 ppm, which is the concentration at which it is administered), they will ingest 1 mg of fluoride. If they drink 2 liters, they will receive 2 mg of fluoride, and so on. The dose gets larger the more water is drunk; and the larger the dose, the more likely it will cause harm. This is particularly serious for a substance like fluoride, which is known to be highly toxic at moderate to high doses, which accumulates in

the bone, and for which there is little, if any, margin of safety to protect the most vulnerable against known health risks (see chapter 20).

We also receive fluoride from sources other than the water supply, and this amount varies from individual to individual. Thus, it is the total dose from all sources we should be concerned about.

To determine potential harm, we also have to take into account the body weight of the consumer. We discuss the difference between dose and dosage below.

Dose versus Dosage

The dose of aspirin or any other drug considered safe for a grown-up is not a safe dose for a baby. Similarly, a safe dose of fluoride for an adult cannot be considered safe for a baby. Thus it is alarming when one discovers that, over the course of the day, bottle-fed babies can receive nearly as much fluoride as an adult who drinks 1 liter of fluoridated water. According to the U.S. Environmental Protection Agency in a 2008 article on why children may be especially sensitive to pesticides, "In relation to their body weight, infants and children eat and drink more than adults."14 The way toxicologists determine the safe dose for different ages is to adjust for the average body weight of the age range in question.

According to the EPA's 1986 calculation of a safe drinking water standard, a safe daily dose of fluoride for a 70-kg (154-lb) adult is supposed to be 8 mg per day.15 In chapter 20, we challenge the faulty reasoning that led to this high figure. But in the meantime, if we adjust this figure of 8 mg per day for body weight, that would mean that only 0.8 mg per day would be safe for a 7-kg (15-lb) infant (i.e., a ten times lower dose because the baby's body weight is ten times lower). Even that dose may be too high for a baby, however, because a baby's developing tissues, particularly the brain, are much more vulnerable to toxic agents than an adult's. An infant is not simply a miniature adult.

Dose divided by a person's body weight is called dosage and is measured in milligrams per kilogram of body weight per day (mg/kg/day). The safe dose for an adult divided by an adult's body weight (assumed to be 70 kg) is called the reference dose, or RfD. Strictly speaking, we should call this a reference dosage, but people seldom do. Note the different units here. If we are talking about dose, we are speaking about mg/day, but if we are talking about a reference dose, or dosage, we are speaking about mg/kg/day. This is a big difference.

Now let's look at a real-life example of using a reference dose. The EPA lists IRIS reference doses for a number of toxic substances. IRIS stands for Integrated Risk Information System; it is used for health-risk assessments. The EPA's RfD for fluoride listed in IRIS is 0.06 mg/kg/day.16

It is worrying to see that this IRIS RfD is easily exceeded by a baby consuming formula made with fluoridated water. For example, a 10-kg infant drinking each day 1 liter of water containing fluoride at 1 ppm will get a dosage of 0.10 mg/kg/day (1 mg/day divided by 10 kg). That is almost twice the IRIS RfD.

It was after the 2006 U.S. National Research Council report17 made it clear that bottle-fed babies were exceeding the IRIS RfD that the ADA finally recommended to its membership, in November 2006, that they advise their patients not to use fluoridated water to make baby formula.18 The CDC followed suit,19 but neither has made much of an effort to get this information to parents.

Different Responses to Same Dose

It is well known that there is a very wide range of sensitivity across the human population to any drug or toxic substance. Some people will be very resistant, while others will be very vulnerable or sensitive to the same substance. Most of us will have an average tolerance; however, we can anticipate that the most sensitive will be at least ten times more vulnerable than the average responder. Those who promote fluoridation gloss over the insufficient margin of safety to protect all citizens, especially the most sensitive, from the known adverse health effects of fluoride (see chapters 13 and 20).

Warnings, Help, and Compensation

One thing that is generally accepted about water fluoridation is that where it is implemented, the rates of dental fluorosis (mottling and discoloration of the enamel; see chapter 11) in children will rise. Very little warning is being given about this, especially to low-income families who bottle-feed their babies with formula made with fluoridated tap water. Nor is any financial help being provided to those families whose children are so affected. It can cost up to $1,000 to treat a fluorosed tooth with veneers—more when the veneers have to be replaced in subsequent years.

According to the CDC, 32 percent of American children are affected by dental fluorosis.20 → Most recently this has been revised upward to 40%
While most of those children have the very mild condition, those with the mild, moderate, or severe condition make up about 10 percent of the total, and many of those may need treatment (see chapter 11). Ten percent being affected would mean some 32,000 children in a city of one million needing cosmetic treatment that few families can afford. Public and media concern is growing on this issue; for example, see the transcript of a TV news clip from CBS in Atlanta, Georgia, broadcast in March 2010, at http://www.cbsatlanta.com/health/22776266/detail.html.21

Mandatory Fluoridation

The imposition of fluoridation on individuals without their informed consent becomes even more egregious when legislation is introduced to mandate the practice for whole states, provinces, or countries. While we do not consider that a local referendum is ethically satisfactory, since the medicine we take should not be determined by our neighbors, such a process may allow discussion, deliberation, and the opportunity for people to express their concerns—at least at the local level. When the practice of adding fluoride to the public water system becomes mandatory at the state, provincial, or even national level, the vast majority of the population has little idea of what is going on, either during the passage of the legislation or subsequently, when the measure is enforced. Informed citizens are usually dispersed in large jurisdictions and have few resources to match the lobbying power of either the national dental associations or governmental health bodies hell-bent on introducing this measure. Those who hold the ethical requirement of

informed consent to be the final argument on this matter will continue to battle at the national and international levels to insist on this principle being recognized. But in practice, in today's world, local democracy—when it is allowed to operate—probably offers citizens a greater chance of protecting themselves against forced fluoridation.

A number of legislatures have introduced mandatory fluoridation legislation in various states within countries and sometimes for the whole country. These include the states of Victoria and Queensland in Australia; the states of California, Connecticut, Georgia, Illinois, Indiana, Louisiana, Michigan, Minnesota, Nebraska, Nevada, Ohio, and Tennessee (as well as Washington, D.C.) in the United States; and the countries of Singapore and the Republic of Ireland. As we write, efforts to introduce mandatory fluoridation are under way in the U.S. states of New Jersey, Oregon, and Pennsylvania.

Mandatory fluoridation measures violate the principle of the crucial role of community participation in health measures outlined in the Ottawa Charter for Health Promotion.22 Mandatory fluoridation also violates the Council of Europe's Convention on Human Rights and Biomedicine, whose article 5 states, "An intervention in the health field may only be carried out after the person concerned has given free and informed consent to it. This person shall beforehand be given appropriate information as to the purpose and nature of the intervention as well as on its consequences and risks. The person concerned may freely withdraw at any time."23

No local, state, or federal government—no matter how well intentioned—has the right to force anyone to take a medicine for a disease that is neither contagious (in a communal sense) nor life threatening.

Summary

Fluoridation—the deliberate addition of fluoride to the public water supply—is a poor medical practice because it violates the principle of informed consent to medication. It is indiscriminate and offers no control over the dose received by an individual. It makes inadequate allowance for differing sensitivity to toxic effects, or for the size and body mass of recipients; this last point is particularly important for young children who may receive proportionately much higher dosages than adults at a time when their bodies are far more vulnerable to toxic agents. Fluoride used in the fluoridation of drinking water is considered to be a drug, not a nutrient. It is chronically toxic at moderate doses. As a drug, it has not been rigorously tested and has not been approved by the U.S. FDA. Fluoridation increases the chances that a child will develop fluorosis of the permanent teeth, which can be disfiguring and require expensive cosmetic treatment in a minority of cases. The notion that fluoridation is equitable is misplaced for two reasons: Children from low-income families are more likely to have poor nutrition, making them more vulnerable to fluoride's toxic effects; and low-income families are least able to afford avoidance measures.

1986

The mystery of declining tooth decay

from Mark Diesendorf

Large temporal reductions in tooth decay, which cannot be attributed to fluoridation, have been observed in both unfluoridated and fluoridated areas of at least eight developed countries over the past thirty years. It is now time for a scientific re-examination of the alleged enormous benefits of fluoridation.

FLUORIDATION consists of raising the concentration of the fluoride ion F^- in water supplies to about 1 part per million (p.p.m.) with the aim of reducing dental caries (tooth decay) in children. In fluoridated areas, there are now many longitudinal (temporal) studies which record large reductions in the incidence of caries[1]. The results of these and of fixed time surveys have led to the 'fluoridation hypothesis', namely that the principal cause of these reductions is fluoridation.

Until the early 1980s, there had been comparatively few longitudinal studies of caries in unfluoridated communities. Only a small minority of the studies in fluoridated areas had regularly examined control populations, and there seemed to be little motivation to study other unfluoridated communities. But during the period 1979–81, especially in western Europe where there is little fluoridation, a number of dental examinations were made and compared with surveys carried out a decade or so before. It soon became clear that large reductions in caries had been occurring in unfluoridated areas (see below). The magnitudes of these reductions are generally comparable with those observed in fluoridated areas over similar periods of time.

In this article, these reductions are reviewed and attention is also drawn to a second category of caries reduction which cannot be explained by fluoridation. This category is observed in children described by proponents of fluoridation as having been 'optimally exposed', that is, children who have received water fluoridated at about 1 p.p.m. from birth. The observation is that caries is declining with time in 'optimally exposed' children of a given age. In some cases, the magnitudes of these reductions are much greater in percentage terms than the earlier reductions in the same area which had been attributed to fluoridation.

The problem of explaining the two categories of reduction goes well beyond the field of dentistry: contributions from nutritionists, immunologists, bacteriologists, epidemiologists and mathematical statisticians, amongst others, may be required.

Table 1 Studies reporting large reductions in dental caries in unfluoridated areas

Location		Years surveyed	References
Australia	Brisbane	1954, '77	2, 3
	Sydney	1961, '63, '67	4
Denmark	Various towns	1972, '79	53
Holland	The Hague	1969, '72, '75, '78	38
	Various towns	1965, '80	11
New Zealand	Auckland (parts)	1966, '74, '81	12
Norway	Various towns	1970, '80	54
Sweden	Various towns	1973, '78, '81	39
	North Sweden	1967, '77	55
United Kingdom	Bristol	1970, '79	56
	Bristol	1973, '79	56
	Devon	1971, '81	37
	Gloucestershire	Annually from 1964	37*
	Isle of Wight	1971, '80	57
	North-West England	1969, '80	58
	Scotland	1970, '80	59
	Shropshire	1970, '80	10
	Somerset	1975–79 annually	60
	Somerset	1963–79	61
United States	Dedham, Mass.	1958, '74	40
	Norwood, Mass.	1958, '72, '78	40
	Massachusetts: sample of schools	1951, '81	41
	Ohio	1972, '78	62

* Unpublished communication from J. Tee (1980), Area Dental Officer, Gloucestershire, to R. J. Anderson *et al.*[37]

Caries in unfluoridated areas

Table 1 lists over 20 studies which report substantial temporal reductions in caries in children's permanent teeth in unfluoridated areas of the developed world. In many of these cases, the magnitudes of these reductions are comparable with those observed in fluoridated areas and attributed to fluoridation.

Several of these studies give clues as to factors which are unlikely to be the main causes of the reductions. A comparison of the 1954 and 1977 dental health surveys in Brisbane[2,3] indicates to a reduction of about 50% in caries, as measured by the number of decayed, missing and filled permanent teeth (DMFT) per child and averaged over the age groups, in the 23-year period. The 1977 survey distinguished between children who took fluoride tablets regularly, irregularly or not at all. Although there were differences in caries incidences between the three categories (which could reflect factors unrelated to fluoride levels), even the "no tablet" group had on average 40% less caries experience than that recorded in 1954. So fluoride tablets were not the principal cause of the reductions observed in Brisbane.

The first Sydney study[4] showed that children with "naturally sound" teeth increased from 3.8% in 1961 to 20.2% in 1967 and 28% in 1972. The paper, which was titled enthusiastically "The Dental Health Revolution", was originally used widely to promote fluoridation in Australia. The authors stated that: "Almost certainly, the availability of fluoride both in tablet form and delivered through town water supplies has been the predominant factor. . . . These very large reductions represent a modern triumph of preventive health care"[4]. Yet the major proportion of the reported improvement had already occurred before Sydney was fluoridated in 1968. Moreover, no evidence was presented that fluoride tablets were widely used in the 1960s. Fluoride toothpaste was only introduced into Australia in 1967[3]. Although the index "naturally sound" teeth is unsuitable for more detailed

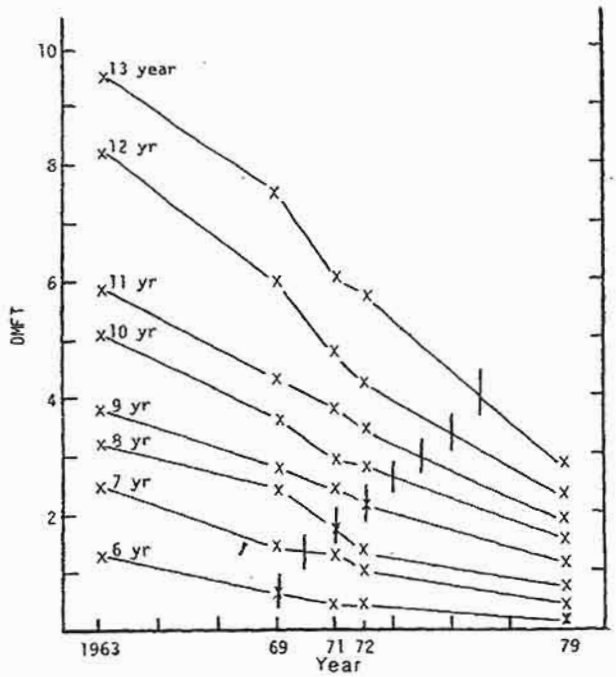

Fig. 1 Decline in caries, as measured by DMFT, in Tamworth, Australia, for children in age groups 6 years to 13 years. Data compiled from refs 14, 15. The vertical line cutting graph for each age group denotes year at which maximum possible benefit from fluoridation was reached. Tamworth was fluoridated in 1963.

studies which distinguish decayed, missing and filled teeth, the populations examined were very large (over 9,000 children at each examination) and the results clear-cut.

A second Sydney study[5] used the DMFT index, but was irrelevant for establishing any link with fluoridation, since it reported only on examinations in 1963 and 1982, but not around 1968 when Sydney was fluoridated. As in several other fluoridation studies, the key data were either not collected or not reported[6]. Although the two Sydney papers have an author in common (James S. Lawson, a senior officer of the New South Wales Health Commission), the second paper does not even cite the first. This suggests that, once it became clear that the first Sydney study contained evidence unfavourable to fluoridation, it was a source of embarrassment to some fluoridation proponents who are apparently trying to denigrate it.

However, independent confirmation of the large reductions in caries before fluoridation reported in the first Sydney study[4] is readily obtained by comparing the results of two surveys[7,8] separated by 20 years by Barnard. These surveys showed that the mean DIMF index ('I' denotes a permanent tooth which cannot be restored) for school children aged 13 and 14 declined from 11.0 in 1954–55 to 6.0 in 1972. The four years from 1968, when fluoridation commenced in Sydney, to 1972, would not have contributed significantly to the decline in caries prevalence in this age group[9].

The authors of one of the British studies[10] cited in Table 1 point out that sales of fluoride toothpaste in the United Kingdom were less than 5% of total sales in 1970, but rose to more than 95% of sales in 1977. They quote unpublished annual data from unfluoridated parts of Gloucestershire, collected from 1964 onwards, which show substantial improvements in children's teeth before the use of fluoride toothpaste became significant.

Many of the studies in the Netherlands, reviewed by Kalsbeek[11], were carried out to evaluate the effectiveness of the school dental health programme. Temporal reductions in DMFT of about 50% occurred between 1970 and 1980, whether or not the children had taken part in the dental health education program. Kalsbeek also reviewed the use of fluoride tablets and toothpaste and concluded from the data that "factors other than the effects of different fluoride programmes must play a role."

The study in the partly fluoridated city of Auckland, New Zealand[12], examined the influence of social class (which reflects environmental and lifestyle factors, such as diet) as well as fluoridation on dental health as measured by the levels of dental treatment received by children. The paper showed that treatment levels have continued to decline in both fluoridated and unfluoridated parts of the city and that these reductions are related strongly to social class, there being less caries in the "above average social rank" group than in other children. Thus the main ethical argument for fluoridation, that it should assist the disadvantaged, is not borne out by this study.

Fluoridation's benefits

On 15 December 1980, the Dental Health Education and Research Foundation, one of the main fluoridation promoting bodies in New South Wales (NSW), issued a press release entitled, "Fluoridation dramatically cuts tooth decay in Tamworth"[13]. This document, which highlighted results of a study conducted by the Department of Preventive Dentistry, Sydney University, and the Health Commission of NSW, stated in part:

> Tamworth's water supply was fluoridated in 1963, and the last survey in the area was conducted in August 1979. It shows decay reductions ranging from 71% in 15-year-olds to 95% in 6-year-olds. ... All those surveyed were continuous residents using town water.

The "95%" reduction actually corresponded to a reduction in DMFT from 1.3 in 1963 to 0.1 in 1979[14], which is 92%. The press release implied incorrectly that all this reduction was due to fluoridation. However, it has been claimed ever since

Table 2 Extent of fluoridation in Australia, 1977 and 1983

State or territory	Capital city	Year city fluoridated*	% Of state fluoridated† in 1977	% Of state fluoridated† in 1983
ACT	Canberra	1964	100	100
Tasmania	Hobart	1964	74	77
NSW	Sydney	1968	81	81
WA	Perth	1968	83	83
SA	Adelaide	1971	71	70
Victoria	Melbourne	1977	0.7 then 73	71
Queensland	Brisbane	Not fluoridated	10	5

* Each capital city has the majority of the population of its state or territory.

† That is, the percentage of population of state/territory which drinks fluoridated water. Data from Annual Reports of Director-General of Health, for example ref. 17.

the commencement of fluoridation that the maximum possible benefits from fluoridation are obtained in children who have drunk fluoridated water from birth. Six-year-olds would have done this by 1969, when, according to the published data[15], they had a DMFT index of 0.6. The further reduction in caries in optimally exposed 6-year-olds, observed in years following 1969, cannot be due to fluoridation.

Thus, one can say that at best fluoridation could have approximately halved the DMFT rate in 6-year-olds between 1963 and 1969. (Since there was no control population, one could also say that at worst fluoridation might have had no effect in that period.) But from 1969 to 1979, caries in 6-year-olds was reduced a further 83%, by some other factor(s) than fluoridation.

Figure 1 shows that the unknown factors caused in children of each age from 6 years to 9 years similar large reductions in caries. Unfortunately, there are no published data for Tamworth beyond 1979 or in the years between 1972 and 1979, and so it cannot be confirmed whether the large reductions observed[14,15] from 1972 to 1979 in children aged 10 to 15 were also due to these unknown factors.

A similar reduction beyond the maximum possible for fluoridation is observed for children of each age from 6 to 9 in the published data from Canberra[16], which cover the period from 1964, the stated year of fluoridation, to 1974. In particular, DMFT rates declined by 50% in 6-year-olds from 1970 to 1974 and by 54% in 7-year-olds from 1971 to 1974. These reductions in optimally exposed children cannot be due to fluoridation. Published post-1974 data are needed to check on further reductions in optimally exposed children aged over 9 years.

From 1977 onwards, data have been systematically collected from the school dental services in each Australian state and territory[9,17]. Table 2 shows the degree of fluoridation in each of these states/territories in 1977 and 1983 and also the dates of fluoridation of the capital cities of these regions. Each of these cities dominates the population of the state or territory in which it lies. The evidence presented in Fig. 2 and Table 2 suggests that states and territories which had been extensively fluoridated for at least 9 years before 1977 (Tasmania, Western Australia and New South Wales) had qualitatively similar large reductions in caries from 1977 to 1983 as a state which was only extensively fluoridated in 1977 (Victoria) and a state which had a small and declining fraction of fluoridation (Queensland). Although the results of the school dental health survey are recorded by age and state, the data have only been published[9,17,18] so far for ages 6–13 averaged in each state, or for each age for the whole of Australia. There is evidence that the use of fluoride toothpaste in Australia reached a high plateau around 1978, so these observed reductions in caries can be due neither to fluoride toothpaste[9] nor to fluoridated water.

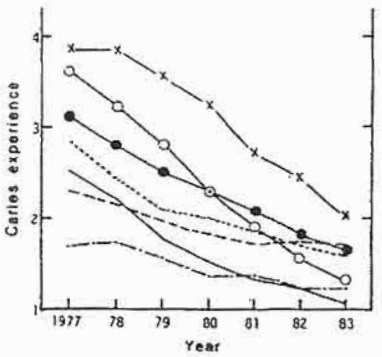

Fig. 2 Decline in the average number of (permanent) teeth per child with caries experience in each Australian state and the Australian Capital Territory as observed in school dental services[17]. 'Caries experience' can be one or more decayed, missing or filled teeth, and consists of an average for children aged 6–13 years. See Table 2 for information on the extent of fluoridation in each state/territory in 1977 and 1983 and the year when the main population centre of each state/territory was fluoridated. ×, Victoria; ○, Tasmania; ●, Queensland; - - - -, SA; ———, NSW; — —, WA; ·—·—·, ACT.

It is to be hoped that similar data on caries reductions in "optimally exposed" children will be sought in other fluoridated countries. In a region of Gloucestershire, United Kingdom where the main water supply was naturally fluoridated with 0.9 p.p.m. fluoride until 1972, reductions in caries of 51% were observed in 12-year-old children between 1964 and 1979[19]. Factors other than fluoridated water must have caused these reductions. After 1972, the main water supply was drawn from a bore with less than 0.2 p.p.m. fluoride, so a recent survey of caries there would be of great interest.

Benefits overestimated?

In some fluoridated areas (for example Tamworth, Australia), temporal reductions in caries have been wrongly credited to fluoridation. The magnitude of these reductions is similar in both fluoridated and unfluoridated areas, and is also generally comparable with that traditionally attributed to fluoridation. Can it be concluded that communities which prefer not to fluoridate, either because of concern about potential health hazards[20–25] or for ethical reasons (for example compulsory medication; medication with an uncontrolled dose), do not necessarily face higher levels of tooth decay than fluoridated communities? In other words, is it reasonable to ask whether it could be generally true that a major part of the benefits currently attributed to fluoridation is really due to other causes?

Such a hypothesis would seem to be possible in principle because it is well known that fluoridation is neither 'necessary' nor 'sufficient' (the words between inverted commas being used in the formal logic sense) for sound teeth; that is, some children can have sound teeth without fluoridation, and some children can have very decayed teeth even though they consume fluoridated water[25].

To confirm or refute the hypothesis, it is necessary (but not 'sufficient') to examine the absolute values of caries prevalence in fluoridated and unfluoridated areas. If it is true that the absolute values of caries prevalence in some unfluoridated areas are comparable with those in some unfluoridated areas of the same country, then the hypothesis is supported (but not proven), and there would be a strong case for the scientific re-examination of the epidemiological studies which appear to demonstrate large benefits from fluoridation.

The earliest set of studies comparing caries in fluoridated and unfluoridated areas were time-independent surveys of caries prevalence in areas with 'high' natural levels of fluoride in water supplies, conducted by H. T. Dean and others in the United States[26]. The surveys purported to show that there is an "inverse relationship" between caries and fluoride concentration. From the viewpoint of modern epidemiology, these early studies were rather primitive. They could be criticized for the virtual absence of quantitative, statistical methods, their nonrandom method of selecting data and the high sensitivity of the results to the way in which the study populations were grouped[25].

Results running counter to the alleged inverse relationship have been reported from time-independent surveys in naturally fluoridated locations in India[27], Sweden[28], Japan[29], the United States[30] and New Zealand[31,63]. The Japanese survey[29] found a minimum in caries prevalence in communities with water F-concentrations in the range 0.3–0.4 p.p.m.; above and below this range, caries prevalence increased rapidly.

These surveys[27–31] also selected their study regions nonrandomly. But recently Ziegelbecker[32] attempted to make a selection close to a random sample by considering 'all' available published data on caries prevalence in naturally fluoridated areas. His large data set, which includes Dean's as a sub-set, comprises 48,000 children aged 12–14 years drawn from 136 community water supplies in seven countries. He found essentially no correlation between caries and log of fluoride concentration. The surveys[27–32] are generally omitted from lists[1] of studies on the role of fluoridation in caries prevention.

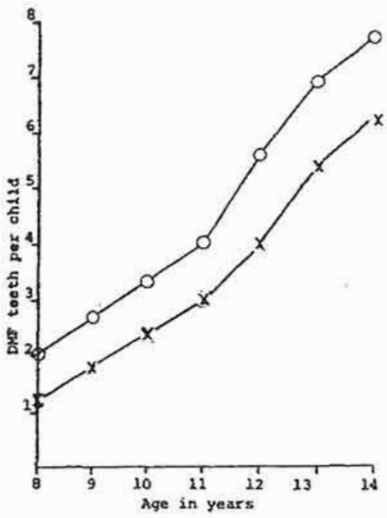

Fig. 3 The variation with age of decayed, missing and filled permanent teeth (DMFT) in fluoridated test towns (×) and unfluoridated control towns (○) in Britain, graphed from data published by the UK Department of Health[33]. Note that the rate of increase of DMFT is essentially the same in both groups. Children in the fluoridated areas have an average only one less cavity than children of the same age in the unfluoridated areas.

Further evidence can be drawn from Fig. 2. In 1983, the absolute value of caries prevalence in the Australian state of Queensland (which is only 5% fluoridated) was approximately equal to that in the states of Western Australia (83% fluoridated) and South Australia (70% fluoridated).

The classical British fluoridation trials at Watford and Gwalchmai were longitudinal controlled studies. In this regard they were better designed than the majority of other studies which have been conducted around the world. However, as in the case of almost all other surveys, the examinations were not 'blind'. The review of the British trials by the UK Department of Health after 11 years of fluoridation showed that children in fluoridated towns had approximately one less DMFT (that is, essentially one less cavity) than children of the same age in unfluoridated towns (see Fig. 3). The rate of increase in caries with age was the same in both populations[33].

Thus there are a number of counter-examples to the widely-held belief that "All studies show that communities where water contains about 1 p.p.m. fluoride have about 50% lower caries prevalence than communities where water has much less than 1 p.p.m. fluoride".

At this point the empirical data presented here may be summarized as follows. In the developed world:

(1) there have been large temporal reductions in caries in unfluoridated areas of at least eight countries;

(2) there have been large temporal reductions in several fluoridated areas which cannot be attributed to fluoridation;

(3) the absolute values of caries prevalence in several fluoridated areas are comparable with those in several unfluoridated regions of the same country.

Hence there is a case for scientific re-examination of the experimental design and statistical analysis of those studies which appear to prove or "demonstrate" that fluoridation causes large reductions in caries. Indeed the few re-examinations which have already been done confirm that there are grounds for concern.

The original justification for fluoridation in the United States, Britain, Canada, Australia, New Zealand and several other English-speaking countries was based almost entirely on the North American studies, which were of two kinds. The limitations of the first set, the time-independent surveys conducted in naturally fluoridated areas of the United States[26], have been referred to above.

The second set of North American studies consists of five longitudinal studies—carried out at Newburgh, Grand Rapids, Evanston and Brantford (two studies)—which commenced in the mid-1940s. Only three of them had controls for the full period of the study. These studies were criticized rigorously in a detailed monograph by Sutton[34], on the grounds of inadequate experimental design (for example, no 'blind' examinations and inadequate baseline measurement), poor or negligible statistical analysis and, in particular, failure to take account of large variations in caries prevalence observed in the control towns. The second edition of Sutton's monograph contains reprints of replies by authors of three of the North American studies and another author, together with Sutton's comments on these replies. It is difficult to avoid the conclusion that Sutton's critique still stands. Indeed, this was even the view of the pro-fluoridation Tasmanian Royal Commission[35]. Yet, in major, recent reviews of fluoridation, such as that by the British Royal College of Physicians[36], these North American studies are still referred to as providing the foundations for fluoridation, and Sutton's work[34] is not cited.

An examination has just been completed of the experimental design of all of the eight published fluoridation studies conducted in Australia. One (Tasmania) is a time-independent survey. Four (Townsville, Perth, Kalgoorlie and the second Sydney study) are longitudinal studies with only two examinations of the test group and either no control or only a single examination of a comparison group. The remaining three studies (Tamworth, Canberra and the first Sydney study) have several examinations of the test group, but no comparison group at all. Thus there has not been a single controlled longitudinal study in Australia. (M.D., to be published). Moreover, it has been shown above that three of the Australian studies (the first Sydney[4], Tamworth[14,15] and Canberra[16]) inadvertently provide evidence that some other factor(s) than fluoridation is/are playing an important role in the decline of caries prevalence.

Hence the hypothesis that fluoridation has very large benefits requires re-examination by epidemiologists, mathematical statisticians and others outside of the dental profession. The danger of failing to perform scientific research on the mechanisms underlying the large reductions in caries discussed in this paper is that the strong emphasis on fluoridation and fluorides may be distracting attention away from the real major factors. These factors could actually be driving a cyclical variation of caries with time[37]. It is possible that the condition of children's teeth could return to the poor state observed in the 1950s, even in the presence of a wide battery of F-treatments.

Causes of caries reductions

Many of the authors who reported the reductions in unfluoridated areas acknowledged that the explanation has not yet been determined scientifically[11,37–41]. It is after all much easier to perform a study which measures temporal changes in the prevalence of a multifactorial disease than to identify the causes of such changes.

Nevertheless, the authors of some of these studies have speculated that important causes of the reductions which they observe might be topical fluorides[38,53] (such as in toothpastes, rinses and gels), fluoride tablets[4,38], school dental health programmes[9], a lower *frequency* of sugar intake[39], the widespread use of antibiotics which may be suppressing *Streptococcus mutans* bacteria in the mouth[41], the increase in total fluoride intake from the environment[9,42], or a cyclical variation in time resulting from as yet unknown causes[37].

The present overview has revealed that several of the studies contain evidence against some of these proposed factors. We have seen that the Brisbane study[3] and

the Dutch review[11] suggest that fluoride tablets may not be important; the Sydney study[4], one of the British studies[10] and the Dutch review[11] each provides evidence against fluoride toothpaste; and the Dutch review[11] found no benefit in their school dental health education programmes.

Although there is evidence that fluoride toothpaste cannot be an important mechanism of caries reduction in some of the studies reported here, it must be stated that, unlike the case of fluoridation, there are also a few well-designed randomised controlled trials which demonstrate substantial reductions in caries from fluoride toothpaste[43]. Hence, the hypothesis can be made that topical fluorides sometimes improve children's teeth, although they are not necessary. So topical fluorides may comprise one of several factors contributing to the solution of the scientific problem of explaining the reduction in tooth decay.

Leverett[42] has speculated that the caries reductions in his smaller set of unfluoridated locations may be due to "an increase in fluoride in the food chain, especially from the use of fluoridated water in food processing, increased use of infant formulas with measurable fluoride content, and even unintentional ingestion of fluoride dentifrices." This hypothesis cannot explain the reductions in prefluoridation Sydney[4], or those in unfluoridated parts of Gloucestershire which started in the late 1960s[10]. The ingestion of fluoride toothpastes (and gels) by young children is well documented and could account for an intake of about 0.5 mg F^- per day in the very young[44]. But the food processing pathway is unlikely to be significant in western Europe where there is hardly any fluoridation, and infant formulas which are made up with unfloridated water will give only small contributions. Thus it appears that Leverett's hypothesis may at best be relevant to a minority of the studies listed in Table 1.

Here, the working hypothesis is presented that fluoridation and other systemic uses of fluoride, such as fluoride tablets, have at best a minor effect in reducing caries; that the main causes of the observed reductions in caries are changes in dietary patterns, possible changes in the immune status of populations and, under some circumstances, the use of topical fluorides. Indeed, a promising explanation is that the apparent benefit from fluorides is derived from their topical action. Then, since fluoridated water has a fluoride ion concentration 10^{-3} times that of fluoride toothpaste, its action in reducing caries is likely to be much weaker.

It is known that immunity plays a role in the development of caries, as it does with other diseases. Research is currently in progress to try to develop a vaccine against caries[45–47]. None of the data presented in the present paper provides evidence against immunity as a factor.

Dentists often argue against changes in dietary patterns as a major factor, on the grounds that sugar consumption has remained approximately constant in most developed countries over the past few decades. However, this is a simplistic argument. First, crude industry figures on total sales of sugar in developed countries contain no information on the distribution of sugar consumption with age and time of day. The form of sugar ingested—for example in canned food, soft drinks or processed cereals—may also be important. Second, tooth decay is increasing together with increases in sugar and other fermentable carbohydrates in the diet in several developing countries[48,49]. This was also the case with Australian aborigines, even when their water supplies consisted of bores containing fluoride at close to the "optimal" concentration for the local climate[50,51] Third, there is more to diet than sugar. For instance, there is some evidence, even conceded occasionally by pro-fluoride bodies[52], that certain foods which do not contain fluorides (for example wholegrain cereals, nuts and dairy products) may protect against tooth decay. So the whole question of the relationship between total diet and tooth decay needs much greater input from nutritionists and dietitians.

Perhaps the real mystery of declining tooth decay is why so much effort has gone into poor quality research on fluoridation, instead of on the more fundamental questions of diet and immunity.

The main body of this research was performed while the author was a principal research scientist in the CSIRO Division of Mathematics and Statistics, Canberra. □

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allowable concentrations of fluoride and the lack of a requirement for notification of fluoride content clearly compromises the utility of bottled water (as opposed to distilled water) as an alternative to fluoridated community water.

A final source of fluoride, or at least fluorine in some form, is from the air. This is largely due to trace amounts of pesticides and other industrial chemicals in the atmosphere. For the most part the fluoridated substances in the air are organic fluorides (as are some medications such as Prozac and Ciprofloxacin) rather than the fluoride ion found in water, dental products, foods, and beverages. Although our knowledge of the fate of fluorine from organic fluorides as the result of metabolism in the human body is very limited, it seems unlikely that the "fluoride" that comes from atmospheric sources adds significantly to the fluoride ion burden in humans.

Various estimates of the total fluoride exposure of individuals in the United States have been made, but the most comprehensive effort is probably that of an NRC committee (National Research Council, 2006). Tables 5.3 through 5.5, below, were constructed by the Fairbanks Fluoride Task Force from data in that report. The NRC committee's estimates of fluoride exposure from water were based on estimates of water consumption (EPA, 2000), which had been used in many of the studies considered by the committee. Because updated estimates of water consumption are now available (EPA, 2004), the task force substituted the updated estimates of water consumption and repeated the calculations used to construct Tables 5.3 through 5.5. The results are displayed in Tables 5.6 through 5.8.

Table 5.3. Estimated fluoride exposure (mg/kg body weight/day) of U.S. populations on water with 1.0 ppm fluoride, based on water intakes estimated in NRC (2006)

Population	water[a]	toothpaste[b]	background food[b]	pesticides & air[b]	total exposure[c]	% from water
Nursing infant	.0260		.0046	.0019	.033	79
Non-nursing Infant	.0860		.0114	.0019	.099	87
1–2 year old	.0314	.0115	.0210	.0020	.066	48
3–5 year old	.0292	.0114	.0181	.0012	.060	49
6–12 year old	.0202	.0075	.0123	.0007	.041	49
13–19 year old	.0152	.0033	.0097	.0007	.029	52
20–49 year old	.0196	.0014	.0114	.0006	.033	59
50+ year old	.0208	.0014	.0102	.0006	.033	63

a. Assuming all water, tap plus other, at 1.0 ppm
b. NRC (2006), Table 2-9
c. NRC (2006), Table 2-11

Table 5.4. Estimated fluoride exposure (mg/kg body weight/day) of U.S. populations on water with ***0.7 ppm fluoride,*** *based on water intakes estimated in NRC (2006)*

Population	water[a]	toothpaste[b]	background food[b]	pesticides & air[b]	total exposure[c]	% from water
Nursing infant	.0182		.0046	.0019	.025	73
Non-nursing Infant	.0602		.0114	.0019	.074	81
1–2 year old	.0220	.0115	.0210	.0020	.056	39
3–5 year old	.0204	.0114	.0181	.0012	.051	40
6–12 year old	.0141	.0075	.0123	.0007	.035	40
13–19 year old	.0106	.0033	.0097	.0007	.024	44
20–49 year old	.0138	.0014	.0114	.0006	.027	51
50+ year old	.0146	.0014	.0102	.0006	.027	54

a. Calculated from Table 5.3, assuming all water, tap plus other, at 0.7ppm NRC (2006)
b. NRC (2006), Table 2-9
c. NRC (2006), Table 2-11

Table 5.5. Estimated fluoride exposure (mg/kg body weight/day) of U.S. populations on water with ***0.3 ppm fluoride,*** *based on water intakes estimated in NRC (2006)*

Population	water[a]	toothpaste[b]	background food[b]	pesticides & air[b]	total exposure[c]	% from water
Nursing infant	.0078		.0046	.0019	.014	56
Non-nursing Infant	.0258		.0114	.0019	.039	66
1–2 year old	.0094	.0115	.0210	.0020	.044	20
3–5 year old	.0088	.0114	.0181	.0012	.040	22
6–12 year old	.0061	.0075	.0123	.0007	.027	23
13–19 year old	.0046	.0033	.0097	.0007	.018	26
20–49 year old	.0059	.0014	.0114	.0006	.019	31
50+ year old	.0062	.0014	.0102	.0006	.018	34

a. Calculated from Table 5.3, assuming all water, tap plus other, at 0.3ppm
b. NRC (2006), Table 2-9
c. NRC (2006), Table 2-11

Table 5.6. Estimated fluoride exposure (mg/kg body weight/day) of U.S. populations on water with ***1.0 ppm fluoride,*** *based on water intakes estimated by EPA in 2004*

Population	water[a]	toothpaste[b]	background food[b]	pesticides & air[b]	total exposure	% from water
Nursing infant	.017		.0046	.0019	.024	71
Non-nursing Infant	.055		.0114	.0019	.068	81
1–2 year old	.029	.0115	.0210	.0020	.064	45
3–5 year old	.026	.0114	.0181	.0012	.057	46
6–12 year old	.017	.0075	.0123	.0007	.038	45
13–19 year old	.014	.0033	.0097	.0007	.028	50
20–49 year old	.018	.0014	.0114	.0006	.032	56
50+ year old	.018	.0014	.0102	.0006	.030	60

a. Calculated from Table 5.3, assuming all water, tap plus other, at 1.0ppm
b. NRC (2006), Table 2-9

Major victory: Calgary city council votes to remove fluoride from water supply

Monday, February 14, 2011 by: Ethan A. Huff, staff writer

(NaturalNews) Calgary, Canada, with a metropolitan population of well over one million, will no longer lace its water supply with toxic fluoride. After much heated debate from both sides, the Calgary city council voted 10 - 3 to stop adding the hazardous chemical byproduct of the aluminum and phosphate fertilizer industries to the city's drinking water, ending more than 20 years of needless poisoning and scoring a significant victory for public health.

"It's an issue that has been debated vociferously around the world for 50 years," said Druh Farrell, the municipal council member that led the charge to remove fluoride. "[Fluoridation] became an established point of view, but now the wisdom of it is being questioned around the world."

Many smaller towns and cities have successfully resisted water fluoridation or voted to end it throughout the past several decades, but the fact that a large metropolitan city has now done it speaks volumes to the awakening that is taking place. Calgary's decision to cease fluoridation is key, as several other large cities throughout North America are right now considering doing the same thing.

"Fluoride is neither a nutrient nor required for healthy teeth," said attorney Paul Beeber, president of the New York State Coalition Opposed to Fluoridation, concerning New York's potential removal of fluoride. "Studies show fluoride ingestion doesn't reduce tooth decay."

In the U.S., efforts are currently underway in both New York City and San Diego to fight artificial water fluoridation. City councilman Peter Vallone of New York City has proposed a bill to end fluoridation in the Big Apple, which if successful will have huge implications for ending fluoridation throughout the U.S. And concerned citizens in San Diego continue to fight efforts by the city to expand fluoridation throughout Southern California (http://www.naturalnews.com/030933_f...).

Everyday citizens and concerned individuals need to continue to step up and speak out about fluoride if this mass poisoning is to end. Great victories are taking place, but they are all the more reason to step up the fight even more. Now is the time to vigorously attend city council meetings, present evidence to officials, and demand that the forced medication cease. The days of water fluoridation are numbered.

To learn more about fluoride, visit:
http://www.fluorideaction.net

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Fluoride & The Brain: An Interview with Dr. Phyllis Mullenix

The following interview, between Paul Connett & Phyllis Mullenix, took place on October 18, 1997. An edited video-taped copy of this interview can be obtained for $12 by emailing Fluoride Action Network. A magazine article discussing Mullenix's experience, can be accessed here. A pdf file of Mullenix's 1995 paper "Neurotoxicity of Sodium Fluoride in Rats" can be accessed here.

I. ACADEMIC BACKGROUND

Connett: *We're talking with Dr. Phyllis Mullenix, who in 1995, published a very important work on the neurotoxic effects of fluoride in rat studies. And Phyllis would you begin by telling us your background? What are your qualifications?*

Mullenix: Well, I got my PhD in pharmacology from the University of Kansas back in 1975. From University of Kansas Medical Center I went to John Hopkins School of Public Health in Baltimore between 1975 and 1977. And then in 1977 I was hired to come to Boston and work at Harvard with Dr. Herbert Needleman on the lead project. And so, I started then in 1977 and I've been in the Boston area for the past 20 years.

I was at the Children's Hospital in Harvard Medical School in the Psychiatry Departments and Department of Neuropathology at the Harvard Med School between 1977 and 1982. Then [in] 1982 I left and went to the Forsythe Dental Center in Boston. I went first into the Department of Pharmacology and then in 1983 we established the first toxicology department in any dental research institution in the world, in 1983.

Connett: *And, if I may interrupt, your task at that point, your brief as you understood it, was to examine the toxicological effects of the kind of materials that we're using in dentistry?*

Mullenix: Yes, Dr. Hein, who was the director of the institute at the time, wrote a nice newspaper article that was in the Forsythe Dental Center news in the spring of 1984 which described who I was and why I was brought in to the department and that I was brought in to head up this department to look at the environmental impact and the toxicity of products that are used by dentists and the dental community. And in particular they specifically mentioned fluoride, mercury, nitrous oxide, and some of those things.

II. NEUROTOXICITY OF FLUORIDE

Connett: *Ok. Could you briefly summarize your paper, and where was it published first of all?*

Mullenix: My paper concerning the neurotoxicity of sodium fluoride in rats was published in the *Neurotoxicology and Teratology* journal. That's a peer reviewed journal... And that was published in 1995. It was submitted in 1994, but it was published, it appeared on the shelf, in '95.

Connett: *After extensive peer review?*

Mullenix: That's right. As a matter of fact, it went through extra reviewers because the editor at the time recognized that this was a controversial subject and that to be on the safe side he suggested that they send it to an extra reviewer... and they took a good deal of time with it, and did it right.

Connett: *And what did you find?*

Mullenix: The study basically found three things. First of all, that if you put sodium fluoride in the drinking water of young animals, that with time - meaning a period of weeks in a rat's lifetime - they would develop changes in their behavioral patterns. And that pattern change was a hypoactivity pattern. They became slower, 'couch potatoes' if you like. But it was definitely a hypoactivity pattern. And it had a specific pattern to it which was very, very strikingly similar to the pattern that I had seen in substances or drugs that they used to treat acute lymphocytic leukemia in children, which clinically cause IQ deficits. And when I saw that specific pattern... that I was getting when I exposed animals to radiation or chemotherapy and steroids... that was very striking.

So, that was one thing - in young animals that were exposed, they became hypoactive.

I also found that if I started the exposure at a little later age, I would get the same pattern, but I would get it at a blood level of fluoride that was lower, even, than the young animals. So it suggested that, in particular females, that the older animal was more susceptible to this fluoride in the drinking water.

And a part of this whole common theme - what's happening at different ages - we also did a prenatal study. Because I wanted to see if I could do one specific exposure in the prenatal situation giving a subcu[taneous] shot of sodium fluoride at a specific age where a certain part of the brain is developing, if the fetuses of this mother, when they grew up, if they had any type of permanent behavioral damage.

And we gave the subcu[taneous] injections to the mother, we gave no other fluoride exposure, and when those pups were born and when they grew up and we tested them, they had a permanent change. And their pattern was this very distinct changes that are compatible with hyperactivity.

Connett: *Hyperactive.*

Mullenix: Right.

Connett: *So this is above, more active than usual?*

Mullenix: That's right. And some people would say, well doesn't it seem a little odd that if you gave the prenatal exposure you get a hyperactivity, and if you give a post-natal exposure you get a hypoactivity? And I say not at all. That's not unusual at all because the stage of brain development in the prenatal situation is extremely different from that in the postnatal situation. So there are different regions of the brain that are developing, therefore you've got different regions of the brain that are going to be susceptible. So it is not at all uncommon to have the long term outcome be strikingly different.

Connett: *And you also found that the fluoride accumulated in the brain tissue?*

Mullenix: Yes. Besides the prenatal exposures and the postnatal, the third thing that we wanted to look at was - what were the levels of fluoride in the brain? We had gone back in the literature, and it was said, I think it was Gary Whitford's studies that had said... that fluoride did not get across the blood-brain barrier and get into the brain to any extent. But I had a problem with that study, because what they did was they took fluoride and they gave an IV injection and then 1 hour later they looked at the levels in the brain.

But that's a far different cry from how people really get fluoride, they get it, you know, orally and day-to-day. And so, looking at fluoride levels in brain tissue 1 hour after injecting an IV does not mimic the real world situation at all...

So we went in with our drinking-water exposure, took out the brains - we dissected the brains in these animals into seven different regions - and then analyzed each region for the fluoride content. Now what we found was that, absolutely no question, there was major accumulations of fluoride in all the regions of the brain, and that some areas looked like there were greater accumulations than others, that were sex-determinant. That was a very interesting piece of information.

Just the fact that we could any level of fluoride at all, when we weren't expecting the brain to accumulate any fluoride, was a very big surprise and very, very disturbing to some people, of all things, that fluoride was accumulating in the brain.

[Note: At this point in the interview, Connett asked Mullenix questions concerning her relationship with Jack Hein, the Director of Forsythe, & Harold Hodge, a prominent expert on fluoride toxicology who oversaw Mullenix's work. To read this portion of the interview, scroll down to Section V ("The Manhattan Project Connection") or click here.]

III. REACTION TO MULLENIX'S FINDINGS

Connett: *Now, when you got these results, when it became apparent that fluoride, both prenatally and postnally, effected rat behavior, what kind of responses did you get from your institution and elsewhere?*

Mullenix: Well, there was two separate types of responses.

First of all, when I went to Jack Hein [the Director of Forsythe], and I said, look I think there's a problem with this stuff and I explained the data and everything, Dr. Hein got very excited. He thought this was extremely important. And he said, I want you to fly down to the National Institute of Dental Research and tell them your results. Forsythe paid for my trip down there.

I went there. It was in September of 1990. I'll never forget it.

Jack Hein also went with me, and he presented this to Harold Loe, who was then the Director of the National Institute of Dental Research, and I was to give this seminar.

Well just prior to my seminar, I walked over to the main corridor of the National Institutes of Health, and I walked in and all on the walls of this main corridor was this story called "The Miracle of Fluoride", all over the walls. And it had newspaper articles and artifacts, everything, from back in the 1940s and '50s, which described and made fun of the anti-fluoridation movement at that time. It called the people crackpots and it made jokes, it had stories about little old ladies in tennis shoes, you know, screaming about communist plots and everything else. And I'm very upset at this point because I knew how they made fun of people, about anti-fluoridationists, and I'm getting ready to walk into the National Institute of Dental Research and tell them that I thought that fluoride was lowering the IQ of children.

And so, I was really very shocked by that. I had no idea that there was that much political controversy.

So when I went in and I gave the seminar, I was amazed. The room was full. It was a small, private room. There were a lot of people from public relations there. There were a lot people from the public health service because they had, what looked like to me, a military uniform on. There was an individual from the Food & Drug Administration. And I proceeded to give my seminar, and I even made a joke about the little old ladies, and I said I'm a little old lady, but I don't have tennis shoes on. And nobody laughed. I mean there wasn't a single smile in the entire room. And they proceeded to really grill me on the technique and the technology, and basically, I had to be wrong.

Then, Jack Hein got a letter from the Director, Harold Loe, from the National Institute of Dental Research about two or three weeks later. And basically it was thanking him for the seminar, and he described me as being very enthusiastic, that the technology was extremely innovative, that it was very important that they get in and they look at this. And then he made suggestions of how the National Institute of Dental Research should follow up on this and provide money to do this research in the future.

And then that proceeded to start several tactics on how to get money; and they led me in circles for months, and then into years, of first following this procedure for getting the money through a contract and then I went down that road and then I found out that wasn't plausible. And...

Connett: *And your paper hasn't been published at this point?*

Mullenix: Oh, absolutely not. It was all preliminary data. And, then, at that time, I mean I wasn't real sure, I only had done a few experiments and so I said, yea, I really need to do more experiments, I need help to go forward. Because the only money to do this is whatever I came up with.

So, we felt obligated. I had this information. I was on the fence. I either had to go forward or I had to bury it. And I wasn't about to bury it.

I was academically into this because I'd been given a grant from the National Cancer Institute to look at the effects of neurotoxicity on the treatments for childhood leukemia. So, I was being praised on one side of the fence and how great the technique was, how sensitive it was. I'd been given a big grant to do that. And yet when I found the very same problem with this fluoride in drinking water, there all of a sudden they were questioning the technique, they were questioning me, and you know it was a completely different acceptance; that something had to be wrong with me. So here I was being applauded on one side and defamed on the other.

So at that time then at [Forsythe], the first time I really came out of the closet, so to speak, at the institution, other than Jack Hein - and I got the impression that Dr. Hein really didn't talk to very many faculty members about my work and fluoride research and what the results were, he kept that somewhat quiet - and I didn't talk about it until February of 1992.

And then when I stood up in the institution and I gave a seminar which told my results, the looks on the faces I'll never forget. It was almost complete horror. And I told them the dilemma I was in. I thought I was studying a control group and I wasn't expecting the answers I was getting [that fluoride was affecting the behavior of rats], and I was basically asking help.

What happened though was... 24 hours after that seminar, the Director of Research came up and talked to the second author on the paper that I was working with, Dr. Pam Den Besten, and basically said that, what would you think if we were not going to, as an institution, not going to allow you to publish this information?

Well, Dr. Den Besten got very upset that, you know, they started talking about ways of keeping us from publishing this information. That all of a sudden they were going to make these papers go through an approval system before they could be going into print.

Well, she came and told me and I got very upset about this because we'd never done this kind of thing. You write the paper, you send it out for peer review. You don't go through the institution. We're not like a government agency or something like that where you have to have approval by your institution before you can publish this information.

So I got very upset. This was on Friday. And, the Director of Research then started working on Dr. Den Besten, and finally she got very exasperated with the whole situation and said you go talk with Dr. Mullenix yourself.

So the seminar was on a Thursday, this on Friday - they were talking about not allowing me to publish it - and then on Monday, finally, the Director of Research came to me personally and sat down in my office. And said to me things, such as, first of all he said you have to do more studies because this can't be correct.

I said, I would love to do more studies. Help me do more studies. You know, maybe there was something we've done wrong. We need to do further studies. So help me.

The institution, he says, well we don't have, we can't give you support for doing this. He also said that you are jeopardizing the funds to our institution from the National Institute of Dental Research if you go forward and come out with this.

He said that I was going to cause hysteria on the part of the public and that they just didn't want me to go into this controversial area.

And there were some other remarks that these types of results and the way I was presenting it was an 'hysterical response.'

So I got very upset with this because I didn't exactly like being portrayed as an hysterical female all of a sudden when for ten years I'd been the Head of the Department and encouraged by the Director to even do these studies in the first place.

But unfortunately, at that time what happened was, my supporter and the reason that I did these studies - Dr. Hodge had died - and then Jack Hein went into retirement in 1991. And then what happened was the successors, the people, the Assistant Directors and Director of Research, then they became the Director. And at that time they had some consortium money from groups like Colgate, and whatever, and they were not in favor of the fluoride research at all.

Connett: *Well, never the less, you went ahead and you submitted this thing for publication?*

Mullenix: Yes.

Connett: *It got extensive peer review?*

Mullenix: Yes.

Connett: *And tell us what happened when it became known that it had been accepted for publication?*

Mullenix: I first got the acceptance over the telephone. I walked down to the administrators office at Forsythe and told them that the paper was going to be published. The assistant to the administrator said well we should notify the National Institute of Dental Research. Do you mind if we tell them? And I said, well, do you whatever you want, you know, I don't care.

And so they called Pat Bryant at the National Institute of Dental Research. Pat Bryant then proceeded to call me. And in several telephone conversations she basically asked me, she says, will you fly down to Washington and tell us what you found? And will you give us a copy of your paper before it had actually been finished with the peer review process? I said no, I won't give you a copy of the paper. I will be glad to talk about it however and tell you what the results are going to be. But before it had finished the peer review process and editing and everything, I didn't think it was appropriate to give this paper out...

So from the time that they had found out that this was accepted for publication to the time that they set up that television conference - which took place over at Harvard - was about three weeks all total. They paid for the television conference, they set it up, they had their people at their end and I think it was in Arlington, or some place in Virginia. And then we were at the Harvard campus at this end, and we had several people there. And we proceeded with about a two to three hour television conference where I explained the data.

Connett: *And you overheard something whilst they were setting these cameras up?*

Mullenix: Oh yes that was a funny part.

They didn't know that we could hear what they were saying, [since] they couldn't see or hear us. And we heard Pat Bryant, in fact several people heard on this end, heard Pat Bryant instructing the people at NIDR and other government people, she said don't make this an inquisition. We're trying to find out what she's going to say to the public so that we know what is about to be presented. And so she was really asking them several times not to make this an inquisition.

Well, it *was* an inquisition.

And, in fact, it was so much so that it was noticed on both sides, that about a week after the television conference [Pat Bryant] called up and she apologized to me, that it was an inquisition and that they were, you know, not very receptive. Which was odd because the group that they had collected down in Washington were, yeah there were some scientists that I recognized, but more important, the room was full of public relations people.

So I couldn't understand why, if you're going to listen to scientific data and this kind of thing, you've got public relations people there. I also did recognize one person from the Food & Drug Administration that was there, it was Dr. Tom [Zavotkin - sp?] and he asked a few questions and everything. And I talked with him subsequent to that and he thought it was because it was some kind of grant review and he thought that they were going to give me money, or I was asking for money to do research. And when he found out then that it was just because I was about to publish a paper, he was amazed, he was totally shocked. That they did this kind of thing.

As a matter of fact, even the people at Harvard said we're always trying to get Washington to pay attention to some of our works, where *we* have to pay for the television conference, *we* have to pay for it. How did you get the NIDR and NIH to pay for a television conference where [they're] coming to us?...

IV. FORSYTHE FIRES MULLENIX

Connett: *But despite this enormous interest in your work, which prompted the teleconference and so on, they still would not give you funding to continue this work?*

Mullenix: No. What was funny was after this presentation Pat Bryant on the phone said well the way to go forward here is a program project. And I said, yes, that would be nice, I think that would be a way to go forward. I said, however, I've got a bit of a problem - I've just been fired by Forsythe. And at the same time I'm doing the television conference in Harvard, they're moving my stuff out at Forsythe as fast as possible. In fact, I had to negotiate with the lawyers to delay moving my equipment out at least a month to give me some time.

Connett: *And what reason did they give you for sacking you from Forysthe?*

Mullenix: Well, let's just say that the reasons changed over a period of time through a lawsuit. Basically they said that I didn't get enough funds to do my research, number one, And, number two, the projects I worked on were not 'dentally related.' And that fluoride, they also that they weren't interested in that kind of science, to look into the safety of fluoride. They didn't consider that, well, as they put it, that's not 'their idea of science.'

Connett: *But since that time, two or three members, or at least two members of the Forsythe Center have got some very large grants from, from where? Tell us about that.*

Mullenix: Well actually I've heard that they have got some large grants from NIDR, yes. And... at the time, there was consortium money that went in from industries into Forsythe. I think they had one that was even in their newsletter about $250,000 from a couple of the industries and it was noteworthy that this money went to the individuals that actually were giving me a hard time about my fluoride research in the first place. So it was an unusual situation.

And when Pat Bryant at NIDR said oh you've got to do a program project, and then I said well that's going to be difficult because I've been fired and I have to move out, she basically said oh well then when you get an institution then we'll talk. Well I subsequently moved over to Children's Hospital. I did get an institution. I did put a grant in. And submitted it three times. And basically it went nowhere. Absolutely nowhere.

V. THE MANHATTAN PROJECT CONNECTION

Connett: *Ok, what I'd like to do now, is there were two people you were associated with at Forsythe Dental Center. There was Harold Hodge and Jack Hein.*

Mullenix: Yes.

Connett: *Could you tell me the background on these two people?*

Mullenix: Jack Hein was the director of Forsythe and he'd been the director for many, many years. But prior to being the director of the Forsythe Dental Center he was actually head of the dental laboratories I believe for Colgate. And he maintained the status of a consultant for Colgate for many years. But most importantly Jack Hein was the individual that was responsible for MFP.

Connett: *Monofluorophosphate.*

Mullenix: Right. And really the individual responsible for fluoride being put in toothpaste in the first place. So he had a very long history in the study of fluoride.

But more than that, Jack Hein was the student of Harold C. Hodge. And Harold Hodge was one of the founders of the Society of Toxicology. He was also one of the chief pharmacologists of the Manhattan Project, and in that Manhattan Project he had done a lot of the studies on toxicology of fluorides, in looking at the adverse effects that you could expect from fluoride exposures extending from the exposures to uranium hexafluoride.

So Jack Hein as a student of Harold Hodge's, and at the University of Rochester at the time when this is going on, he actually did experiments under Harold Hodge's supervision.

And, then when I came into Forsythe, Jack Hein wanted this toxicology department to be set up - and I felt it was a great thing to do - Jack Hein suggested that Harold Hodge was retiring from his current professor position, and that it would be a great thing if we could get Harold Hodge to retire and come to Forsythe and join in our department and become a part of the toxicology department.

So Harold Hodge came in 1983 to the Forsythe Dental Center and became a member of the toxicology department that I was made the head of.

Connett: *And he was pretty famous at that time as being one of the gurus of fluoride. He's written books on fluoride.*

Mullenix: Oh, Dr. Hodge did all of the research during the Manhattan Project, was responsible for directing all of the studies, he's published major works, is known internationally... [and] was responsible for the data that was used as the basis for all of the fluoridation projects in this country. He's written a book on Fluorine Chemistry. You will see his works through a lot of publications through the Atomic Energy Commission. So, yes, he was very much connected with the fluoride issue.

Connett: *But it's only much, much later that you have discovered that he had, in fact, proposed looking at fluoride's impact on rat brain, or rat behavior, many years before.*

Mullenix: Yes, now that was a real shock to me. Because, when I was at Forsythe in 1983, and one of the reasons I was brought over from Children's Hospital to Forsythe was because they knew what I was doing - I was working on developing a computer pattern recognition system with Dr. William Kernan, a physicist, and we were trying to develop this system and we had explained what we were going to use it for - how it was an objective measurement, how it could be applied - and Dr. Hein thought this was a fabulous thing, and the institution really supported and pushed this.

And so, as we were working on this computer system, Dr. Hodge would come up every day, and see how we were doing, see how we were progressing. He would ask multiple questions about how we did this and how we did that...

But he didn't say anything about any particular knowledge about fluoride and the effects on the central nervous system. The only thing he talked about, at that time, was about how weird it was during the Manhattan Project, how one scientist couldn't talk to another scientist and go from one laboratory to the next. But he never said anything that he knew that fluoride would affect the central nervous system. Not in those seven years between 1983 and 1990 when he died.

So I was shocked then in 1996, after Dr. Hodge's death, that some investigative reporters, Joel Griffiths and Cliff Honicker, presented to me various documents, declassified documents. And of the documents in this whole series of papers was a request from Harold Hodge to the military, or Colonel Stafford Warren, saying something to the effect that they wanted money to do studies to look at the effects of fluoride on the central nervous system in an animal model.

And they specifically stated that they had evidence, clinical evidence, that fluoride would cause confusion, drowsiness, lassitude, and that they were afraid that workers who worked with uranium hexafluoride were going to become a danger, either to themselves or to other people that they were working with, if they should have their brains effected by fluoride, and that they thought that this is something that should be examined.

So they asked for money to set this up. The military gave them money to do this project, they set up a budget. And then there was another document, not six months later, saying to stop these studies, or if they've started them, or haven't started them, not to start them.

And so I saw this series of documents. I was totally shocked. Because that told me for the first time that Harold Hodge knew that fluoride affected the central nervous system. And yet, I thought, and he led me to believe, or they let me believe, that I was doing something that had never been done before, that there was no connection between fluoride and the central nervous system, that it all went into the bone, and it [didn't] affect the central nervous system.

So when I saw those documents I called up Dr. Hein. I asked him, I said did Harold Hodge ever tell you that he's done studies on the effects of fluoride on the central nervous system, and that he tried to do a study in animals like I did, published in '95, that he tried to do that fifty years ago, and that it was stopped?

And Dr. Hein told me no, that he didn't know anything about that. I asked him if he knew anything about the existence of these documents, and he said no, that Dr. Hodge never mentioned anything.

Connett: *That's an amazing story. So he had to wait fifty years to see the outcome of an idea...*

Mullenix: Of an idea that was from fifty years ago.

And I don't know why, I mean I have no idea if that was the reason that they brought a neurotox person in to study in a dental institution, and let them develop a computer pattern recognition system that was really quite risky at the time, and very expensive to do, to let me take the time, to let me put resources into that, and then study fluoride? I mean, it kind of boggles the imagination, how this connection was made.

VI. CONCLUDING REMARKS

Connett: *Well, Phyllis, to wrap up, how do you see... explain all of these strange things happening to you?*

Mullenix: I wish I understood it. I really don't.

I feel like, number one, I was betrayed. That Dr. Hodge never explained to me that he knew that fluoride affected the central nervous system. Perhaps he was signed to

secrecy because it was the Manhattan Project. Perhaps not. I don't know. I will never know. No one will know...

Why they would say that I didn't work on anything dentally related when I published studies on nitrous oxide - laughing gas - it's used by dentists. Why they said fluoride wasn't a dental related issue. Why safety of fluoride didn't make my research relevant at a dental institution. There's no explanation for this. And the explanations went all around.

And then they said well I didn't get enough funds for my research. Except that, when I went to file another grant application, the then director, acting director of the institution refused to sign the grant. So, on one hand, they criticized me for not having money, but then they wouldn't sign my grant, you know, to go and get another grant money. And it was all very bizarre.

So I couldn't tell you, really, what the reasons are why I was fired.

Connett: *But clearly, clearly the National Institute of Dental Research and others in Washington were very, very concerned of the ramifications of...*

Mullenix: Oh, absolutely. Even after [the television conference], there were phone calls from the ADA; there were phone calls from Pat Bryant, will you please give us a copy of the paper? And I still wouldn't do it until all of the editorials, and all of the peer review, was totally done. And that television conference took place in early June [1994], and I was still getting phone calls into the fall of that year trying to get their hands on the paper. And I didn't actually put the paper in their hands until like January, when it was already committed that it was going to come out in a journal.

Connett: *Obviously you sensed that they were going to try sabotage that in some way?*

Mullenix: I was nervous. It was inappropriate to be demanding copies of a paper before it was finished with a peer review system. I wasn't about to have that happen. Both the NIDR and the ADA wanted copies of this paper before.

Also, right after that television conference, they asked for a copy of every thing I'd written on this technique, all the substances that I studied. And I sent down a whole packet, almost my entire publishing career to them for their review and they looked through it.

Connett: *And, finally Phyllis, before this happened, would you have described yourself as an environmentalist?*

Mullenix: Oh my, no. [laughs]

Connett: *Ok, would you have...*

Mullenix: As a matter of fact, I mean, I did a lot of consulting for industry. I was a laboratory scientist and I really didn't get into any political...

Connett: *An activist, would you have described yourself as an activist?*

Mullenix: Oh my word no. I was a bench scientist. I liked working with my rats. I still like working with my rats. I prefer them, they make more sense some times than people. [laughs] And I would like to go back to doing the rat [studies], because this whole thing has been totally destructive.

So I wouldn't consider myself an activist at all. Even today, after what I've gone through. I wouldn't consider myself an activist. I just simply want to do the research that obviously needs to be done in this situation. And I'd like to go back in. But I can not get the approvals. I can not get the support or the funding, or even the approval by institutions that this research can go forward. Because there's simply no money to do

it. And no help from the government to go forward with this issue.

Connett: *Phyllis Mullenix, thank you very much.*

Mullenix: Thank you.

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WATER FLUORIDATION

by Dr. Lawrence Wilson

I recently read over a hundred studies on both sides of the water fluoridation issue. Here is a summary of the findings, along with updates. I have also reposted a more recent article by Kelleigh Nelson at the end of this article and a recent posting by Dr. Mercola from the ***Fluoride Action Network***, a very worthwhile association.

UPDATE, AUGUST 12, 2011

A recent medical article documented how fluoridation reduces IQ. This article should be enough to stop fluoridation dead in its tracks. This may be far more important than tooth decay. Here is the link: http://articles.mercola.com/sites/articles/archive/2011/08/12/fluoride-and-the-brain-no-margin-of-safety.aspx?e_cid=20110812_DNL_art_1.

FLUORIDE AND TOOTH DECAY

Tooth decay has indeed decreased around the world. However, fluoride is not the cause. Tooth decay has declined as much or more in non-fluoridated areas as in those with fluoridation. This has been reported in Germany, British Columbia, New Zealand, Cuba, Finland, and the United States. Some small studies show slight benefits of fluoridation. One American study of 39,207 children showed reduced decay of deciduous teeth in 5-year olds in fluoridated areas compared with unfluoridated areas, but no reduction in decay in permanent teeth. The larger studies worldwide show little or no dental benefits of water fluoridation.

The Journal Fluoride, Vol. 27, #1, 13-22, 1994 reported that in a study of 98% of the children in New Zealand over 14 years, fluoridation had no dental benefit on these children. In fact, non-fluoridated communities had slightly less decay. Tooth decay

correlated with the level of income and nutrition, not fluoridation.

A study of over 400,000 children in India also showed no benefit of water fluoridation. Studies in England and Scotland found the same result. Dr. Albert Schatz, discoverer of streptomycin, found the same thing in Chile in a study spanning 40 years. A recent study in Tucson, Arizona by Dr. Cornelius Steelink, University of Arizona, showed an increase in decayed, missing or filled teeth with increased fluoride in the drinking water.

As a result of these and other studies, almost all major nations except the US and Britain have stopped fluoridating. 98% of Europe has stopped it. Ireland, one of the few European nations still doing it, is considering discontinuing it. Canada has advised against giving it to children less than 6 years old. India, China and Japan studied it thoroughly and discontinued it.

FLUORIDE SAFETY

Fluorine is a highly toxic element. Proponents say it is a nutrient. Scientists are mixed on this point. If it is a necessary nutrient, only a trace amount is needed in the body. We get this from foods such as tea, and small amounts are found naturally in most drinking water.

Sodium fluoride, the chemical used in water fluoridation, is a cumulative toxin. It is sold as rat poison, used in pesticides, and is the active ingredient in Saran nerve gas. Fluoride tablets require a prescription, unlike any other nutrient mineral. All fluoride toothpaste comes with a warning label. The label states "Keep out of the reach of children under 6. If you swallow more than used for brushing, seek professional assistance or contact a poison control center immediately." This warning applies to anything greater than a pea-sized drop of toothpaste on your brush.

Fluoride is considered one of the worst, if not the worst airborne pollutant, responsible for decimating fish and wildlife populations. The United States is one of 22 nations that signed a treaty promising not to dump fluorides into the oceans, lakes or rivers.

In the doses that fluoridated water provides, fluoride is associated with higher rates of birth defects, cancer and immunosuppression, lower IQ of children, dental and skeletal

fluorosis, and neurological problems. It has also been shown to cause increased bone fractures, cataracts and infant mortality, and some 20 other health effects. A study in Brain Research, vol. 784:1998 showed that fluoride in the water fed to rats increased the absorption of aluminum into the rats brains, causing alterations in the brains similar to Alzheimer's Disease. Also, fluoride is highly corrosive. Several studies in Massachusetts and elsewhere found higher levels of lead in the drinking water in fluoridated areas. This is most likely due to corrosion of lead pipe joints as a result of the corrosive chemical.

A new study also showed that fluoride accumulates in the pineal gland. This is a hormonal control center and causes severe imbalances in some sensitive people. The work, titled *Fluoride Deposition In The Aged Pineal Gland* was done as a PhD thesis by Jennifer Luke and published in *Caries Research*. It can also be found at www.fluoridealert.org.

The fluoride itself isn't the only problem. The chemical used to fluoridate is not pure. Hydrofluosilicic acid and sodium fluoride are industrial wastes, by-products of the phosphate fertilizer industry. This was challenged by the fluoride promoters in Ohio, but later they were forced to admit this is the truth. They contain traces of lead, arsenic, mercury, kerosene, napha, and other pollutants from the smokestack scrubbers of phosphate factories. They also contain radioactive elements.

WE ALREADY INGEST TOO MUCH FLUORIDE

Fluoride is now in the food chain, thanks to 50 years of water fluoridation, fluoride in pesticides, and airborne pollution. As a result, people are already getting more than the recommended 1 mg per day just from foods and beverages. Fruit juices, baby foods, and other select items are particularly high, due to processing and pesticide residues.

As a result, we don't need more fluoride. Dental fluorosis, or fluoride toxicity, is a growing problem. An article in the British Medical Journal, Aug. 26, 2000;189:216-220 reported that 54% of the children living in fluoridated areas have signs of fluorosis.

http://www.nap.edu/openbook.php?record_id=11571

In 1992, speaking on the Canadian television program *Marketplace*, former United States Environmental Protection Agency scientist Robert Carton claimed that "fluoridation is the greatest case of scientific fraud of this century." The practice was described as the "longest running public health controversy in North America" in the broadcast.[33]

1. ^ "Looking back at 40 years of fluoride" (*Marketplace*, Canadian Broadcasting Company, *11-24-92*) http://archives.cbc.ca/programs/481-1844/page/1/

A 2001 study found that "fluoride, particularly in toothpastes, is a very important preventive agent against dental caries," but added that "additional fluoride to that currently available in toothpaste does not appear to be benefiting the teeth of the majority of people."[35]

Dr. Geoffrey Smith, Dental Surgeon, *New Scientist*, May 5, 1983
"Dental Fluorosis, no matter how slight is an irreversible pathological condition recognised by authorities around the world as the first readily detectable clinical symptom of previous chronic fluoride poisoning. To suggest we should ignore such a sign is as irrational as saying that the blue-black line which appears on the gums due to chronic lead poisoning is of no significance because it doesn't cause any pain or discomfort."

Dr. P.H. Phillips, biochemist, University of Wisconsin
"Fluoride is an accumulative poison which accumulates in the skeletal structures, including the teeth, when the body is exposed to small daily intakes of this element. ...it is like lead accumulation in the bone until saturation occurs and then lead poisoning sets in."

FREEDOM OF CHOICE

Several nations, including Germany, stopped fluoridating because they realized it is immoral to mass medicate the entire population, especially when fluoride tablets, drops, toothpaste and other preparations are inexpensive and readily available.

In 1992, the Safe Water Foundation filed suit against the city of Fond DuLac, Wisconsin (Wisconsin Appellate Case #93-2275). They showed that fluoridation is mass medication of the population with a controlled substance, without the knowledge or consent of the participants. Even for mental patients, consent is required for medical treatment. Additionally, the dosage is not regulated, because some people drink more water than others.

Wealthy people can afford bottled water or expensive reverse osmosis filters to take it out. The poor are forced to drink the medicated water. The poor suffer the most from the toxic effects of fluoride.

LEADING EXPERTS CHANGE THEIR MINDS

IMPORTANT

Dr. Hardy Limeback, DDS, PhD, is head of the Department of Preventive Dentistry at the University of Toronto, and president of the Canadian Association for Dental Research. He is Canada's leading fluoride authority, and until recently the nation's primary fluoride promoter. Two years ago he changed his mind. He publically apologized for 15 years of misleading the people of Canada on the issue of fluoridation. Dr. Limeback said that Toronto, fluoridated for 36 years, has a higher incidence of cavities than Vancouver, which has never fluoridated their water. He said the Centers for Disease Control are basing their fluoride recommendation on 50-year-old studies that don't reflect new research.

Dr John Colquhoun was the Principal Dental Officer for Auckland, the largest city in New Zealand, and a staunch fluoridation advocate - until he was given the task of reviewing the world-wide data on fluoride effectiveness and safety. His review is titled "Why I Changed My Mind About Fluoridation". In it, he details how data was manipulated to support fluoridation in the English-speaking countries.

Dr. Phyllis Mullinex was commissioned by the US Army, MEDCOM to research possible neurological effects of fluoridating the water supply at Fort Detrick, Maryland. She worked at Harvard University Dental School. She expected a routine investigation. However, her results shocked even herself when she found that rats fed fluoride developed a variety of neurological defects. She was forced to advise the army not to fluoridate - and lost her job as a result.

Perhaps the most incredible turnaround is by the Environmental Protection Agency's Union of Scientists and Engineers. In May 1999, they announced they oppose their own agency's stand on water fluoridation. Senior vice-president of the union, Dr. William Hurzy wrote, "recent, peer-reviewed toxicity data, when applied to EPA's standard method for controlling risks from toxic chemicals, require an immediate halt to the use of the nation's drinking water reservoirs as disposal sites for the toxic waste of the phosphate fertilizer industry".

WHAT DO THE DENTISTS SAY?

I debated the head of the dental society when Phoenix considered fluoridation in 1990, and participated recently in the debate in Wooster, Ohio. The dentists and public health officials did not independently review the recent research. Instead, they continued the refrain that everyone knows fluoride is safe, reduces tooth decay 35-60%, and is worth imposing on everyone as a public health measure.

When challenged, they resorted to character assassination of anyone who does not agree with them. Their basic argument is - trust us and stop asking questions!

WHAT CAUSES TOOTH DECAY?

Dental hygiene and dental care are certainly important factors. A recent review of tooth decay in the Journal of the American Dental Association, July 2000 suggests that fluoride plays a role, but works topically. However, the decline in tooth decay rates worldwide, regardless of fluoridation and before fluoride toothpaste came into widespread use, calls this into question.

Many studies show that poor nutrition, especially consumption of mineral-deficient foods and sugars, negatively affect the teeth. Although largely composed of calcium and phosphorus, many minerals are needed for the teeth. These include zinc, copper, manganese, boron, vanadium and others. Vitamin C helps build the collagen matrix that bones grow within. Vitamin D is very important, and others like vitamin A may also play a role. Refined foods are deficient in minerals and vitamins.

Weston Price, DDS did extensive research on tooth decay around the world. His book, *Nutrition and Physical Degeneration*, is a classic on dental disease and nutrition. He found that wherever refined, canned and other processed food replaced traditional diets, tooth decay became a major problem. Although some people are more disposed to dental problems that others, it is not a genetic difference. Dr. Price was able to show that in one or two generations the teeth among many groups deteriorated from the use of refined food diets.

CONCLUSION

Except for trusting some dentists and public health officials who may be well-intentioned, but lie and attempt to demolish the character of their opponents, I can find no reason to recommend water fluoridation. You can read the material for yourself and make up your own mind. Your health may depend on it. Wooster, Ohio voted down fluoridation in the 2000 elections. Flagstaff, Arizona also rejected fluoridation in 2001.

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Here is another article about fluoridation with some additional information:

AMERICAN CITIZENS AS GUINEA PIGS

By Kelleigh Nelson September 12, 2010

Fluoride

In the movie, *Conspiracy Theory*, Mel Gibson says, "You know what

they put in the water don't you? Fluoride! Yeah, fluoride, on the pretext that it strengthens your teeth. That's ridiculous. You know what this stuff does to you? It actually weakens your will, takes away the capacity for free and creative thought, and makes you a slave to the state."

It turns out that statement is factual and true. The movie "Conspiracy Theory," used several absolutely true statements about subjects most constitutionalists are aware of, in a way that "neutralized" the actual facts. The above statement repeated shortly after the movie would be answered with, "Oh, that was in that Conspiracy movie, it's not really true." This is a typical tactic of the corporate powers that be and the nanny state, and serves only to negate the truth.

What is sodium Fluoride?

First of all, there is no such substance as "Fluoride" listed in the periodic chart of elements, nor in the Merck Index. Instead, a GAS called fluorine is listed. The use of this gas in various industries such as aluminum manufacturing and the nuclear industry create certain toxic byproducts which have "captured" fluorine molecules. Fluoride waste products are also derived from the industrial manufacture of zinc, uranium, aerosols, insecticides, fertilizers, plastics, lubricants and pharmaceuticals. One such toxic, poisonous "byproduct" is called sodium Fluoride, which according to the Merck Index is primarily used as rat and cockroach poison and is also the active ingredient in most tooth pastes and as an additive to drinking water.

Fluorine is the most highly reactive, and chemically unstable of all existing chemical elements. Fluorine is not found by itself in nature because it is so unstable that it chemically combines, violently in many cases, with practically any other element. Fluorine has the strongest effect of all the halogens (Bromine, Chlorine, Fluorine). Halogens are the non-metallic elements. Fluoride is one of the major ingredients in the controversial psycho-active psychiatric drug, PROZAC which is used so prevalently in the UK that it is now in the water and the filtration systems cannot remove it. And sodium Fluoride is also in the deadly Sarin military nerve gas, designated Isopropyl-Methyl-Phosphoryl Fluoride.

Alcoa Aluminum in Alcoa, TN, near Maryville, is only about 20 minutes from where I live. Fluoride is used to make aluminum. Many years ago

I spoke to my veterinarian regarding our dogs and teeth cleaning. Our dogs get their teeth brushed every night with a chicken flavored dog toothpaste that has NO FLUORIDE in it. Why? Because if the dogs swallowed fluoride on a daily basis as they do with the chicken flavored paste, it would kill them. Occasionally, I would have the veterinarian clean our dogs' teeth while they were under anesthesia. Our vet asked if we wanted a fluoride treatment on their teeth while they were under. I immediately went out and bought a copy of Christopher Bryson's THE FLUORIDE DECEPTION and gave it to my vet. (This book is available from News With Views and I highly urge you to purchase a copy.) Under no circumstances did I want fluoride used on our pets. In discussing Alcoa Aluminum and their use of fluoride in the manufacture of aluminum products, the vet commented that he'd seen fluorosis in the teeth of cattle near Alcoa where the water is loaded with fluoride. Fluorosis is an irreversible condition caused by excessive ingestion of fluoride during the tooth forming (and bone forming) years which damages the enamel forming cells. It also has destructive effects on the rest of the body.

At the time when sodium Fluoride was first put in the water in Grand Rapids, Michigan in 1945, there were no fluoridated products available commercially. Crest introduced its fluoridated toothpaste in 1955, and Colgate added fluoride in 1967. We now have so many fluoridated products, from our toothpaste to floss and mouthwash, which many choose to use topically. We should be asking, "Why do we need toxic chemical byproducts in our water and all these products?" Check your toothpaste tube, it says in bold, do not swallow. It says this on toothpaste tubes, because toothpaste (fluoride) should most definitely not be swallowed (ingested).

So the question that begs to be asked, why would we think that it is okay to drink (ingest) fluoride, but not swallow toothpaste? The answer is, clearly, it is not okay. There is absolutely no proof that fluoridated water contributes to the dental health of American children.
Despite dental pressure, 99% of western continental Europe has rejected, banned, or stopped fluoridation due to environmental, health, legal, or ethical concerns. Only about 5% of the world population is fluoridated and more than 50% of these people live in North America. Here is a recent news report on Fluoride from Australia.

When was it first used?

This insanity of putting sodium Fluoride into drinking water had its first occurrence in the German ghettos and in Nazi Germany's infamous concentration camps. The Gestapo had little concern about dental hygiene or the effect on teeth. Their reason for the mass medicating of water with sodium Fluoride was to sterilize humans and force the people in their prison camps into calm, malleable, submissive and docile attitudes. "The Crime and Punishment of I.G. Farben," by Joseph Borkin goes into detail regarding this very early practice. If you can find a copy, buy it!

Who does it benefit?

Sodium Fluoride is quite expensive for the worlds' chemical companies to dispose of, but in the 50s and 60s, Alcoa Aluminum and the entire aluminum industry (who had an overabundance of the toxic waste) somehow sold the FDA and our government on the insane and highly profitable idea of buying this poison at a 20,000% markup and injecting it into our water supply and dental rinse.

Of course, much of the water goes down the drain, but we also bathe in it and it is absorbed by our skin. The chemical industry and others have not only a free hazardous waste disposal system, but we also pay them handsomely in the process with taxpayer dollars.

Professor Kaj Roholm, former Chief of the Toxicology Committee for the National Research Council classifies hydrofluorosilic acid and hexafluorosilic acid as "extremely toxic." One chemical company selling fluoride to water suppliers describes it as "a colorless to straw yellow, transparent, fuming, corrosive liquid with a pungent odor and irritating action on the skin."

Dr. Phyllis Mullenix

In 1995, Dr. Phyllis Mullenix published a very important work on the neurotoxic effects of fluoride in rat studies. Dr. Mullinex got her PhD in pharmacology from the University of Kansas in 1975. From University of Kansas Medical Center she went to John Hopkins School of Public Health in Baltimore between 1975 and 1977. In 1977 she was hired to work at Harvard with Dr. Herbert Needleman on the lead project. She was at the Children's Hospital in Harvard Medical School in the Psychiatry Departments and Department of Neuropathology at the Harvard Med School between 1977 and 1982. Then, in 1982 she went

to the Forsythe Dental Center in Boston, first into the Department of Pharmacology and then in 1983 the first toxicology department in any dental research institution in the world was established. She was brought in to head up this department to look at the environmental impact and the toxicity of products that are used by dentists and the dental community. And in particular they specifically mentioned fluoride, mercury, and nitrous oxide.

Her paper concerning the neurotoxicity of sodium fluoride in rats was published in the *Neurotoxicology and Teratology Journal*, a peer reviewed journal in 1995. The study basically found three things. First of all, sodium fluoride in the drinking water of young animals, showed that with time - meaning a period of weeks in a rat's lifetime - they would develop changes in their behavioral patterns, and that pattern change was a hypo activity pattern. They became slower, like 'couch potatoes.' It had a specific pattern to it which was very strikingly similar to the pattern that she had seen in substances or drugs that they used to treat acute lymphocytic leukemia in children, which clinically cause IQ deficits. They also looked at fluoride levels in the brain an hour after IV injections, but Dr. Mullinex felt this did not mimic properly what the population of America was ingesting in their drinking water. What they found after a test on the animals drinking fluoridated water was that there were major accumulations of fluoride in all the regions of the brain, and that some areas looked like there were greater accumulations than others, that were sex-determinant.

Harold Hodge was one of the founders of the Society of Toxicology. He was also one of the chief pharmacologists of the Manhattan Project, and in that Manhattan Project he had done a lot of the studies on toxicology of fluorides, in looking at the adverse effects that you could expect from fluoride extending from the exposures to uranium hexafluoride. (Fluoride was used to make the atomic bomb). Hodge talked about how weird it was during the Manhattan Project, how one scientist couldn't talk to another scientist and go from one laboratory to the next. But he never said anything to Dr. Mullinex that he knew fluoride would affect the central nervous system.

In 1996, after Dr. Hodge's death some investigative reporters, Joel Griffiths and Cliff Honicker, had various declassified documents, and of the documents in this whole series of papers was a request from Harold Hodge to the military, saying something to the effect that they wanted money to do studies to look at the effects of fluoride on the

central nervous system in an animal model. They specifically stated that they had evidence, clinical evidence, that fluoride would cause confusion, drowsiness, lassitude, and that they were afraid that workers who worked with uranium hexafluoride were going to become a danger, either to themselves or to other people that they were working with, if they should have their brains effected by fluoride, and that they thought that this is something that should be examined. Hodge also was responsible for human radiation experiments that were discussed in part 4 and 5.

Dr. Mullinex gave her speech regarding her research on fluoride to the National Institute of Health (NIH), mentioned in part 2 of this series, in late 1990. The FDA was there, the National Institute of Dental Health and others. When she mentioned the results of her research on fluoride and the damage she saw, the audience attacked her with questions regarding her research methodology. Eventually she lost her job because of what she exposed. But then she exposed those that fought her and the results of her research.

Many honest scientists have attempted to blow the whistle on sodium fluoride's false propaganda campaign. They have ended up being "black-listed," and their valid points disputing the vested interests of these mega corporations never get the press they deserve. If one follows the money the "control" behind this is prominent American families. The 1952 campaign to convince the public of fluoride's benefits was rammed down our throats by the Public Health Departments and various dental organizations. Sadly, sodium Fluoride has now become usual and common and is even offered in dental offices as specialized treatments for "sensitive teeth" where the gums have receded.

Side effects of sodium Fluoride poison

Independent scientific research evidence over the past 60 plus years has shown that sodium Fluoride shortens our life span, promotes various cancers and mental disturbances, and makes humans stupid, docile, and subservient, all easily done in the drinking water of Americans. There is also increasing evidence that aluminum in the brain is a causative factor in Alzheimer's Disease, (something we rarely saw with the epidemic proportions of today before the 50s). Evidence points towards sodium Fluoride's strong affinity to "bond" with this dangerous aluminum (remember it is a byproduct of aluminum

manufacturing) and also it has the ability to 'trick' the blood-brain barrier by imitating hydrogen ion thus allowing this chemical access to brain tissue.

In 1992, the American scientists, Robert Isaacson, Julie Varner, and Karl Jensen found that fluoridated water carried aluminum into rat brains, producing Alzheimer's-like changes in brain tissue. Phyllis Mullenix, who gave lab mice moderate doses of fluoride and generated symptoms resembling ADHD, fears that the high incidence of both diseases in the general population is direct evidence of Fluoride's toxic effects and that both the number and kind of such injuries may worsen in the coming years.

Arthritis, increased risk of hip fracture, Alzheimer's, heart disease from fluoride concentrates in the arteries which attracts calcium and can contribute to their hardening, thyroid problems, Down's Syndrome, breathing difficulties, reproduction problems, and other central-nervous-system disorders have all been linked by scientists to fluoride exposure.

Fluorides are cumulative toxins. The fact that fluorides accumulate in the body is the reason that U.S. law requires the Surgeon General to set a Maximum Contaminant Level (MCL) for fluoride content in public water supplies as determined by the EPA. This requirement is specifically for the purpose of avoiding a condition known as Crippling Skeletal Fluorosis (CSF), a disease that progresses through three stages. The MCL, designed to prevent only the third and crippling stage of this disease, is set at 4ppm or 4mg per liter. It was assumed that people retain half of this amount (2mg), and therefore 4mg per liter is considered "safe." However, a daily dose of 2-8 mg is known to cause the third crippling stage of CSF. See this link for historic use of Fluoride.

I would highly suggest the purchase of "The Fluoride Deception" by Christopher Bryson which is available from NewsWithViews. It is probably the most comprehensive book on the subject.

Of course, be sure you are not drinking fluoride. The only type of water to drink to avoid it, unfortunately, is natural spring water. Carbon filters unfortunately will not remove it. Avoid fancier water filters as they

seem to damage the water. Distilled and reverse osmosis water are also fluoride-free, but contain no minerals and should not be used for drinking water for this reason for more than a few months, at most. Never drink reverse osmosis water for any length of time, as it tends not to be well absorbed. Read Water For Drinking for more on this subject.

References

Excellent web sites are www.fluoridealert.org, www.nofluoride.com, and www.fluoridation.com. Of course, there are dental society and government sites that recommend fluoridation.

The following are among the best papers on fluoridation I have come across.

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Top Ten Arguments Against Water Fluoridation

1) Fluoridation is a violation of the individual's right to informed consent to medication.

2) Fluoride is not an essential nutrient. No biological process in animals or humans has been shown to depend on it. On the contrary, it is known that fluoride can interfere with many important biological processes and vital cellular constituents, such as enzymes and G-proteins. This makes fluoride potentially toxic even at low doses.

3) Children in fluoridated countries are greatly over-exposed to fluoride. When fluoridation began in 1940s, 10% of children were expected to develop dental fluorosis (damage to the enamel involving discoloration and/or mottling) in its *very mild* form. Today, the prevalence in fluoridated countries is much higher—41% of *all* American children aged 12-15 are now impacted with some form of dental fluorosis (CDC, 2010), with over 10% in categories (*mild, moderate* and *severe*) that may need expensive treatment.

4) The chemicals used to fluoridate water supplies are largely hazardous by-products of the fertilizer industry. These chemicals cannot be disposed of into the sea by international law, and have never been required to undergo randomized clinical trials for safety or effectiveness by any regulatory agency in the world. The U.S. FDA classifies fluoride as an "unapproved drug."

5) There is mounting evidence that swallowing fluoride causes harm. Fluoride has been found to damage soft tissues (brain, kidneys, and endocrine system), as well as teeth (dental fluorosis) and bones (skeletal fluorosis). There are now 24 studies that show a relationship between fairly modest exposure to naturally-occurring fluoride and reduced IQ in children. Two of these studies suggest that the threshold for damage may be reached at fluoride levels similar to those used in water fluoridation (http://fluoridealert.org/iq.studies.html).

6) Swallowing fluoride provides little or no benefit to the teeth. Even promoters of fluoridation agree that fluoride works topically (on the outer surface of the teeth), and not via some internal biological mechanism (CDC, 1999). A recent U.S. study found no relationship between the amount of fluoride a child ingested and level of tooth decay (Warren et al., 2009). Topical treatment in the form of fluoridated toothpaste is universally available, so it is a mistake to swallow fluoride and expose all the tissues of the body to its harmful effects.

7) Human breast milk is very low in fluoride. Breast milk averages only 0.007 ppm F (NRC, 2006). Even in areas with high fluoride levels, nursing children receive only a small fraction of the mother's fluoride intake, ensuring that the sensitive brains and bodies of breast-fed infants are protected from the developmental effects of this toxin. In contrast, a bottle-fed baby in a fluoridated area (0.7-1.2 ppm F) gets up to 200 times more fluoride than a breast-fed baby, resulting in an increased risk of dental fluorosis and other adverse effects.

8) There is no control of dose and no follow-up. Once fluoride is added to water, there is no way to control who gets the drug or how much is ingested. Nor has there been any systematic medical follow-up that would allow a picture of short-term or long-term side effects of the drug to be built up. These failings fly in the face of accepted medical practice.

9) Certain subgroups are particularly affected by fluoridation. People vary considerably in their sensitivity to any toxic substance, including fluoride. Infants, the elderly, diabetics, those with poor nutrition (e.g. low calcium and low iodine), and those with kidney disease are especially vulnerable to specific adverse effects of fluoride. Black and Mexican-Americans have a higher prevalence of the more severe forms of dental fluorosis (see Table 23, CDC, 2005).

10) Fluoridation discriminates against those with low incomes. People on low incomes are least able to afford avoidance measures (reverse osmosis or bottled water), or treatment of dental fluorosis (see Point 3) and other fluoride-related ailments (see Point 5).

For more information see the webpage of the Fluoride Action Network HYPERLINK "http://www.FluorideAlert.org/"www.FluorideAlert.org; the videotape, "Professional Perspectives on Water Fluoridation" and The Case Against Fluoride by Connett, Beck and Micklem (Chelsea Green, 2010).

discredit or ignore than the hundreds of earlier experiments, of varying quality and from around the world, that have linked fluoride to mottled teeth, skeletal damage, genetic defects and other ills. During the two-year experiment, rats and mice drank water with different levels of sodium fluoride. None of the animals drinking fluoride-free water developed cancer, nor did any of those drinking water with the lowest fluoride concentration, 11 parts per million (ppm).

But of the 50 male rats consuming 45-ppm water, one developed osteosarcoma. Four of 80 male rats drinking 79-ppm fluoride developed osteosarcoma. No mice or female rats showed signs of bone cancer. Although the animals drank higher concentrations of fluoride than people do (the legal standard is four ppm), such megadosing is standard toxicological practice. It's the only way to detect an effect without using an impossibly large number of test animals to stand in for the humans exposed to the substance.

Political decisions were at odds with expert advice

Although the final NTP report will not be released for months, several independent toxicologists find the results significant. Most important, the rats who did not drink fluoride did not get cancer, indicating that the malignancies are "not a fluke," says EPA scientist William Marcus.

the more fluoride, the more cancers

There is also a convincing relationship between dose and response: the more fluoride, the more cancers. Pathologist David Kaufman of the University of North Carolina warns that the rat data must be examined to see if the cancers appeared in the long bones of the arms and legs, as osteosarcomas do in humans, or in other places, which might make the results less relevant to people. Still, Kaufman says the NTP data "make fluoride look like a weak carcinogen...

If fluoride causes bone cancer in lab rats, then why, after 45 years of fluoridation, haven't researchers seen a rash of osteosarcomas in fluoridated cities? Because epidemiology is too crude to detect it even if the cancers are there. In the 1970s, the National Cancer Institute found no sign of higher cancer rates in fluoridated cities. But that reassuring finding may be misleading. According to Donald Taves, a fluoride expert, if the difference were anything less than 7 percent it would not be detectable. Another obstacle to definitive epidemiology is mobility: just because someone got osteosarcoma in a fluoridated city does not mean he had been living there all his life.

The NTP results assume an added importance when combined with recent data on the shrinking benefits of fluoridation. According to the American Dental Association (ADA), tooth decay is anywhere from 50 to 70 percent less in fluoridated areas. But figures from the National Institute of Dental Research (NIDR), part of the National Institutes of Health, suggest otherwise. A 1987 survey of almost 40,000 school children found that tooth decay had declined sharply everywhere. Children who had always lived in fluoridated areas had 18 percent less decay, compared with their peers who had lived in nonfluoridated areas. This 18 percent translates into a difference of fewer than one cavity per child. Similarly, in a 1986 paper in the British Journal Nature, Australian researcher Mark Diesendorf assessed **24 studies from eight countries and found that cavity rates had declined equally in fluoridated and nonfluoridated, areas, suggesting fluoridated water isn't that important.**

24 studies from eight countries and found that cavity rates had declined equally in fluoridated and nonfluoridated, areas, suggesting fluoridated water isn't that important.

How can that be? "A good case can be made that it has to do with fluoride in toothpaste and rinses," says dental-health expert Brian Burt of the University of Michigan. And even if drinking fluoridated water is slightly risky, there is no hint that fluoridated toothpaste-as long as you don't swallow any-is dangerous. Tooth decay may also be declining because of better diet and hygiene. Also, foods and beverages processed with fluoridated water are ubiquitous. (Many bottled waters, though, do not have fluoride.) As a result, argues Alan Gray, a leading pro-fluoridation dentist in Canada, "it is becoming difficult to provide accurate, ethical advice" about fluoridation.

Among environmental controversies, fluoridation is unique in that one side has consistently denied that questions of risk or benefit even exist. The ADA states, "Antifluoridation groups attempt to create the illusion of a scientific controversy [which is] merely a ploy to create doubt about a well researched, well-demonstrated preventive measure." But even well-researched articles raise hackles. **When, in 1988, Chemical & Engineering News presented a balanced report on fluoridation, it attracted the wrath of the medical establishment.** Says Taves, "Too many scientists lost their objectivity. This has become a religion on both sides. "

Safe water. And that undercut the scientific process. The NIDR kept files on people perceived as threats to fluoridation. **Political decisions were at odds with expert advice: a panel convened by the surgeon general in 1983 expressed concern, in closed sessions, about skeletal and dental damage from fluoride. At one point, a member said, "You would have to have rocks in your head, in my opinion, to allow your child much more than two parts per million [fluoride]." Said another, "I think we all agree on that." Even so, in 1986 EPA raised the fluoride standard from about two ppm to four.**

This month EPA opened a review of the standard. Once EPA receives the official NTP report, it will establish a target "safe" fluoride level. The Safe Drinking Water Act requires that the level be zero for carcinogens, but the standard may be based on what is technically feasible. Fluoridation can be stopped immediately, but many communities with naturally fluoridated water-up to 12 ppm-would have to remove it. As EPA wrestles with the standard, fears John Sullivan of the American Water Works Association, "confusion will reign": local laws will still require fluoridation, a practice that may cause cancer.

As they await EPA's decision, pro-fluoridationists are invoking arguments of social justice. Dental researcher Ernest Newbrun: of the University of California, San Francisco, contends that fluoridation promotes the health of children of "all races and all socioeconomic classes," not only those with enough money or discipline or access to the health system to take a fluoride supplement every day.

He and others is morally wrong not to provide the benefits of fluoride. Although the NIDR's and other surveys suggest that fluoride in toothpastes and dental rinses also ensures healthy teeth for those who use the products, those who do not might suffer.

No one can foresee how the fluoride debate will play out this time. But since the 1950s, the country's environmental consciousness has been heightened. In the end, deciding whether or not to fluoridate turns less on science than on values. The sheer weight of good research may finally, after four decades, begin to inform those judgments and even overwhelm the unscientific rhetoric that has characterized both sides of the debate for far too long.

Sharon Begley

Newsweek, Feb 5, 1990

SCIENCE

Don't Drink the Water?

Brush your teeth, but the fluoride from your tap may not do much good—and may cause cancer

Remember the great fluoride debate? Back in the 1950s, every voice of authority, from the U.S. Public Health Service to the PTA, supported adding fluoride to the water supply as an effective and totally safe way to promote healthy teeth. The only opponents seemed to be John Birchers and other extremists who regarded the scheme as a diabolical communist plot. In the years since, most of the nation's major cities fluoridated their water, and the issue appeared closed. No less an objective voice than Consumer Reports declared in 1978, "The survival of this fake controversy . . . represents one of the major triumphs of quackery over science in our generation."

In fact, the debate never ended. Now it may explode as never before, posing new challenges to medical dogma and giving parents one more thing to worry about. Government researchers have new evidence that casts doubt on the benefits of fluoridation and suggests that it is not without risk. The most incendiary results come from the National Toxicology Program (NTP), which in 1977 was ordered by Congress to determine whether fluoride causes cancer. This week NTP plans to release data showing that lab rats given fluoridated water had a higher rate of a rare bone cancer called osteosarcoma. According to a memo by the Environmental Protection Agency, "very preliminary data from recent health studies . . . indicate that fluoride may be a carcinogen."

Fluoridation proponents are already criticizing the NTP study, but it will be harder to discredit or ignore than the hundreds of earlier experiments, of varying quality and from around the world, that have linked fluoride to mottled teeth, skeletal damage, genetic defects and other ills. During the two-year experiment, rats and mice drank water with different levels of sodium fluoride. None of the animals drinking fluoride-free water developed cancer, nor did any of those drinking water with the lowest fluoride concentration, 11 parts per million (ppm). But of the 50 male rats consuming 45-ppm water, one developed osteosarcoma. Four of 80 male rats drinking 79-ppm fluoride developed osteosarcoma. No mice or female rats showed signs of bone cancer. Although the animals drank higher concentrations of fluoride than people do (the legal standard is four ppm), such megadosing is standard toxicological practice. It's the only way to detect an effect without using an impossibly large number of test animals to stand in for the humans exposed to the substance.

Although the final NTP report will not be released for months, several independent toxicologists find the results significant. Most important, the rats who did not drink fluoride did not get cancer, indicating that the malignancies are "not a fluke," says EPA scientist William Marcus. There is also a convincing relationship between dose and response: the more fluoride, the more cancers. Pathologist David Kaufman of the University of North Carolina warns that the rat data must be examined to see if the cancers appeared in the long bones of the arms and legs, as osteosarcomas do in humans, or in other places, which might make the results less relevant to people. Still, Kaufman says the NTP data "make fluoride look like a weak carcinogen. It's obviously something to worry about"—but not panic over. There are about 750 cases of osteosarcoma in the United States annually; even if fluoride caused all of them—an impossibility—the lifetime risk to any individual from drinking fluoridated tap water would still be only about one in 5,000.

Too crude: If fluoride causes bone cancer in lab rats, then why, after 45 years of fluoridation, haven't researchers seen a rash of osteosarcomas in fluoridated cities? Because epidemiology is too crude to detect it even if the cancers are there. In the 1970s, the National Cancer Institute found no sign of higher cancer rates in fluoridated

From the beginning, controversy: *In 1965, the protests reached the reservoir's edge*

UPI-BETTMANN NEWSPHOTOS

ROB NELSON—BLACK STAR

Fluoridation: *Atlanta's waterworks*

Fluoride Facts

- Fluoride—in water or toothpaste—helps teeth resist decay. It seems to work by redepositing calcium and other ions in tooth enamel, repairing and strengthening it.
- 53% of the U.S. population drinks water containing fluoride. 121 million people have artificially fluoridated water; 9 million drink from naturally fluoridated supplies.
- 41 of the 50 largest U.S. cities have fluoride in the water; those that don't include L.A. and San Diego.
- The legal standard for fluoride in drinking water is four parts per million; for toothpastes, 1,100 ppm.

cities. But that reassuring finding may be misleading. According to Donald Taves, a fluoride expert, if the difference were anything less than 7 percent it would not be detectable. Another obstacle to definitive epidemiology is mobility: just because someone got osteosarcoma in a fluoridated city does not mean he had been living there all his life.

The NTP results assume an added importance when combined with recent data on the shrinking benefits of fluoridation. According to the American Dental Association (ADA), tooth decay is anywhere from 50 to 70 percent less in fluoridated areas. But figures from the National Institute of Dental Research (NIDR), part of the National Institutes of Health, suggest otherwise. A 1987 survey of almost 40,000 schoolchildren found that tooth decay had declined sharply everywhere. Children who had always lived in fluoridated areas had 18 percent less decay, compared with their peers who had lived in nonfluoridated areas. This 18 percent translates into a difference of fewer than one cavity per child. Similarly, in a 1986 paper in the British journal Nature, Australian researcher Mark Diesendorf assessed 24 studies from eight countries and found that cavity rates had declined equally in fluoridated and nonfluoridated areas, suggesting fluoridated water isn't that important.

How can that be? "A good case can be made that it has to do with fluoride in toothpaste and rinses," says dental-health expert Brian Burt of the University of Michigan. And even if drinking fluoridated water is slightly risky, there is no hint that fluoridated toothpaste—as long as you don't swallow any—is dangerous. Tooth decay may also be declining because of better diet and hygiene. Also, foods and beverages processed with fluoridated water are ubiquitous. (Many bottled waters, though, do not have fluoride.) As a result, argues Alan Gray, a leading pro-fluoridation dentist in Canada, "it is becoming difficult to provide accurate, ethical advice" about fluoridation.

Among environmental controversies, fluoridation is unique in that one side has consistently denied that questions of risk or benefit even exist. The ADA states, "Anti-fluoridation groups attempt to create the illusion of a scientific controversy [which is] merely a ploy to create doubt about a well-researched, well-demonstrated preventive measure." But even well-researched articles raise hackles. When, in 1988, Chemical & Engineering News presented a balanced report on fluoridation, it attracted the wrath of the medical establishment. Says Taves, "Too many scientists lost their objectivity. This has become a religion on both sides."

Safe water: And that undercut the scientific process. The NIDR kept files on people perceived as threats to fluoridation. Political decisions were at odds with expert advice: a panel convened by the surgeon general in 1983 expressed concern, in closed sessions, about skeletal and dental damage from fluoride. At one point, a member said, "You would have to have rocks in your head, in my opinion, to allow your child much more than two parts per million [fluoride]." Said another, "I think we all agree on that." Even so, in 1986 EPA *raised* the fluoride standard from about two ppm to four.

This month EPA opened a review of the standard. Once EPA receives the official NTP report, it will establish a target "safe" fluoride level. The Safe Drinking Water Act requires that the level be zero for carcinogens, but the standard may be based on what is technically feasible. Fluoridation can be stopped immediately, but many communities with naturally fluoridated water—up to 12 ppm—would have to remove it. As EPA wrestles with the standard, fears John Sullivan of the American Water Works Association, "confusion will reign": local laws will still require fluoridation, a practice that may cause cancer.

As they await EPA's decision, pro-fluoridationists are invoking arguments of social justice. Dental researcher Ernest Newbrun of the University of California, San Francisco, contends that fluoridation promotes the health of children of "all races and all socioeconomic classes," not only those with enough money or discipline or access to the health system to take a fluoride supplement every day. He and others say it is morally wrong not to provide the benefits of fluoride. Although the NIDR's and other surveys suggest that fluoride in toothpastes and dental rinses also ensures healthy teeth for those who use the products, those who do not might suffer.

No one can foresee how the fluoride debate will play out this time. But since the 1950s, the country's environmental consciousness has been heightened. In the end, deciding whether or not to fluoridate turns less on science than on values. The sheer weight of good research may finally, after four decades, begin to inform those judgments and even overwhelm the unscientific rhetoric that has characterized both sides of the debate for far too long.

SHARON BEGLEY

PHOTOS BY JACQUES CHENET—NEWSWEEK

After every meal: *Toothpastes to fight cavities*

Parenting Toddlers

Toothpaste History

In addition to the toothbrush history, the following toothpaste history will be something interesting for your child as well. This will give them more insight into the origin of brushing teeth.

Toothpaste History

The development of toothpaste began as long ago as 300/500BC in China and India. According to Chinese history, a learned man, Huang-Ti, studied the care of teeth and claimed different types of pain felt in the mouth could be cured by sticking gold and silver needles into different parts of the jaw and gum. It was theories such as these that led to the development of dental cream.

First attempts at tooth cleaning included using abrasives such as crushed bone, crushed egg and oyster shells, which were used to clean debris from teeth. Tooth powders were the first noticeable advance and were made up of elements like powdered charcoal, powdered bark and some flavouring agents. This would be applied to teeth using a simple stick.

Toothpowder or dentifrice was first available in Britain in the late eighteenth century. It came in a ceramic pot and was available either as a powder or paste. The rich applied it with brushes and the poor with their fingers.

Modern toothpastes were developed in the 1800s. A dentist called Peabody was the first to add soap to toothpaste in 1824. Chalk was first added to toothpaste by John Harris in the 1850s. In 1873, toothpaste was first mass-produced into nice smelling toothpaste in a jar. In 1892, Dr. Washington Sheffield of Connecticut was the first to put toothpaste into a collapsible tube. Sheffield's toothpaste was called Dr. Sheffield's Creme Dentifrice. Advancements in synthetic detergents (after World War II) replaced the soap used in toothpaste with emulsifying agents such as Sodium Lauryl Sulphate and Sodium Ricinoleate.

The 1960's saw the introduction of fluoride into toothpaste. This development was followed in the 1980's with the addition of soluble calcium fluoride to fluoride toothpastes. It is therefore within the last thirty years that toothpastes contains the two ingredients - calcium and fluoride. Nowadays, there are controversial views on the effectiveness and safety of fluoride toothpaste. For those who are safety concious, the use of natural toothpaste might be a better choice.

How toothpaste work

Our mouth contains one or more of 500 types of microorganisms. Some of these, mainly streptococcus mutans, create sticky plaque from food residue in your mouth. Microorganisms in our mouth feed on left over food to create acid and particles called volatile sulfur molecules. The acid eats into tooth enamel to produce cavities while volatile sulfur molecules give breath its foul odor.

Toothpaste works with toothbrush to clean teeth and fight plaque bacteria. Toothpaste contains abrasives which physically scrub away plaque. In addition, toothpaste abrasives help remove food stains from teeth and polish tooth surfaces. Some toothpastes contain ingredients which chemically hinder the growth of plaque bacteria. These include ingredients like natural Xylitol and artificial triclosan.

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MEMORANDUM

LEGISLATIVE REFERENCE BUREAU — RESEARCH & ANALYSIS SECTION

WWW.MILWAUKEE.GOV/LRB

To: Ald. James Bohl, Jr.

From: Richard L. Withers, Ext. 8532

Date: May 10, 2011

Subject: Community Fluoridation of Water

This memorandum, and the articles and references compiled in the accompanying binder, respond to your request for a review of recommendations, reports, analyses and scientific studies that support the conclusion that the current practice of fluoridating City of Milwaukee water supplies is not only unnecessary but constitutes a significant hazard to health, especially in vulnerable populations.

The practice of community fluoridation of water has generated much debate over the last 65 years since Grand Rapids, Michigan, first added sodium fluoride to its water supply in January of 1945. The history of the debate has been described as one where "persons of goodwill and bad, [are] engaged in a bitter struggle for the public high ground" and where, "political machinations and ethical conundrums abound."[1]

The purpose of this memorandum is to provide reliable facts and persuasive arguments presently available in opposition to the continued fluoridation of Milwaukee water, and to avoid the hyperbole and 'spin' to which all sides of this debate have been prone.

The memorandum and the accompanying materials are organized as follows:

I. Community Fluoridation of Water
   - A. A Brief History
   - B. Fluoride Added to Community Water Supplies: A Product of Toxic Industrial Waste

II. Community Fluoridation to Promote Oral Health
   - A. The Case for Improving Oral Health
   - B. The Case for Fluoridation in Improving Oral Health Refuted

III. Bioethical Concerns

IV. Medical Issues and Risks of Fluoridation
   - A. Dental and Skeletal Fluorosis
   - B. Carcinogenicity
   - C. Metabolic Effects
   - D. Central Nervous and Reproductive Toxicity

V. Community Fluoridation Outside the United States

A. Canada
B. Europe

I. Community Fluoridation of Water

A. A Brief History

The first community fluoridation project in the United States was sponsored by the U.S. Public Health Service to study a growing consensus in the dental research community over the decades of the 1930's and 1940's that regular ingestion of fluoridated drinking water would reduce the incidence of childhood tooth decay. In effect, the children of Grand Rapids were the experimental subjects to prove or disprove this medical hypothesis.

The consensus of the dental research community was not shared by significant portions of the medical community. Early skeptical voices were raised, not just about the beneficial claims in support of fluoridation, but sounding a warning about risks. One of the most influential critics of fluoridation was Dr. Charles Gordon Heyd, President of the American Medical Association in 1936 and 1937. Dr. Heyd wrote:

> "I am appalled at the prospect of using water as a vehicle for drugs. Fluoride is a corrosive poison that will produce serious effects on a long-range basis. Any attempt to use water this way is deplorable."
>
> "...no physician in his right mind would hand to his patient a bottle filled with a dangerous drug with instructions to take as much or as little of it as he wished... And yet, the Public Health Service is engaged upon a widespread propaganda program to insist that communities do exactly that....The purpose of administering fluoride is not to render the water supply pure and potable but to contaminate it with a dangerous, toxic drug for the purpose of administering mass medication to the consumer, without regard to age or physical condition."[2]

Debate about fluoridation of Milwaukee water began in the Common Council in 1948. Dozens of files were introduced including a variety of resolutions and motions. Communication files include opinions from the City Attorney, the Milwaukee District Attorney and the Wisconsin Attorney General. Communication files were received from members of the public, health and dental associations, manufacturers, baking associations, researchers and others. Communication files were also received from the Health Department, the Water Works and the Office of the Mayor."[3]

An advisory referendum was placed upon the ballot. The Election Commission reported the results of the Special Election of April 7, 1953 in Council File 52-2252-a as follows:

"Do you favor fluoridation of Milwaukee water?"

Yes	**93,279**
No	**58,664**

Common Council Resolution File # 52-1221, adopted in July, 1953, provides:

> **Resolution relative to commencing the introduction of sodium-silicofluoride into the City water system.**
>
> **WHEREAS, Pursuant to Resolution File No. 48-1922, adopted October 24, 1950, the Milwaukee Water Works has taken all steps necessary to introduce fluorine into the water it distributes, has a stock of the necessary chemicals on hand, and is prepared to commence fluoridation; now, therefore, be it**
>
> **RESOLVED, By the Common Council of the City of Milwaukee, that the proper city officers be and they hereby are directed to commence the introduction of sodium silicofluoride into water distributed by the Milwaukee Water Works in sufficient quantities to bring the fluoride content of the water to a concentration of approximately one part per million.**

Fluoridation by the Milwaukee Water Works has continued from 1953 until the present. The current level of fluoride content is 1.1 milligrams of fluoride per liter of water. This is within the range of 0.7 to 1.2 milligrams that has been recommended by the U.S. Department of Health and Human Services (DHHS) until a new recommendation proposed on January 7, 2011, by DHHS and the U.S. Environmental Protection Agency (EPA) becomes effective. The new recommendation will set 0.7 milligrams per liter as the optimal fluoride level. Copies of the announcement and the Proposed Recommendation are attached. The original comment period was extended to April 15, 2011. No final recommendation has been published. The proposed recommendation responds to a report issued by the National Academies of Science (NAS) in 2006 recognizing adverse health effects and recommending that the EPA re-evaluate its recommendations based on increased fluoride exposure (e.g., fluoride in toothpaste and other products, topical fluoride applications), and new studies of bone and dental effects.

New fluoridation equipment installed at the Milwaukee Water Works (MWW) Linnwood North facility has the capacity to fluoridate water supplies at the new lower level according to Superintendent Carrie Lewis. However, fluoridation remains at 1.1 milligrams per liter pending issuance of a final recommendation by the federal government and the Wisconsin Department of Health Services. The lower fluoridation level of 0.7 milligrams per liter is supported by the Milwaukee Health Department (MHD) according to MHD Operations Manager Raquel Filmanowicz.

B. Fluoride added to Community Water Supplies: A Product of Toxic Industrial Waste

Pollution control devices used by the phosphate industry to capture fluoride gases produced in the production of commercial fertilizer are known as "wet scrubbers." The wet scrubbing process prevents the escape of gases containing fluorine compounds which previously resulted in harm to vegetation, crops and cattle.

After capture in the scrubbers, fluoride acid (hydrofluorosilicic acid), a classified hazardous waste, is barreled and sold to communities across the country. Many

communities, including Milwaukee, add hydrofluorosilicic acid to water supplies as the primary fluoride chemical for water fluoridation. The Milwaukee Water Works requires periodic certification of the content of hydrofluorosilicic acid used to fluoridate Milwaukee water.

Residents of communities that do not fluoridate the public water supply are nevertheless exposed to fluoridation in cereal, soda, juice, beer and any other processed food and drink manufactured with fluoridated water. This is sometimes referred to as a "halo effect" by proponents of water fluoridation for promoting oral health.

II. Community Fluoridation to Promote Oral Health

A. The Case for Fluoride in Improving Oral Health

Those supporting fluoridation of community water supplies to promote the reduction of tooth decay make the following points:

- Fluoridation is the least expensive and most effective way to reduce tooth decay.
- Fluoridation is safe.
- Fluoridation benefits both children and adults.
- Fluoridation benefits continue for a lifetime when fluoridated water consumption continues.
- Fluoridation is the surest way for everyone in the community to benefit.
- Fluoridation benefits everyone when they drink fluoridated water and consume foods and beverages prepared with it.

B. The Case for Fluoridation In Improving Oral Health Refuted

Fluoride was first investigated as an anti-caries agent because of the inverse relationship noted in many areas of the country between the prevalence of dental caries and the level of fluoride in drinking water. At first, scientists believed that the anti-caries activity of fluoride was the direct result of its incorporation into the apatite crystal of enamel, thus increasing its stability and reducing its acid solubility. The theory of pre-eruptive fluoride incorporation as the principal mechanism of caries prevention has been largely discounted. Recent studies have suggested that the anti-caries action of fluoride may be related to the fluoride levels in the saliva and plaque fluids rather than the enamel surface itself, i.e., the action is topical rather than systemic. There are widespread differences of opinion among experts as to the actual mechanism.

The sources of fluoride intake for the U. S. population are primarily water, food, dental products and air. Children may also receive fluoride in supplements. Although fluoride exposure is generally greater in areas with fluoridated water than in areas with non-fluoridated or low-fluoridated water, populations in both areas are exposed to fluoride from food sources, drinking water, processed beverages and dental products. In one recently published survey it was reported that the average intake of fluoride from food, averaged over all ages and sexes, was 1.76 mg/day. Fluoride exposure differs markedly,

depending upon several factors, e.g., lifestyle, dietary practices, age, gender and health status. It is clear however that drinking water provides minimal topical fluoride.

The Agency for Toxic Substances and Disease Registry (ATSDR) sets the Minimal Risk Level (MRL) for ingestion of fluoride at 0.4 mg/kg/day. (6) In a 20 pound child this amounts to 3.6 mg/day and for a 50 pound child, the minimal risk level is about 9 mg/day. The MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure. However, to avoid an undesirable degree of dental fluorosis, children should consume no more than 0.10 mg of fluoride per kg of body weight per day.

Interestingly, in British Columbia, only 11% of the population live in areas containing fluoridated water, as opposed to 40-70% in other Canadian regions. British Columbia, however, has the lowest rate of tooth decay in Canada. According to a 1987 report by Dr. Allan Gray, then director of the Division of Dental Health services for British Columbia, DMFT (decayed, missing or filled teeth) rates were falling drastically in both fluoridated and non-fluoridated areas.

Mark Diesendorf, an applied mathematician and health researcher in the Human Sciences Program at Australian National University has found, by comparing results from about 24 studies of unfluoridated districts in eight countries, that reductions in dental caries are just as great in non-fluoridated areas as in fluoridated areas. Diesendorf, M, The Mystery of Declining Tooth Decay, Nature, 322:125-129, (1986).

It seems clear that there is a link between fluoride intake and the reduction of dental caries. Although the mechanism is not fully understood, the effect is now thought to be due primarily to topical rather than systemic fluoride. In the early days of fluoridation, there were few other sources of fluoride in the daily diet. The introduction of fluoride into the daily diet (beverages prepared in communities with fluoridated water, toothpaste, food, supplements, etc.) starting in the 1950's has had the effect of reducing dental caries worldwide, even in those countries that do not fluoridate. In fact, fluoride is so widespread today that introducing it into public water supplies seems to have a very minimal effect in reducing dental caries. Current data suggests little difference between the health of teeth in communities having fluoridated water supplies compared to communities having unfluoridated water.

i.e. Brushing teeth vs. drinking

If no difference why put the toxin in our water?

One of the foremost critics of fluoridation, Dr. Paul Connett, and his colleagues have summarized the case against fluoridation of community water in the 2010 publication, *The Case Against Fluoride: How Hazardous Waste Ended Up in Our Drinking Water and the Bad Science and Powerful Politics That Keep It There*. Paul Connett, James Beck, Spedding Micklem, Chelsea Green Publishing.

III. Bioethical Concerns

Fluoride for reducing tooth decay is not considered an essential nutrient; is not a natural substance for infants, children or most adults; is an expensive-to-avoid medication with

an uncontrolled dose; and appears to be harmful to certain at-risk groups. Significant questions are therefore raised whether community fluoridation constitutes medication without informed consent. The costs of avoiding fluoridation in water and other processed food and beverages are significant involving alternative food and water sources or filtration systems. Therefore, the burden of opting out of fluoridation is disproportionately heavy for low-income families and individuals.

ethical issue medication w/out informed consent

The American Medical Association (AMA) has this to say to physicians about informed consent:

"Informed consent is more than simply getting a patient to sign a written consent form. It is a process of communication between a patient and physician that results in the patient's authorization or agreement to undergo a specific medical intervention.

In the communications process, you, as the physician providing or performing the treatment and/or procedure (not a delegated representative), should disclose and discuss with your patient:

- the patient's diagnosis, if known;
- the nature and purpose of a proposed treatment or procedure;
- the risks and benefits of a proposed treatment or procedure;
- alternatives (regardless of their cost or the extent to which the treatment options are covered by health insurance);
- the risks and benefits of the alternative treatment or procedure; and
- the risks and benefits of not receiving or undergoing a treatment or procedure.

In turn, your patient should have an opportunity to ask questions to elicit a better understanding of the treatment or procedure, so that he or she can make an informed decision to proceed or to refuse a particular course of medical intervention."

Clearly, though considered a drug (for the purpose of preventing disease), modern water fluoridation is administered without any of these 'patient' safeguards.

IV. Medical Issues and Risks of Fluoridation

A. Dental and Skeletal Fluorosis

Dental fluorosis, a discoloring or pitting of the teeth, occurs during early childhood while deciduous and permanent teeth and tooth enamel are still being mineralized and before they erupt within the mouth. It is believed that dental fluorosis occurs because of the toxicity of fluoride to the enamel-forming cells of the teeth. The degree to which a child experiences dental fluorosis depends on the amount of fluoride the child ingests. Dental authorities estimate that a child should ingest daily 0.03 mg to 0.07 mgs of fluoride per kg of body weight. When this amount is exceeded, dental fluorosis results. Moreover, the greater the fluoride overdose, the more severe is the dental fluorosis. Even with

supervision, it is possible for a small child to overdose on fluoride each day with only one brushing with a fluoride tooth paste by swallowing much of it during the brushing process.

The current model of fluorosis development proposes that "....fluoride affects the forming enamel by making it porous. The degree and extent of the porosity depend on the concentration of fluoride in tissue fluids when the teeth are developing..." and "....the porosity and discoloration can vary in degree among different areas of the same tooth....." The ultimate result is the increasing porosity of the teeth and, in extreme cases, loss of the affected teeth. Dental fluorosis is an excellent biomarker of excess fluoride ingestion and fluoride intoxication. It is a visible, sometimes easily seen and noticed marker of fluoride intoxication. Unfortunately it tells us of excessive fluoride intake after-the-fact, i. e. after the newly emergent teeth have already been altered.

Varying amounts of fluoride are found naturally in the water supplies of many communities. If too much fluoride is ingested by children it results in a toxic dental condition known as dental fluorosis. This condition is marked by visible mottling and/or discoloring of tooth enamel, pitting of the enamel and disturbed tooth shape. Teeth with moderate dental fluorosis typically "....may have yellow and brown strains..... they are pitted, brittle, and susceptible to fracture." Severe dental fluorosis "...not only produces unattractive teeth but also may increase the risk of tooth loss because it destroys parts of the protective enamel." Historically, dental fluorosis was first noted in children who grew up in areas where the drinking water supplies had a relatively high content of dissolved fluoride. It was noted that children with dental fluorosis had fewer cavities. Thus began the start of the "fluoride tradeoffs" which resulted in 80% to 90% of "treated" children with fewer cavities and 10% to 20% of those with dental fluorosis.

There is now widespread recognition of the fact that the prevalence of dental fluorosis has increased substantially throughout those countries where fluoridation is practiced. However, in spite of some reports to the contrary, there does not appear to be general agreement within the dental community as to whether the severity of dental fluorosis has increased.

The nationwide increase of dental fluorosis was first recognized, documented and published by the National Institute of Dental Research (NIDR) after conducting (1986-1987) a survey that involved 32,241 U.S. school children. The total prevalence of dental fluorosis in this group of children was estimated to be 22.3 percent and included (mostly) very mild to mild dental fluorosis. However some moderate to severe dental fluorosis was also found in approximately 1% to 2% of the children in "optimally" fluoridated water districtsAnother NIDR report published in 1988, studied four areas in Illinois with water concentration of one, two, three and four times the recommended "optimal" fluoride level. As of 1985, in the "optimally" fluoridated areas, twenty nine per cent of all tooth surfaces examined were reported to be affected by dental fluorosis. In those areas that had 2 to 4 times the optimal dose of fluoride in the water supply, dental fluorosis affected close to seventy per cent of the teeth involved. Skeletal fluorosis (osteofluorosis) is a complicated disease with a number of stages. The first two stages are preclinical, that is, the patient feels no symptoms but changes have taken place in the body. In the first preclinical

stage, biochemical changes occur in the blood and bone composition; in the second stage histological changes can be observed in bone biopsies. Some experts call these changes harmful because they are precursors of more serious conditions. Other experts say they are harmless. Most admit that the effects of long term ingestion of fluoridated water on bone are poorly understood.

The clinical stages of osteofluorosis include pain in the bones and joints, muscle weakness, fatigue, calcification of ligaments and bone spurs. Most experts in skeletal fluorosis agree that ingestion of 20 mg of fluoride per day for 20 years or more can cause crippling skeletal fluorosis and doses as low as 2 to 5 mg per day over the same time period can cause the preclinical stages. Moreover, the total quantity of fluoride ingested is the single most important factor in determining the clinical course of osteofluorosis. The severity of the symptoms correlates directly with the level and duration of exposure. For almost 40 years, investigators in the United States have searched for evidence of osteofluorosis. The U. S. Public Health Service reports that:

"....Radiographic changes in bone indicative of skeletal fluorosis, changes in bone mass, and effects on skeletal maturation were not observed at water fluoride concentrations of 1.2mg/l for 10 years and from 3.3 to 6.2 mg/l for a lifetime. In a survey of 170,000 radiographs of patients living in Texas and Oklahoma with water fluoride levels between 4 and 8 mg/l, Stevenson and Watson (1957) found 23 cases of radiographic osteosclerosis, but no evidence of skeletal fluorosis."

Nevertheless, large numbers of people in Japan, China, India. the Middle East and Africa have been diagnosed with skeletal fluorosis. In India, Tanzania and South Africa, crippling forms of skeletal fluorosis have been reported in pediatric age groups as well.

B. Carcinogenicity

An animal study conducted by the National Toxicology Program (NTP) provides evidence that fluoride causes osteosarcoma, a malignant bone tumor. See, Bucher, JR., MR Hejtmancik, JD Toft II, RL Persing, SL Eustis and JK Haseman., Results and conclusions of the NTP's rodent carcinogenicity studies with sodium fluoride., Int. J. Cancer 48:733-737, (1991). Although the NTP concluded that its study gave "equivocal" results with respect to cancer, the background memos and documents suggest that the results are actually stronger than suggested by the report. Similarly, the Procter and Gamble study likely gave stronger evidence of carcinogenicity, notably bone cancer, than suggested in the summary statements.

That fluoride is associated with bone cancer is reasonable from the point of view of what is known about the effects of fluoride: fluoride causes the division of immature bone cells (proliferation of osteoblasts) and fluoride accumulates in the bone and thus can cause damage there. Fluoride has been shown to be genotoxic in numerous test systems which is another property that is associated with carcinogens. In other words, the biochemistry and other toxicology studies support the view that fluoride maybe a bone carcinogen.

Epidemiology studies examining cancer in general and bone cancer in particular have been inconsistent. Studies using ecologic designs (the studies are based on cancer incidence or mortality for given geographic areas, not for individuals) have given conflicting results for cancer in general, for all bone cancer, and for osteosarcoma. The larger case-control studies do not show an association of fluoride or water fluoridation with bone cancer although at least one small study has shown an association. Most of these studies are handicapped by completely inadequate measures of exposure which would mask any effects that may be there because of misclassification of exposure. Given the widespread deliberate exposure of humans to water fluoridation and the suggestive animal data regarding cancer, especially osteosarcoma, it is surprising that a large case-control epidemiology study with good measures of fluoride exposure has not been initiated.

C. Metabolic Effects

Fluorine is contained in significantly fewer than 10 % of more than 700 minerals. Of these, only 5 or 6 minerals are truly common and almost all of these are either insoluble or have very limited solubility in water of neutral pH, although some exhibit enhanced solubility in water in the lower pH (acidic) range.

In those areas of the world where there is an abundance of the common fluorine-containing minerals in contact with either ground or surface water below pH 7, dissolved fluorine-containing minerals will be present in the indigenous water supplies. As a result, those areas will have an increased presence of fluorine in the vegetable and animal food-stuffs produced there. The fluorine that does enter the human food-chain, whether naturally occurring or as a result of artificial fluoridation, corresponds primarily to the sodium salt of the fluoride anion (F^-) and either sodium fluorosilicate or fluorosilicic acid. Clearly it is the nature of these materials which most concern us in this section and, in addition, the nature of the biological materials with which these interact.

The primary action of fluoride in metabolic and enzymatic reactions is related to the formation of "complexes" in one form or another. The fluoride anion has the highest charge density of any negative ion. As a result of this, it is now known that fluoride forms an exceptionally strong hydrogen bond (> 148 kJ/mol.) with substrates in amide-fluoride systems. Strong hydrogen bonding is now recognized as being clearly distinguishable from normal hydrogen bonding.

Another related characteristic of fluoride ion is that it exhibits an affinity for many metal ions, especially magnesium, manganese, aluminum, and calcium and therefore it can effect the bioavailability of these ions either separately or may cause either inhibition or otherwise interact with any enzyme system which requires one of these metals as a co-factor.

The impact of strong hydrogen bonding is that proteins, which consist of a repetitive sequence of amide linkages, are particularly susceptible to this type of hydrogen bonding. The end results of this type of interaction are two-fold. The lesser effect is that the

carbonyl-nitrogen (amide) bond in proteins may become more susceptible to cleavage even though fluoride itself is a less nucleophilic anion. The second, and probably enormously greater, effect is that the spatial arrangement or macromolecular structure of these materials depends heavily upon normal hydrogen bonding to produce the secondary stereochemical structure required for appropriate enzymatic activity to take effect. This has been demonstrated by Edwards and co-workers, who studied the perturbations caused by fluoride on the structure of Cytochrome C peroxidase. Further, ab initio calculations by Emsley et al. lead to the conclusion that the fluoride ion may completely disrupt the Thymine-Adenine linkage in DNA. A survey of the literature reveals no shortage of supporting research results. The conclusions reached in several of these studies are listed below.

Disrupts DNA

- Fluoride inhibits metalloproteins
- Fluoride inhibits DNA polymerase
- Fluoride induces chromosome aberrations
- Fluoride effects the adenyl cyclase system
- Fluoride inhibits yeast enolase
- Fluoride inhibits protein synthesis enzymes
- Fluoride inhibits gycolytic enzymes
- Fluoride inhibits cell growth enzymes
- Fluoride inhibits testosterone synthesis

It is of interest to note that the latter interaction may be responsible for those deleterious effects of fluoride which appear to be restricted to males (e. g. testosterone is involved in bone growth in males but not in females). The above list is by no means exhaustive. Rather, it should be taken to indicate that there is sufficient evidence to warrant more extensive research into this area. However, over all, the results described in the above references "suggest that sodium fluoride is potentially dangerous to humans."

Sum Fluoride Dangerous to humans

The interaction of fluoride in those metabolic processes involving calcium are also of great significance. This type of interaction may have been responsible for the recent observation that even when calcium is supplemented in osteoporotic patients, a large number of those who have also been treated with fluoride still show evidence of calcium deficiency. The lack of availability of calcium, either as a result of precipitation by fluoride or the formation of fluoroapatite, may result in hypocalcemia which may have other widespread and, as yet, poorly understood effects on bone formation and other regulatory mechanisms of the body.

calcium deficiency

Fluoride can seriously disturb the balance of enzymatically activated biochemical reactions. These effects clearly were not well-known at the commencement of fluoridation activities. Recent literature contains many references to original research results that illustrate that fluoride affects the metabolism of a number of common oral bacteria, (e.g., Streptococcus mutans). Thus, while there can be no doubt that fluoridation has contributed to the reduction of dental caries in the past, there is likewise little doubt that the continuation of the fluoridation process in the light of recent evidence outlined above

Smoking gun

is inappropriate without first answering the serious and potentially health-effecting questions raised.

References to the scientific literature are available if requested.

D. Central Nervous and Reproductive Toxicity

Several papers published in the last few years report that fluoride has adverse effects on the central nervous system (CNS), including intelligence and behavioral patterns. These papers encompass biochemical, histological, animal, and human studies and give a consistent picture regarding previously untested adverse consequences of fluoride exposure. Four important features of the animal toxicology and human studies are:

- 1) the fluoride doses are in the range that some humans actually receive; the animal studies are in the range of the upper end of fluoride food and water intake in the U. S.;
- 2) for some effects, the timing of the dose is critical, prenatal and early life exposures appear to be the critical periods for IQ deficits and some behavioral changes,
- 3) the adverse effects due to prenatal exposures are not reversible, and
- 4) the adult onset symptoms may be reversible if fluoride exposure is eliminated.

Dr. Phyllis Mullenix and co-workers published a study on the neurotoxicity of sodium fluoride in rats in 1995. The study used behavioral methodology that focused on behavioral repertoire, responses to novelty, and the temporal or sequential organization of spontaneous behavior. This methodology had been previously used to study alterations in CNS function and behavioral alterations including cognitive deficits (mental retardation) due to chemotherapy for childhood acute lymphoblastic leukemia, amphetamine induced hyperactivity, and triethyltin-induced hypoactivity. Thus, the methodology used to test the sodium fluoride should be considered a validated one.

The study found that prenatal exposures altered the behavioral outcome in male (but not female) offspring in a manner correlated with hyperactivity. There was no overt toxicity based on reduced body weight, suggesting the behavioral alterations were not secondary to another toxicity.

Milk exposures to fluoride affected the behavior of both males and females in a dose dependent manner (based on plasma fluoride levels), although the female rats were affected at lower doses. These doses also induced slight toxicity as judged by body weight gains. The behavioral changes for both sexes and at all doses were consistent with respect to the controls, and were different from the behavioral changes observed in male rats exposed prenatally. The observed behavioral changes are associated with cognitive deficits in other studies as well.

Adult rats were exposed for 6 weeks to 100 ppm fluoride in addition to the no fluoride control. No toxicity was associated with this dose based on differences in body weight.

Female (but not male) rats showed behavioral changes, and these changes were similar to those observed in the weanling exposures, namely cognitive defects.

Several studies have reported central nervous system effects in humans following occupational or environmental exposures to fluoride. About 25% of workers exposed to fluoride from cryolite (a fluoride-containing mineral) who had skeletal fluorosis also had central nervous system effects including fatigue, headache and giddiness. A similar proportion of aluminum smelter workers with skeletal fluorosis also reported psychiatric disturbances including depression, mental sluggishness and memory disturbances. Although these observations are reported for people with high fluoride exposure, the effects from occupational exposures are often used to forewarn of hazards that may also occur, but be harder to measure, at lower doses such as those that may result from environmental exposures.

There are also several studies where behavioral changes or other central nervous system symptoms are associated with fluoride exposure at lower levels. Studies have included reports of generalized progressive fatigue associated with a distinct decline in mental acuity in persons residing within 3 miles of an enamel factory emitting hydrogen fluoride. Cognitive deficits due to fluoride exposure, in the form of a population-wide decrease in intelligence in children, have been reported in several different populations in China in recent few years.

The findings of central nervous system effects (behavior changes and decreased IQ) in the human and animal studies following fluoride exposure is supported by biochemical data that show that fluoride accumulates in both fetal and adult human brain tissues. In other words, it can be shown that the fluoride reaches the brain tissue, and thus is

Reproductive toxicity is the study of toxic effects on the reproductive capacity of males and females. Animal toxicity tests to determine whether or not a substance is a reproductive toxin include:

- 1) alterations in sperm count and quality;
- 2) number of litters and number of conceptuses/litter when male or female animals are exposed to a potential toxicant prior to mating; and
- 3) number of live births when male or female animals are exposed to a potential toxicant prior to mating.

Human epidemiology studies of birth rates may also give insight into reproductive toxins.

Developmental toxicology is the study of conditions (including chemical substances) that lead to abnormal development. Manifestations of developmental toxicity include structural malformations (birth defects), growth retardation, functional impairment and death of the organism. The study of developmental functional deficits, including neurobehavioral effects has emerged in the last twenty years (1), and is thus still in its early years of elucidation.

There have been a number of studies of the effects of fluoride ingestion and of water fluoridation on reproductive capability of humans and animals. In its influential 1991 review of water fluoridation, the US Public Health Service (US PHS) found that fluoride may affect reproduction in animals, although some data were contradictory.

Several laboratory studies of rodents (rats and mice) exposed to fluoride in food or drinking water showed reduced fertility. Heifers exposed to 5 ppm fluoride in water during four breeding seasons calved at a rate that was only 30% of normal. At higher fluoride doses, the effect was earlier and more severe, which is strongly indicative that the effects observed were due to fluoride and not a confounding factor.

reduced fertility

In screech owls, chronic dietary intake of 40 ppm sodium fluoride resulted in significantly smaller egg volume, which is considered a slight-to-moderate reproductive disorder. No gross abnormalities were apparent. Pastel mink fed up to 230 ppm fluoride in their diet did not show adverse reproductive effects such as changes in breeding, gestation, whelping or lactation. However, there was only a 14% survival rate of kits whelped by females fed 385 ppm fluoride.

Several animal studies have examined the effect of fluoride on sperm count, motility and other sperm quality parameters. Examination of albino rats fed 10 mg/kg sodium fluoride for 50 days revealed biochemical alterations that manifest themselves in reduced sperm motility and lower sperm count. Both of these are considered adverse reproductive effects. Withdrawal of sodium fluoride reversed most, but not all of the observed alterations. Addition of ascorbic acid and calcium to the rat diet after withdrawal of the sodium fluoride produced full recovery from the adverse effects of the sodium fluoride.

Because of the lack of any human epidemiology studies, Stan Freni, a participant in the US PHS review, initiated an epidemiological study of the possible association of fluoride concentrations in community water supplies and US birth rates. Freni calculated the annual total fertility rate for white women in the age range 10-49 years for the period 1970-1988 in 30 regions (somewhat equivalent to counties) in 9 states. He compared the total fertility rates with measures of fluoride concentrations in drinking water (up to 10 ppm in some individual systems, but averaged over all the drinking water in the county), the percentage of people drinking highly fluoridated (>3 ppm) water, and various socioeconomic factors that are known to affect fertility rates. After accounting for the socioeconomic and other demographic factors, Freni found an association of decreasing total fertility rate (low birth rates) with increasing water fluoride concentrations for most, but not all, of the regions examined.

Regarding fluoride and reproductive effects: Taken together, the studies summarized here raise serious concerns about the impact of fluoride on human reproduction, even at water fluoridation levels currently considered "safe". The human epidemiology study conducted by Freni (7) does not prove that fluoride in drinking water decreases fertility. However, the association observed in the study is a serious cause of concern, especially because of its consistency with some observations in laboratory and farm animals. It

Serious concerns on human reproduction

clearly shows the need for careful studies that are designed to ascertain if water fluoridation decreases human fertility.

V. Community Fluoridation Outside the United States

Canada

A comprehensive recent study of community fluoridation in Canada is included in the attached materials. A number of potential health risks were reviewed. Nevertheless, the report recommends a minimum of 1.5 milligrams of fluoride per liter exposure, significantly more than the proposed minimum recommendation under consideration in the United States.

Europe

Few communities and countries in Europe fluoridate water supplies. The practice is not necessarily prohibited, but in many countries, such as the Netherlands, local communities are simply not authorized to fluoridate the water.

Several articles are included in the attached materials describing fluoridation practices and population studies.

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[1] *The Fluoride Wars: How a Modest Public Health Measure Became America's Longest-Running Political Melodrama.* By R. Allan Freeze and Jay H. Lehr. © 2009, John Wiley & Sons.

[2] http://www.whonamedit.com/doctor.cfm/2524.html, accessed Feb. 21, 2011. Sponsored by Whonamedit: A Dictionary of Medical Eponyms.

[3] Examples include Common Council Files: ## 48-2099 and 48-2099-a to d; # 52-928; # 52-1018; # 52-1018a; # 52-1019; # 52-1086; 33 52-1221 and 52-1221a to c; # 52-1922b; # 52-2389; # 52-2390; # 52-2391; # 52-2534; # 52-2893; # 52-2894: and # 52-3760.

The New York Times

January 11, 2011

EPA Proposes Phaseout of Fluoride-Based Pesticide

By ELANA SCHOR of

U.S. EPA today proposed to start gradually banning a pesticide often used on cocoa beans and dried fruits that degrades to fluoride, a move closely linked to the Obama administration's decision last week to curb the maximum levels of fluoride in drinking water out of concern for children's health.

EPA's bid to wind down legal use of sulfuryl fluoride, citing the health risk to children posed by aggregate fluoride exposure, marks a long-awaited victory by the three public-health groups that first asked the agency to rein in the pesticide more than five years ago.

One of the three advocacy organizations, the Environmental Working Group (EWG), said the sulfuryl fluoride phaseout appears to be EPA's first official granting of any pesticide restriction petition filed by green advocates.

The Department of Health and Human Services and EPA announced Friday that fluoride, long considered a beneficial tap-water additive that helps prevent cavities, should be restricted to 0.7 milligrams per liter, or the low end of previous legal ranges (*E&ENews PM*, Jan. 7).

In its proposed prohibition on sulfuryl fluoride, EPA acknowledged that the pesticide's residues on food are "responsible for a tiny fraction of aggregate fluoride exposure" but deemed that children's total contact with fluoride in the environment -- through drinking water as well as toothpaste -- posed an excess risk of tooth and bone damage.

This week's twin fluoride restrictions reflect "a growing consensus that Americans are exposed to too much fluoride," EWG senior vice president for research, Jane Houlihan, said today. "It raises the concern that, for many decades now, the public has been overexposed."

First approved for use as an anti-termite insecticide more than 50 years ago, sulfuryl fluoride was federally registered for use on food in 2004 and 2005 by Dow AgroSciences

LLC as an alternative to methyl bromide, a pesticide that began to be phased out of commerce after the 1987 Montreal Protocol identified it as a depleter of the ozone layer.

Soon after the chemical was approved as a food fumigant, the advocacy groups Fluoride Action Network (FAN) and Beyond Pesticides joined EWG in filing a formal objection with EPA. As in the case of Friday's fluoride announcement, today's sulfuryl fluoride limits came in the wake of a revised risk assessment the agency conducted after a 2006 National Academy of Sciences report urged it to consider dental fluorosis as a negative health consequence of exposure rather than a cosmetic impediment.

Dental fluorosis, which manifests as spotting on the teeth among children who consume too much fluoride as their mouths develop, can lead to long-term breakdown of the tooth enamel and other painful effects.

The gradual EPA removal of sulfuryl fluoride allowances will be subject to public comment before taking effect and include a three-year head start for significantly affected industries such as the cocoa and walnuts sectors.

Estimating that the pesticide is applied to 100 percent of cocoa crops, EPA warned in its proposed phaseout that "cocoa imports (which in 2009 were valued at approximately $1.2 billion) would be lost due to either destruction or refusal of shipments by warehouse operators" unless businesses can develop a viable alternative to sulfuryl fluoride for cocoa fumigation.

Today's EPA proposal also references multiple objections Dow had raised in previous years to arguments made by the advocacy groups behind the petition, suggesting that pushback from industry on the sulfuryl fluoride limits can be reasonably expected.

Click here (pdf) to read a pre-publication copy of EPA's proposal to phase out sulfuryl fluoride tolerances.

For more news on energy and the environment, visit www.greenwire.com.
Greenwire is published by Environment & Energy Publishing. Read More »

FLUORIDE AS ROACH – RAT POISON

Recognition and Management of Pesticide Poisonings

You will need Adobe Acrobat Reader to view some of the files on this page. See EPA's PDF page to learn more.

Table of Contents | Ordering Information | Updates and Corrections

The new revised version of EPA's pesticide poisoning handbook is now available. The fifth edition of *Recognition and Management of Pesticide Poisonings* is edited by Dr. Routt Reigart and Dr. James Roberts, and is published by EPA's Office of Pesticide Programs. Both English and Spanish versions are available.

Revisions:
The new edition covers about 1,500 pesticide products in an easy-to-use format. Toxicology, signs and symptoms of poisoning, and treatment are covered in 19 chapters on major types of pesticides. The new edition covers new pesticide products that have come on the market since 1989, includes a new chapter on disinfectants, reviews clinical experiences with pesticide poisonings, and contains detailed references.

View Entire Handbook (238 pp, 895K)

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www.epa.gov/oppfead1/safety/healthcare/handbook/chapter8.htm

CHAPTER 8

Recognition and Management of Pesticide Poisonings, 5th Edition, Chapter 8. The Office of Pesticide Programs, U.S. E.P.A

Other Insecticides, Acaricides, and Repellents

HIGHLIGHTS

- Multiple agents, with widely varying toxicity
- Careful history will usually reveal exposure history
- Agents of particular concern due to wide use are pyrethroids, diethyltoluamide, and borates

Signs and Symptoms:

- Variable and highly related to the specific agent
- Boric acid causes severe erythematous and exfoliative rash (boiled lobster appearance)
- Agents such as boric acid, diethyltoluamide, and pyrethroids should be suspected in cases of unusual nervous system symptoms

Treatment:

- Specific to the agents
- Skin and GI decontamination
- Severe CNS symptoms may require intensive care management

This chapter discusses insecticides, acaricides, and repellents that have toxicologic characteristics distinct from the insecticides discussed in previous chapters. Pesticides reviewed include: alkyl phthalates, benzyl benzoate, borates, chlordimeform, chlorobenzilate, cyhexatin, diethyltoluamide, fluorides, haloaromatic urea compounds, methoprene, propargite, pyrethroids, and sulfur.

ALKYL PHTHALATES

Dimethyl phthalate has been widely used as an insect repellent applied directly to the skin. Dibutylphthalate is impregnated into fabric for the same purpose. It is more resistant to laundering than dimethyl phthalate.

Toxicology

Dimethyl phthalate is strongly irritating to the eyes and mucous membranes. It has caused little or no irritation when applied to skin, and dermal absorption is apparently minimal. It has not caused sensitization. Tests in rodents have indicated low systemic toxicity, but large ingested doses cause gastrointestinal irritation, central nervous system depression, coma, and hypotension.

Treatment

No antidote is available. Supportive measures (hydration, oxygen if needed) are probably adequate to manage all but the most severe poisonings.

BENZYL BENZOATE

Toxicology

Incorporated into lotions and ointments, this agent has been used for many years in veterinary and human medicine against mites and lice. Apart from occasional cases of skin irritation, adverse effects have been few. The efficiency

of skin absorption is not known. Absorbed benzyl benzoate is rapidly biotransformed to hippuric acid which is excreted in the urine. When given in large doses to laboratory animals, benzyl benzoate causes excitement, incoordination, paralysis of the limbs, convulsions, respiratory paralysis, and death. No human poisonings have been reported.

Treatment

1. Skin decontamination. If significant irritant effect appears, medications should be discontinued and the skin cleansed with soap and water. Eye contamination should be treated by prolonged flushing with clean water or saline.

2. Gastrointestinal decontamination. If a potentially toxic amount has been swallowed and retained and the patient is seen soon after exposure, gastrointestinal decontamination should be considered as outlined in Chapter 2.

3. Seizures. If seizures occur, control may require anticonvulsant medication as outlined in Chapter 2.

BORIC ACID AND BORATES

Boric acid is formulated as tablets and powder to kill larvae in livestock confinement areas and cockroaches, ants, and other insects in residences. Rarely, solutions are sprayed as a nonselective herbicide.

Toxicology

Boric acid powders and pellets scattered on the floors of homes do present a hazard to children. Their frequent use for roach control increases access for ingestion. A series of 784 patients has been described with no fatalities and minimum toxicity. Only 12% of these patients had symptoms of toxicity, mostly to the gastrointestinal tract.[1] However, there have been some recent reports of fatal poisonings,[2,3] and a great many poisonings of newborns which occurred in the 1950s and 1960s often ended in death.[4,5] Historically, many poisonings have resulted from injudicious uses in human medicine aimed at suppressing bacterial growth, such as compresses for burns, powders for diaper rash, and irrigation solutions.[6,7] With the increased use of boric acid for roach control, suicidal or accidental ingestion is still likely to occur.[3,7]

Borax dust is moderately irritating to skin. Inhaled dust caused irritation of the respiratory tract among workers in a borax plant. Symptoms included nasal irritation, mucous membrane dryness, cough, shortness of breath, and chest tightness.[8,9]

Commercial Products

ALKYL PHTHALATES

dibutylphthalate
dimethyl phthalate
DMP

BENZYL BENZOATE

BORIC ACID AND BORATES

boric acid
sodium polyborates
Polybor 3
sodium tetraborate
decahydrate
Borax

CHLORDIMEFORM (nr)

CHLOROBENZILATE (nr)

Acaraben
Akar
Benzilan
Folbex

CYHEXATIN (nr)

Acarstin
Metaran
Oxotin
Pennstyl
Plictran

DIETHYLTOLUAMIDE (DEET)

Auton
Detamide
Metadelphene
MGK
Muskol
Off!
Skeeter Beater
Skeeter Cheater
Skintastic for Kids

FLUORIDES

sodium fluoride (wood protection only)
sodium fluosilicate (sodium silico fluoride) (nr)
Prodan
Safsan
sodium fluoaluminate
Cryolite
Kryocide
Prokil

(Continued on the next page)

usually considered contraindicated in these poisonings due to the rapid onset of seizures.

3. Seizures. Treatment is primarily supportive, with control of seizures by anticonvulsants, as outlined in Chapter 2. Persons surviving poisoning by ingestion of DEET have usually recovered within 36 hours or less.[16,17]

FLUORIDES

Sodium fluoride is a crystalline mineral once widely used in the United States for control of larvae and crawling insects in homes, barns, warehouses, and other storage areas. It is highly toxic to all plant and animal life. The only remaining use permitted is for wood treatement

Sodium fluosilicate (sodium silico fluoride) has been used to control ectoparasites on livestock, as well as crawling insects in homes and work buildings. It is approximately as toxic as sodium fluoride. All uses in the U.S. have been cancelled.

Sodium fluoaluminate (Cryolite) is a stable mineral containing fluoride. It is used as an insecticide on some vegetables and fruits. Cryolite has very low water solubility, does not yield fluoride ion on decomposition, and presents very little toxic hazard to mammals, including humans.

Hydrofluoric acid is an important industrial toxicant, but is not used as a pesticide. Sulfuryl fluoride is discussed in Chapter 16, Fumigants.

Toxicology

Sodium fluoride and fluosilicate used as insecticides present a serious hazard to humans because of high inherent toxicity, and the possibility that children crawling on floors of treated dwellings will ingest the material.

Absorption across the skin is probably slight, and methods of pesticide use rarely include a hazard of inhalation, but uptake of ingested fluoride by the gut is efficient and potentially lethal. Excretion is chiefly in the urine. Within the first 24 hours of intoxication, renal clearance of fluoride from the blood is rapid. However, patients go on to continue to excrete large amounts of fluoride for several days. This is thought to be due to a rapid binding of fluoride to a body store, probably bone. The subsequent release of fluoride from bone is gradual enough not to cause a recurrence of toxicity.[26,27] Large loads of absorbed fluoride may potentially poison renal tubule cells, resulting in acute renal failure. Children will have greater skeletal uptake of fluoride than adults, therefore limiting the amount the kidney needs to handle. Despite this, children are still at great risk because of their smaller body mass compared to adults in relation to the amount ingested.[27]

The toxic effects of fluoride in mammals are multiple, and all may threaten life. The primary effects from fluoride result from an inhibition of critical intracellular enzymes and the direct effect on ionized calcium in extra-cellular fluid. Hypocalcemia commonly occurs.[26,28,29,30]

Ingested fluoride is transformed in the stomach to hydrofluoric acid, which has a corrosive effect on the epithelial lining of the gastrointestinal tract. Thirst, abdominal pain, vomiting, and diarrhea are usual symptoms. Hemorrhage in the gastric mucosa, ulceration, erosions, and edema are common signs.[31]

Absorbed fluoride ion reduces extracellular fluid concentrations of calcium and magnesium. Hypocalcemia sometimes results in tetany.[30] Cardiac arrhythmia and shock are often prominent features of severe poisoning. Hypotension and severe arrhythmia, sometimes progressing to ventricular fibrillation, may also occur.[26,32] These probably result from combinations of effects of fluid and electrolyte disturbances including hyperkalemia[32] and direct actions of fluoride on heart and vascular tissues. Fluoride may directly affect the central nervous system, resulting in headache, muscle weakness, stupor, convulsions, and coma.[26,27,28] Respiratory failure and ventricular arrythmias are common causes of death.[26,27]

Confirmation of Poisoning

A population drinking water with a concentration of 1 mg per liter will have a plasma inorganic fluoride concentration between 0.01 and 0.03 mg per liter[28] and rarely above 0.10 mg per liter. In fatal cases of poisoning, plasma levels of 3.5 mg per liter and higher have been recorded, although survival has been reported in patients with levels as high as 14 mg per liter.[26,28]

Treatment: Fluoride Toxicosis

1. Skin decontamination. Wash skin with soap and water as outlined in Chapter 2. Eye contamination should be removed by prolonged flushing of the eye with copious amounts of clean water or saline. If irritation persists, specialized medical treatment should be obtained.

2. Gastrointestinal decontamination. If **sodium fluoride or sodium fluosilicate** has been ingested, consider gastric decontamination as outlined in Chapter 2.

If the victim is obtunded or if vomiting precludes oral administration, the airway should be protected by endotracheal intubation, then the stomach should be gently intubated and lavaged with several ounces of one of the liquids named below. Activated charcoal is not likely to be of use because it does not bind the fluoride ion well.

3. Calcium and magnesium. If the victim is fully alert, and if vomiting does not totally prevent swallowing of a neutralizing agent, prompt oral administration of **milk, calcium gluconate, or magnesium citrate** will precipitate fluoride ion in the gut and therefore may be life-saving. The milk provides the calcium ions that will bind to fluoride, thereby reducing absorption. Magnesium-based antacids have also been used to neutralize the acid and facilitate the production of poorly absorbed salts.[26] There are no data on the optimum amounts to be administered.

4. Blood analysis. A blood specimen should be drawn for serum electrolyte analysis for sodium, potassium, calcium, magnesium, fluoride, and bicarbonate capacity. Blood should also be drawn to type and cross match for blood transfusion.

5. Intravenous fluids (initially 5% dextrose in 0.9% saline) should be started to combat dehydration, shock, and metabolic acidosis. Fluid balance should be monitored closely to forestall fluid overload if renal failure occurs. If metabolic acidosis is detected, sodium bicarbonate should be administered to keep the urine alkaline as this may hasten excretion.[27] Intravenous fluids must be stopped if anuria or oliguria (less than 25-30 mL per hour) develops.

6. Hemodialysis should be reserved for compromised renal function.[26]

7. Monitor cardiac status by continuous electrocardiography. Ventricular arrhythmia may necessitate DC cardioversion.

8. Tetany. If overt or latent tetany occurs, or if hypocalcemia is demonstrated, or if it appears likely that a significant amount of fluoride has been absorbed, administer 10 mL of 10% **calcium gluconate** intravenously, at no more than 1 mL per minute.

Dosage of Calcium Gluconate:
Supplied as 100 mg/mL (10% solution)

- *Adults and children over 12 years:* 10 mL of 10% solution, given slowly, intravenously. Repeat as necessary.
- *Children under 12 years:* 200-500 mg/kg/24 hr divided Q6 hr. For cardiac arrest, 100 mg/kg/dose. Repeat dosage as needed.

9. Oxygen by mask should be administered for hypotension, shock, cardiac arrhythmia, or cyanosis. Shock may require administration of plasma or blood.

10. Acid Burns. Since these compounds can cause severe acid burns to the esophagus and stomach, patients should be referred for surgical evaluation and endoscopy. If burns are documented, treatment for acid burns should be continued by a surgeon or gastroenterologist.

Treatment: Sodium Fluoaluminate (Cryolite)

Cryolite is much less toxic than other fluorides. If a very large amount has been ingested, it may be appropriate to measure serum calcium to insure that hypocalcemia has not occurred. If so, intravenous 10% calcium gluconate would be indicated (see 8 above). It is unlikely that treatment for fluoride toxicity would be necessary following ingestion of sodium fluoaluminate.

HALOAROMATIC SUBSTITUTED UREAS

Diflubenzuron is a haloaromatic substituted urea which controls insects by impairing chitin deposition in the larval exoskeleton. It is formulated in wettable powders, oil dispersible concentrate, and granules for use in agriculture and forestry, for aerial application against gypsy moth, and in settings where fly populations tend to be large, such as feedlots. Teflubenzuron is another haloaromatic substituted urea insecticide with similar toxicologic properties.

Toxicology

There is limited absorption of diflubenzuron across the skin and intestinal lining of mammals, after which enzymatic hydrolysis and excretion rapidly eliminate the pesticide from tissues. Irritant effects are not reported and systemic toxicity is low. Methemoglobinemia is a theoretical risk from chloraniline formed hydrolytically, but no reports of this form of toxicity have been reported in humans or animals from diflubenzuron exposure. Teflubenzuron also shows low systemic toxicity.

Treatment

1. Skin decontamination. Wash skin with soap and water as outlined in Chapter 2. Eye contamination should be removed by prolonged flushing of the eye with copious amounts of clean water or saline. If irritation persists, obtain specialized medical treatment. Sensitization reactions may require steroid therapy.

2. Gastrointestinal decontamination. If large amounts of propargite have been ingested and the patient is seen within an hour, consider gastrointestinal decontamination. For small ingestions, consider oral administration of activated charcoal and sorbitol.

Treatment

1. Skin decontamination. Wash skin with soap and water. Contamination of the eyes should be removed by prolonged flushing with clean saline or water. If eye irritation persists, obtain ophthamologic care.

2. Gastrointestinal decontamination. Unless an extraordinary amount of sulfur (several grams) has been ingested shortly prior to treatment, there is probably no need for gastrointestinal decontamination. Adsorbability of sulfur on activated charcoal has not been tested.

The most serious consequence of sulfur ingestion is likely to be that of catharsis, resulting in dehydration and electrolyte depletion, particularly in children. If diarrhea is severe, oral or intravenous administration of glucose and/or electrolyte solutions may be appropriate.

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FATAL POI: FLUOROAC

REPORT OF A CASE

John L. Brockmann, M.D.; Arthur V. McDowell, M.D.; William G. Leeds, M.D.

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Since this article does not have an abstract, we have provided the first 150 words of the full text.

Journal of the American Medical Association 1955, 159 (16): 1529-32.

"Fatal Poisoning with Sodium Fluoroacetate: Report of A Case."

EXCERPT

Extensive chemical research carried out in recent years has produced a rodenticide, sodium fluoroacetate, which has been found to be very effective and extremely toxic. Sodium fluoroacetate, or "1080" as it was called during the investigation period, is said to be one of the most noxious substances known, since it is toxic to all mammals, man included. Sodium fluoroacetate was first reported by Kalmbach. With this poison rat control can be much more easily and effectively carried out than ever before; however, because of its extreme toxicity (three teaspoons of the watered solution used for rats is sufficient to cause death in the adult human being) many necessary precautions have been taken in its employment and administration. It is distributed only to qualified members of governmental agencies and to properly insured and licensed pest control agencies and operators. In addition, strict regulations are imposed upon its use, and standard procedures

Articles Citing This Article

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factors undoubtedly enter into the picture, such as improved understanding of the disease and of the function of the adrenal glands, improved sanitation, better living conditions, the use of vitamins, the better care of patients with tuberculosis, and the control of intercurrent infections by antibiotics. Singly or collectively, none of these factors can be considered more than contributory to survival. However, the prolonged survival times here recorded (no deaths under 9 years, and five of eight patients surviving 15 to 18 years) suggest that this extract may have special virtues in prolonging life. It also suggests that it warrants further study.

SUMMARY AND CONCLUSIONS

The survival time in cases of adrenal cortical hypofunction (Addison's disease) has generally been materially prolonged through modern methods of treatment. Eight of my patients have survived for over 15 years. This covers an experience in the treatment of more than 150 patients seen during the last 35 years. In a series of eight consecutive patients (1933-1940) treated with Swingle's suprarenal cortical extract and with an adequate intake of salt daily, all survived for at least 9 years, seven survived 10 years or more, and five survived from 15 to 18 years. Two patients recovered. In several patients recourse was made to other adrenal preparations; two received pellet implantations late in the course of their disease and two were given small doses of cortisone. The survival time in this series sets a new record in adrenal cortical hypofunction though the number of cases concerned is admittedly small. The results here presented suggest the desirability of finding ways and means to make Swingle's suprarenal cortical extract more generally available and at a lower cost or to compound some balanced mixture of adrenal hormones that will serve as an effective and inexpensive substitute.

1342 du Pont Bldg.

Housewives' Dermatitis.—[This] is one of the commonest of occupational hazards. All soaps and detergents degrease the skin to some extent, in proportion to their cleaning efficiency. The results are essentially the same, whether the skin fat is emulsified and rinsed off or whether it is removed by solution. The denuded keratin becomes exposed to the action of the cleaner, for a shorter or a longer time, depending upon the ability of the sebaceous glands to replace the lost fat and on the emulsifying and spreading properties of the sweat. . . . Once dermatitis has developed, avoidance of further exposure is essential, but at this stage rubber gloves are rarely tolerated. Mild cases recover with the use of bland applications; but in the severest forms recovery may take months or the dyskeratotic process may persist indefinitely because the germinal cells have been damaged. No cream can give back to denatured basal cells the power to form a normal keratinous layer. . . . Toilet soaps have a beneficial effect on the skin. . . . When the skin is cleaned with soap and water an adsorption layer of soap is formed and is converted in less than an hour into a layer of free fatty acids which cling tenaciously to the skin. These fatty acids have beneficial (bactericidal) effects as well as very occasional harmful (allergenic) effects. Toilet soaps rarely cause dermatitis although they may aggravate existing dermatitis as, for that matter, may water itself.—B. Russell, M.D., Advances in the Treatment of Skin Diseases, *The Practitioner*, October, 1955.

CLINICAL NOTES

FATAL POISONING WITH SODIUM FLUOROACETATE

REPORT OF A CASE

John L. Brockmann, M.D.
Arthur V. McDowell, M.D.
and
William G. Leeds, M.D., Hartford, Conn.

Extensive chemical research carried out in recent years has produced a rodenticide, sodium fluoroacetate, which has been found to be very effective and extremely toxic. Sodium fluoroacetate, or "1080" as it was called during the investigation period, is said to be one of the most noxious substances known, since it is toxic to all mammals, man included.[1] Sodium fluoroacetate was first reported by Kalmbach.[2] With this poison rat control can be much more easily and effectively carried out than ever before; however, because of its extreme toxicity (three teaspoons of the watered solution used for rats is sufficient to cause death in the adult human being) many necessary precautions have been taken in its employment and administration. It is distributed only to qualified members of governmental agencies and to properly insured and licensed pest control agencies and operators. In addition, strict regulations are imposed upon its use, and standard procedures are outlined in attempts to prevent the inadvertent poisoning of human beings and domestic animals. A technical bulletin is published by the manufacturers with instructions for usage.[3]

The drug is a colorless, odorless, and tasteless water-soluble salt. As a rat poison, it is prepared by dilution to one part in 300 or 500 parts of water. The drug is equally effective as a poison regardless of mode of entry. It may be absorbed through the intact skin but not readily; however, it is easily absorbed through cuts and abrasions.[3] It is rapidly absorbed in the gastrointestinal tract and may also be absorbed through the lungs by breathing dust containing the poison.[4] The theory has been advanced by several writers that sodium fluoroacetate acts as a metabolic poison producing its lethal effects, not as a free fluoride, but as an intact molecule.[5] The exact mechanism has not, as yet, been uncovered, but it is felt that sodium fluoroacetate competes in reactions where acetate normally takes part. It has been suggested

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From Hartford Hospital. Dr. Brockmann is now a lieutenant (j.g.) in the U. S. Naval Reserve.

Dr. Abraham Stolman, Toxicologist for the state of Connecticut, made the determinations of fluoride content. The Pathology Department of Hartford Hospital (particularly Dr. George McAdams) made the post-mortem examination and synopsis of the findings.

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4. Sodium Fluoroacetate, Clinical Memorandum, Technical Development Branch, Communicable Disease Center, U. S. Public Health Service, Savannah, Ga., May, 1952.
5. Liebecq, C., and Peters, R. A.: The Toxicity of Fluoroacetate and the Tricarboxylic Acid Cycle, Biochim. et biophys. acta 3: 215, 1949.

that this interference with acetate metabolism may cause a piling-up of citrate.[6]

Chenoweth and Gilman have studied extensively the reactions produced by sodium fluoroacetate in animals and have classified the animals in groups according to the reactions brought about in them by the agent.[6] The organ systems affected are chiefly the cardiorespiratory and central nervous systems. Briefly, the classification includes the following: group 1, in which the action is a cardiac one, with death due to ventricular fibrillation; group 2, in which there is both a cardiorespiratory and central nervous system response, with death in respiratory failure during convulsions or ventricular fibrillation; group 3, in which the primary effect is on the central nervous system, with no cardiac abnormality; and group 4, in which there is an atypical response, including bradycardia and respiratory depression. In man, sodium fluoroacetate has been found to produce a mixed response, with elements of cardiorespiratory and central nervous system damage similar to that produced in monkeys.[7]

According to the available literature, there have been 22 known cases of poisoning with sodium fluoroacetate, 16 of which were fatal [4]; however, only 2 cases have been reported in the medical literature to our knowledge. One of these was fatal, the other was not. Gajdusek and Luther in 1950 [1] reported a case of nonfatal poisoning in a 2-year-old infant, and in 1952 Harrisson and others [7c] reported a case of fatal poisoning. In each of these articles, the experimental and toxicologic literature was thoroughly reviewed, and the references are complete. Thus far there has been no case reported in which the exact quantity of the poison taken was known. Careful calculations in the cases mentioned have given approximations of the amount ingested. It has been estimated that about 5 mg. per kilogram of body weight is lethal to man. In the case reported by Harrisson and others a minimum of 6 mg. per kilogram was ingested.[7c] In this paper a case of fatal poisoning with sodium fluoroacetate is reported. We feel that this case is of particular interest because the patient lived for five days after ingesting the poison and extensive laboratory work was therefore possible. A postmortem examination with analysis of the organ contents was also obtained.

REPORT OF A CASE

A 17-year-old boy, son of a professional rat exterminator, entered the emergency room of our hospital at 4 a. m., Jan. 1, 1954, and told the nurse that he had ingested a solution of sodium fluoroacetate. The amount ingested could not be determined accurately, but a previously unopened 8 oz. can of the material was found in his room half empty. Apparently, the boy had dissolved a large amount of the poison in water and swallowed the solution, after which he promptly vomited. He stated that he had noted almost immediate epigastric pain. He came to the hospital about 45 to 60 minutes after this incident. At the time of admission the patient was alert and responsive but complained of epigastric pain. A gastric lavage with starch water and magnesium sulfate was carried out immediately. During this procedure, the patient gradually became more and more unresponsive, and by 5:20 a. m. he was comatose. A half hour later he had a grand mal convulsion associated with fecal incontinence.

Physical Examination.—Blood pressure was 110 mm. Hg systolic and 70 mm. Hg diastolic. The pulse was irregular at a rate of 72 per minute, and the respirations were 16 per minute. The patient was in deep coma, unresponsive to painful stimuli. The skin was warm and dry. There was dusky cyanosis of the nailbeds and lips. The pupils were constricted but reacted normally to light. The neck was supple. Lungs were completely clear throughout. Examination of the heart revealed the point of maximal impulse to be well localized in the fifth left intercostal space medial to the midclavicular line. There was a normal sinus rhythm, with frequent ventricular premature beats (about 16 to 20 per minute). The heart tones were of poor quality. No murmurs were heard. The abdomen was negative. Neurological examination revealed the cranial nerves to be intact, as far as could be determined. The abdominal reflexes were absent, and the Babinski sign was present bilaterally. There were frequent chewing movements of the jaws. The remainder of the examination was not remarkable. An electrocardiogram obtained at this time showed the rate to be 72 and showed occasional ventricular premature contractions, a prolonged Q-T interval, the QRS complex negative in lead 1, the T wave negative in leads 2, 3, and aVF, and the T wave notched in leads V_2, V_4, and V_6. This was interpreted as showing right axis deviation, ventricular premature contractions, and evidence of diffuse myocardial abnormality.

Drug Therapy.—The patient was given oxygen by nasal catheter and procainamide (Pronestyl) hydrochloride, 500 mg. in 500 cc. of 5% dextrose in water, intravenously. During the next four hours, the heart sounds improved in quality and the rhythm was completely regular. Phenobarbital sodium or amobarbital (Amytal) sodium was used to control the signs of cortical irritability. Eight hours after admission, the patient vomited some dark brown material, which gave a chemical reaction for blood by the benzidine test. Examination at this time revealed that the cyanosis had disappeared and the vital signs were normal. The skin was flushed. The heart seemed dilated as evidenced by a very diffuse point of maximum impulse, 2 or 3 cm. outside the midclavicular line. Coma persisted. The neurological signs mentioned above were again demonstrated.

During the next 12 hours, the patient became very restless, thrashing about in bed. There were frequent episodes of severe carpopedal spasm, while at other times all the muscles of the body became very spastic and it seemed that another grand mal seizure was imminent. These periods of neuromuscular hyperactivity were temporarily controlled by intravenous therapy with calcium gluconate, 10 cc. of a 10% solution. On the morning of the second day, acute pulmonary edema supervened. The patient was digitalized with lanatoside C. The pulmonary edema cleared readily, but coma persisted. The pupils were small and fixed to light, and the respiratory rate increased to 40 per minute. Blood pressure was 100/70 mm. Hg and the pulse 160 per minute and feeble. An electrocardiogram taken at this point revealed supraventricular tachycardia and the evidences of diffuse myocardial abnormality noted above. There were no premature beats. During the ensuing four hours, the pulse rate rose to 180 per minute and the blood pressure dropped to 84/0 mm. Hg. Cheyne-Stokes respirations became evident, and the heart became further enlarged. Suction of the upper respiratory tract had to be carried out frequently, and an endotracheal tube was inserted. Because of the hypotension, levarterenol (Levophed) bitartrate therapy, 4 mg. in 1,000 cc. of 5% dextrose in water, was started by intravenous drip. This produced no appreciable effect on the blood pressure, although the pulse slowed a little and seemed stronger. At 11:30 p. m. of the second day, examination showed no change in the physical findings, except that the pupils once again reacted to light. Levarterenol bitartrate therapy was discontinued, and procainamide hydrochloride, 500 mg. in 500 cc. of 5% dextrose in water, was again administered intravenously. Mephentermine (Wyamine) sulfate therapy, 15 mg. every two hours by intramuscular injection, was tried. Calcium gluconate was given repeatedly to control the carpopedal spasm.

Condition of Patient.—On the third day the clinical picture and forms of therapy remained unchanged except for a further drop in blood pressure to the point of being unobtainable. Another electrocardiogram showed no important changes when

6. Chenoweth, M. B.: Monofluoroacetic Acid and Related Compounds, J. Pharmacol. & Exper. Therap. 97: 383-424, 1949.

7. (*a*) Footnote 1. (*b*) Footnote 4. (*c*) Harrisson, J. W. E., and others: Acute Poisoning with Sodium Fluoroacetate (Compound 1080), J. A. M. A. 149: 1520-1522 (Aug. 23) 1952.

compared with the previous ones, except for the addition of digitalis effect. The temperature continued to rise and reached a maximum of 104.6 F (40.3 C) late on the third day. On the fourth hospital day there was a tremendous increase in the amount of tracheobronchial secretions, which necessitated almost constant suctioning. On this day it was decided to alternate 5% alcohol in 10% dextrose in water intravenously with procainamide hydrochloride intravenously. This treatment was carried on through the fifth day, when the patient's condition seemed to improve a little. The blood pressure was obtainable at 100/60 mm. Hg, though the pulse remained rapid (180 per minute) and feeble. The heart sounds remained "flabby" in character. The extremities became warmer and had a good color. The muscular and carpopedal spasms decreased remarkably after the start of intravenous therapy with alcohol. On the night of the fifth day, the tracheobronchial secretions became so copious and tenacious that an adequate airway was impossible without a tracheostomy. This procedure was therefore carried out. Thick yellowish-white mucoid material was suctioned from the lower trachea, and the airway immediately sounded clear and dry. Examination of the lungs at this time revealed them to be well aerated throughout. Coma, rapid and feeble heart action, hypotension, the neurological signs noted above, and a good urinary output remained the principal features of the clinical course, together with a steadily rising temperature. At 3 a. m. on Jan. 6, 1954 (the sixth hospital day), the temperature reached 108 F (42.3 C) in spite of all measures to reduce it. From this point on the patient's respirations became extremely labored and rapid, the blood pressure once again was unobtainable, and the pulse increased to such a rapid rate that it was impossible to count it with any degree of accuracy. At 8 a. m. on Jan. 6, the patient died.

Postmortem Examination.—Postmortem examination revealed mediastinal emphysema, obvious and moderate in amount. The lungs were heavy (right 950 gm., left 750 gm.), edematous, and congested, but there was no frank consolidation. The bronchi were hemorrhagic but without exudate. Hemorrhagic-appearing mucosa was noted in the stomach but not in the esophagus. The spleen weighed 320 gm. and was red in color and firm to the touch. The right kidney contained a 2 mm., red-yellow area just beneath the surface. Other than congestion, the kidneys were normal. Crepitation could be felt and air bubbles could be seen in the adventitia of the aorta and immediate branches. The brain weighed 1,600 gm. and showed marked edema, with flattening of sulci and upward herniation of the cerebellum through the tentorium. Culture from the heart blood showed a moderate growth of hemolytic Micrococcus pyogenes var. aureus, coagulase positive. The remaining organs were grossly unremarkable.

Microscopic examination of the esophagus showed denudation of the epithelium here and there, with a coagulation necrosis present and minimal acute inflammatory reaction. The stomach mucosa was intact and essentially not remarkable. The lungs revealed a striking bronchopneumonia, with marked alveolar hemorrhages, congestion, and small clumps of bacteria. The brain contained small perivascular hemorrhages and changes in the ganglion cells compatible with the general cerebral edema. Finally, there was a small recent infarction in the cortex of the right kidney. Portions of liver, brain, kidney, pericardiac fluid, gastric contents, blood, heart, urine, and bile were examined for fluoride by the sodium silicofluoride test. The bile alone contained detectable amounts—0.02 mg. per 100 cc. by the zirconium-alizarin red test. The final anatomic diagnosis made was poisoning with sodium fluoroacetate; bronchopneumonia, with hemolytic septicemia due to a pyogenes var. aureus organism; focal infarction of right kidney; and mediastinal emphysema.

COMMENT

Since there is no known antidote to sodium fluoroacetate, therapy must be directed toward prevention of (1) serious cardiac arrhythmias, (2) central nervous system irritability, (3) peripheral vascular collapse, and (4) the general complications encountered in any patient who is comatose. In our recent experience with this disease entity, we have found certain agents that proved to be of definite, though short-lived, benefit to the patient. Myocardial irritability responded, at least temporarily, in a rather striking manner to the use of intravenously given procainamide hydrochloride, which completely abolished the premature ventricular contractions. Clinically, there was a distinct improvement in the quality of the heart sounds during and after the use of this drug. Several writers have agreed that procaine hydrochloride should be given by the intracardiac route in the case of ventricular fibrillation, known to be one of the immediate causes of death in human beings subjected to the poison. The ectopic beats, presumably the first signs of myocardial irritability, which might have progressed to more serious arrhythmias, such as ventricular tachycardia and ventricular fibrillation, were controlled by the use of procainamide hydrochloride. We feel that the possibility of death by ventricular fibrillation was prevented by use of this drug. The use of a digitalis preparation in the face of what was known to be a "toxic myocarditis" might give rise to some discussion. We had hoped to avoid the use of this substance, but our hand was forced by the intervention of acute pulmonary edema representing the most serious threat to life at the time it occurred. Short-acting lanatoside C was used because it was felt that this would relieve the pulmonary edema, which it did, without producing a cumulative cardiotoxic effect. There seemed to be no ill effects on cardiac irritability produced by this agent.

Secondly, the central nervous system manifestations, carpopedal spasm and generalized muscular hyperirritability, had to be dealt with. The barbiturates, calcium gluconate and magnesium sulfate, seemed to provide transient alleviation of these manifestations. With the institution of intravenous therapy with alcohol, these signs disappeared entirely and rather remarkably. It would seem likely, therefore, that this agent might, along with procainamide hydrochloride, be valuable in the therapy of this condition. In the third place, it was necessary to attempt to prevent death due to peripheral vascular collapse. In order to combat shock and to maintain an adequate renal blood flow, we used the vasodepressor drug mephentermine sulfate. We were thus able to maintain the patient's blood pressure above 100 mm. Hg systolic for a good part of the time and a good urinary output throughout the patient's hospital course. Finally, as in all cases of coma, alert nursing care was essential. Constant turning, frequent suctioning of the nasopharyngeal and even the tracheal and bronchial passages, use of alcohol sponges for fever, and nearly constant checking of the patient's vital signs were some of the more important of the many duties falling under the term nursing care.

In this case of fluoroacetate poisoning we considered, also, the use of agents recommended in the literature, such as magnesium sulfate in doses of 50 mg. per kilogram. This is known to prevent death in rats if given intramuscularly before or immediately after the ingestion of the poison. We also considered the use of monoacetin, which provides acetate and, thereby, theoretically produces its beneficial effect by this mechanism. We were advised by personal communication [8] that these

8. Fairhall, L.: Personal communication to the authors.

agents were usually helpful only immediately after the intake of the poison; therefore, we relied on the therapy outlined herein and used much smaller doses of magnesium sulfate. Monoacetin was not available to us. It has also been suggested that chlorpromazine might be useful in that it provides an "artificial hibernation," which reduces central nervous system and cardiac irritability, as well as lowers the temperature.

SUMMARY

Certain drugs can be used with benefit in a case of sodium fluoroacetate poisoning. These are (1) procainamide (Pronestyl) hydrochloride for cardiac arrythmias, (2) intravenously used alcohol for central nervous system irritability, and (3) vasodepressor drugs such as mephentermine (Wyamine) sulfate to maintain blood pressure. In the future these drugs, which we feel prolonged the life of our patient, should be tried in cases of sodium fluoroacetate poisoning, as they may save the lives of patients who have ingested smaller amounts of the poison.

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TRANSILLUMINATORS AND ILLUMINATED RETRACTORS FOR RETINAL DETACHMENT AND SURGERY

Conrad Berens, M.D., New York

The need for a compact and practical source of illumination for certain eye operations and for diagnosis instigated the development of a set of plastic retractors and transilluminators. These devices have been found to be useful and practical for retraction illumination, focal illumination, and transillumination of the eyeball.

A set of five tips, a sturdy flashlight handle, batteries, and an extra light bulb are encased in a compact leather case (see figure). The flashlight handle is made in two sizes. The small handle accommodates the no. 912 Eveready batteries, and the larger handle accommodates the regular no. 915 Eveready batteries, as well as the new long-life mercury batteries. The handles are machined from aluminium and are clear anodized, to prevent tarnishing. They contain no springs nor switches to wear out and are controlled by turning the base of the small flashlight and the base or top of the larger one. The three metal parts of the flashlight may be boiled or sterilized in alcohol and the inexpensive standard bulb and batteries inserted after sterilization. Three of the plastic light tips have a metallic mirror coating and are covered with plastic lacquer, allowing light to emerge only at the light-emitting surface.

The illuminated retractor (figure, *A*) is a modification of a retractor previously described [1] and has proved invaluable in operations on the retina and the inferior oblique muscle,[2] in orbital operations, and especially in evisceration of the eyeball.[3] The curved plastic tip (figure, *B*) permits light to emerge from the tip of the inner surface of the curve and is useful for transillumination of the posterior part of the globe in cases where tumors are suspected and for locating posterior retinal tears. The plastic tip, which is bent at a right angle (figure, *C*), is especially useful in examining the eye and transilluminating teeth and has been recommended as an accessory for a pocket flashlight for physicians.[4] The focal illuminator (figure, *D*) provides oblique illumination for the eye and may also be used in the examination for Purkinje vascular images. A cobalt blue glass tip (figure, *E*) is used for examining a fluorescein-stained cornea. The plastic tips may be sterilized in C. R. I. germicide (methyldodecylbenzyltrimethyl ammonium chloride, 17.5%, and inert ingredients, by weight, 82.5%). Neither alcohol nor boiling water should be used for sterilization of the plastic parts.

Transilluminators and illuminated retractor for retinal detachment and muscle and tumor surgery. *A*, plastic retractor; *B*, transilluminator for posterior part of the eyeball; *C*, transilluminator for anterior part of the eyeball; *D*, focal illuminator; and *E*, cobalt blue glass illuminator.

The advantages of the flashlight include the sturdy construction with no springs or switch, which wear out, and the fact that the flashlight may be sterilized without harming the instrument. Standard bulbs and batteries are used and offer no problem in replacement. The various plastic tips provide excellent illumination for retinal and muscle and tumor surgery and focal illumination for examination. The plastic tips are easily changed and present no problem so far as breakage is concerned. The set of transilluminators and illuminated retractors, flashlight, batteries, and the cobalt glass tip are fitted into a compact leather case. The instrument is also available with a cord for use with a rheostat.

708 Park Ave.

This study was aided by a grant from the Ophthalmological Foundation, Inc., and the Department of Research of the New York Association for the Blind.

The apparatus described is made by R. O. Guldeu, Philadelphia 20. The long-life mercury batteries are made by P. R. Mallory & Co., Inc., North Tarrytown, N. Y. The C. R. I. germicide is made by Storz Instrument Co., St. Louis.

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Review Article

Sodium Fluoroacetate Poisoning

Proudfoot, Alex T[1]; Bradberry, Sally M[1 2]; Vale, J Allister[1 2]

Abstract

Sodium fluoroacetate was introduced as a rodenticide in the US in 1946. However, its considerable efficacy against target species is offset by comparable toxicity to other mammals and, to a lesser extent, birds and its use as a general rodenticide was therefore severely curtailed by 1990. Currently, sodium fluoroacetate is licensed in the US for use against coyotes, which prey on sheep and goats, and in Australia and New Zealand to kill unwanted introduced species.

The extreme toxicity of fluoroacetate to mammals and insects stems from its similarity to acetate, which has a pivotal role in cellular metabolism. Fluoroacetate combines with coenzyme A (CoA-SH) to form fluoroacetyl CoA, which can substitute for acetyl CoA in the tricarboxylic acid cycle and reacts with citrate synthase to produce fluorocitrate, a metabolite of which then binds very tightly to aconitase, thereby halting the cycle. Many of the features of fluoroacetate poisoning are, therefore, largely direct and indirect consequences of impaired oxidative metabolism. Energy production is reduced and intermediates of the tricarboxylic acid cycle subsequent to citrate are depleted. Among these is oxoglutarate, a precursor of glutamate, which is not only an excitatory neurotransmitter in the CNS but is also required for efficient removal of ammonia via the urea cycle. Increased ammonia concentrations may contribute to the incidence of seizures. Glutamate is also required for glutamine synthesis and glutamine depletion has been observed in the brain of fluoroacetate-poisoned rodents. Reduced cellular oxidative metabolism contributes to a lactic acidosis. Inability to oxidise fatty acids via the tricarboxylic acid cycle leads to ketone body accumulation and worsening acidosis. Adenosine triphosphate (ATP) depletion results in inhibition of high energy-consuming reactions such as gluconeogenesis. Fluoroacetate poisoning is associated with citrate accumulation in several tissues, including the brain. Fluoride liberated from fluoroacetate, citrate and fluorocitrate are calcium chelators and there are both animal and clinical data to support hypocalcaemia as a mechanism of fluoroacetate toxicity. However, the available evidence suggests the fluoride component does not contribute.

Acute poisoning with sodium fluoroacetate is uncommon. Ingestion is the major route by which poisoning occurs. Nausea, vomiting and abdominal pain are common within 1 hour of ingestion. Sweating, apprehension, confusion and agitation follow. Both supraventricular and ventricular arrhythmias have been reported and nonspecific ST- and T-wave changes are common, the QTc may be prolonged and hypotension may develop. Seizures are the main neurological feature. Coma may persist for several days. Although several possible antidotes have been investigated, they are of unproven value

in humans. The immediate, and probably only, management of fluoroacetate poisoning is therefore supportive, including the correction of hypocalcaemia.

September 19, 2011

12 Held in Sale of Pest Poisons, One 60 Times as Potent as the Legal Limit

By **WILLIAM K. RASHBAUM**

The investigation began with a vial of blue-green liquid. Roughly two inches tall, it came in a yellow and blue box covered with Chinese characters and, in English, the words "The cat be unemployed."

It was rat poison, illegal and highly toxic.

The pesticide, which was apparently smuggled into the United States from China, contained one deadly ingredient in a concentration almost 61 times as great as what federal regulations allow, according to court papers.

The chemical, brodifacoum, is so dangerous, officials said, that its use is illegal in urban areas unless it is applied by licensed professionals wearing protective gear and using special equipment. Federal regulators have recently moved to further restrict its use, in part because its ingestion could kill a small child.

Several of the vials were among about 6,000 packages of rat and cockroach poison seized from shops and street vendors in and around Chinatown during a five-month undercover investigation into the sale of illegal pesticides, state and local officials announced at a news conference Monday.

The vials, they said, first came to the attention of the authorities because a woman who had bought one in the East Broadway Mall in Chinatown last year later mistook the pesticide for medicine, consumed it and became seriously ill, losing two-thirds of her blood volume, according to the court papers. Brodifacoum is an anticoagulant that kills rodents by causing them to bleed to death internally. Another chemical in one of the pesticides, sodium fluoroacetate, is a metabolic poison used to kill coyotes.

The inquiry, which involved a half-dozen agencies that enforce laws regarding pesticides, culminated last week when investigators executed 14 search warrants, mostly in Chinatown,

arrested 12 people on federal and state criminal charges — all of them misdemeanors — and conducted nearly four dozen civil inspections, officials said.

The agencies involved in the investigation, including the offices of the district attorney and the United States attorney in Manhattan, the Environmental Protection Agency and the State Department of Environmental Conservation, said their investigation highlighted the widespread sale of toxic pesticides in densely populated neighborhoods where vermin abound.

"All across the city we find products like these," Judith Enck, the E.P.A.'s regional administrator, said at the news conference, referring to a display of colorful unregulated pesticides that she said could easily be confused for children's toys or candy. "People and businesses that make and sell these products are playing Russian roulette with people's health."

Many of the products, she said in an interview, are carcinogenic or poisonous to nerve cells and "have the potential to do long-term damage."

The E.P.A., Ms. Enck added, "is particularly concerned about children coming into contact with these products, because children are particularly vulnerable to the toxic impact — their bodies are still developing."

During the last five months, undercover investigators bought illegal pesticides in shops on Madison, Mott, South Eldridge and Pike Streets in Chinatown, the officials said.

Ten of those arrested last week were charged in state court and will be prosecuted by the Manhattan district attorney's office; two others — one identified in court papers as a wholesaler of the illegal pesticides, the other a grocery store owner who was charged with selling thousands of packets of the products to undercover investigators — will be prosecuted in federal court by the office of the United States attorney in Manhattan.

The Manhattan district attorney, Cyrus R. Vance Jr., whose investigators seized the majority of the illegal pesticides, suggested that it would be worth considering legislation to allow prosecutors to seek harsher penalties for such crimes, based on the concentrations and quantities sold.

"The rodenticides and roach killers that were seized as part of this investigation," Mr. Vance said, "are dangerous, unregulated products that contain chemicals so toxic they exceed government regulation scores at times."

And, he added, they "are particularly dangerous to kids because they look and smell like cookies or other objects that would attract the human touch."

Preet Bharara, the United States attorney in Manhattan, said in a statement that "these defendants were literally peddling poison to an unwitting public, putting the health and safety of their customers and their families in jeopardy."

Children are especially vulnerable because many pesticides are placed on floors and the children sometimes place bait pellets in their mouths, according to Adrian J. Enache, a toxicologist who leads the E.P.A.'s pesticides program in New York.

The American Association of Poison Control Centers receives 12,000 to 15,000 reports each year of children younger than 6 being exposed to these kinds of pesticides. But Ms. Enck and other officials said it was hard to gauge the scope of the problem and its impact on children because many cases of poisoning go unreported. Ms. Enck said that when children exhibit symptoms that include eye and skin irritations and nausea, parents are often unaware that the pesticides are at fault.

The inquiry is continuing, with investigators focusing on identifying and tracking down the sources of the unregistered pesticides, according to David G. McLeod Jr., the assistant special agent in charge of the E.P.A.'s criminal investigation division in New York.

The wholesaler, Jai Ping Chen, 43, was charged with five counts of conspiracy and four counts of selling unregistered pesticides. The grocery store owner, Cheng Yan Huang, 56, was charged with nine counts of similar crimes. If convicted, both face a year in prison for each count.

Mr. Chen's lawyer, Adam D. Perlmutter, declined to comment; Martin S. Cohen, a lawyer who represents Mr. Huang, did not respond to telephone and e-mail messages.

The 10 men and women charged in state court face multiple misdemeanor charges, in some cases hundreds of counts, and while the jail time is negligible, many violations carry a maximum fine of $5,000 per count.

Ms. Enck said she believed people bought the poison because they thought it was "the strongest and most potent product."

She added: "Unfortunately, these are readily available and there is an assumption that if they're sold in stores, they're legal. And another reason is they are relatively cheap."

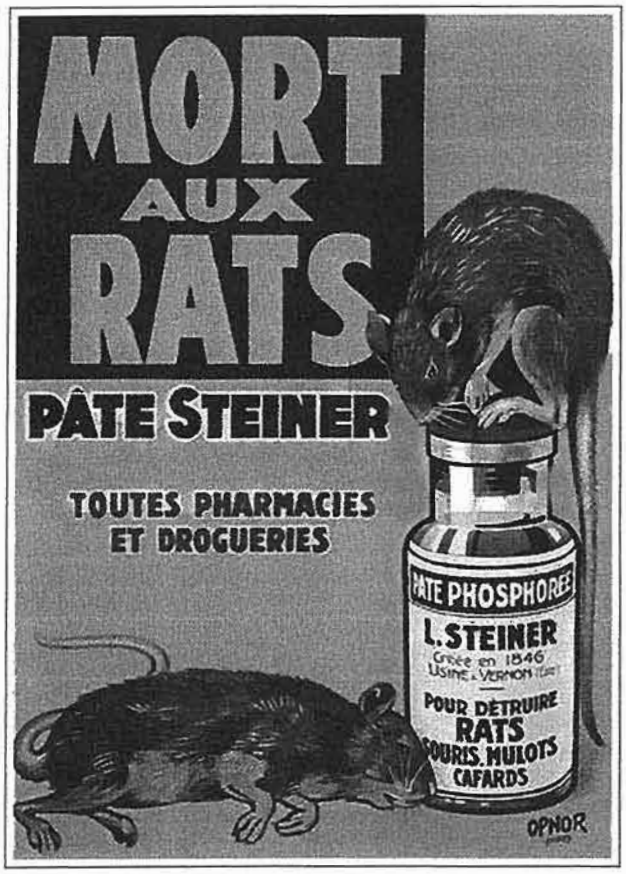
Terrierman's Daily Dose

* See Pages 3+4

Information on working terriers, dogs, natural history, hunting, and the environment, with occasional political commentary as I see fit. This web log is associated with the Terrierman.com web site. Please see this web site for more information on working terriers, or to order the book.

SATURDAY, APRIL 26, 2008

Rat Poison and Wildlife Conservation



Rats are responsible for more animal extinctions than any other cause. The chief victims have been birds native to small tropical islands. Rats prey on both eggs and baby birds and, in some cases, adult flightless birds as well.

For this reason, rat poison may be the the single most important equipment in the world of bird conservation -- though using the right type in the right manner and in the right location is critical. In the wrong hands, rat poison can not only kill rats and mice -- it can kill

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birds, small native animals, fish, pets and even humans.

Winter poisoning is more effective than summer baiting, and effective use of rodenticides requires acclimating the rats to bait stations and the carrier food source (boiled eggs, corn meal, canary seed, water, etc.)

Rat eradication efforts benefit greatly by periodic switching of poison types, from warfarin (maintain for several months) to zinc phosphate, for example. Ultimately, effective long term rat eradication requires either removing food sources or maintaining a nearly-permanent poisoning regime.

A quick history of the most common types of rat poisons:

- **Red Squill** (*Urginea or Scilla maritima*) is a flowering plant native to the Mediterranean and was used as a rat poison as early as 1500 BC. The bulb of the red squill plant weighs up to four pounds and is sliced and dried before it is set out for rat consumption. Red squill is a safe poison because non-target animals that consume it invariably vomit to rid themselves of the toxin. Rats are unable to vomit, however, and so cannot purge their system of the toxins which eventually paralyze their hearts. Red squill has a very strong bitter taste and works well for a single time baiting situation, but rats quickly learn to stay away from it after that first dosing.

- **Strychinine is a very old poison** and may have been used by Alexander the Great's wife to poison him after he took a homosexual lover (who was poisoned at the same time). Strychnine originates from a small tree-like plant (*Strychnos*) once endemic to the Indus valley of India. A similar plant of the same family (but which grows in vine form) is used by the Indians of the Amazon to make curare -- a poison used to kill monkeys with poison-tipped darts. Rats tend to shy away from strychnine, but it is very effective on mice. Because strychnine is easy to abuse, and safer rodenticides are far more effective, access to this poison is now strictly controlled.

- **Arsenic is another very old poison.** Aristotle made reference to the poison "sandarach" (arsenic trisulfide) in the 4th century B.C. Arsenic was commonly sold as "Ratsbane" by the 1500s, and is mentioned by Shakespeare in Henry V, Part II ("I had as lief they would put ratsbane in my mouth as offer to stop it with security.") Arsenic is less effective and more toxic than other readily available rodenticides and is now almost never used for vermin work.

- **Warfarin is a modern slow-kill repeat-bait poison and among the safest and most effective rat poisons in common use.**

Warfarin was discovered after Canadian cattle ate improperly stored sweet clover and began to hemorhage and die. In 1930 the active ingredient "coumarin" was isolated from this clover. In 1940 the Wisconsin Alumni Research Foundation patented a coumarin compounded called Warfarin (named after the foundation's initials). In 1952 warfarin was first used as an anticoagulant on humans, and today it (or some other coumarin derivative) is used by patients with artificial heart valves or who are in danger of thrombosis (blood clots). When used as a rodenticide, warfarin should be set out in feed-on-demand bait stations for at least two weeks. Other anticoagulants that work about the same as warfarin are brodifacoum, bromadiolone, chlorophacinone, diphacinone, fumarin, pival, and PMP. Some rat and mouse populations have become resistant to warfarin and other anti-coagulants -- a good reason to temporarily discontine warfarin after a few months and switch to zinc phosphate or another quick-killing raticide.

- **Zinc Phosphate is an effective quick-kill poison and is readily available.** It has an offensive odor (it smells like garlic) and is unattractive in color. Rats and mice seem to be attracted by the odor, however, and all species of rats and mice accept it. Zinc phosphide is not absorbed through the skin while mixing, and is not water soluable. Very occassionally animals die from eating the carcasses of rats or mice that have been killed with zinc phosphide, but this is so rare that zinc phosphide is listed as only a mildly hazardous rodenticide. Cause of death is heart failure.

- **Norbormide (S-6999) is a new single-dose rat poison that is essentially nontoxic to humans and is odorless.** Norbormide appears to be nontoxic to birds and other mammals including mice. It kills by contricting blood vessels and comes as a white powder. It is sold as Raticide, Raticate and Shoxin.

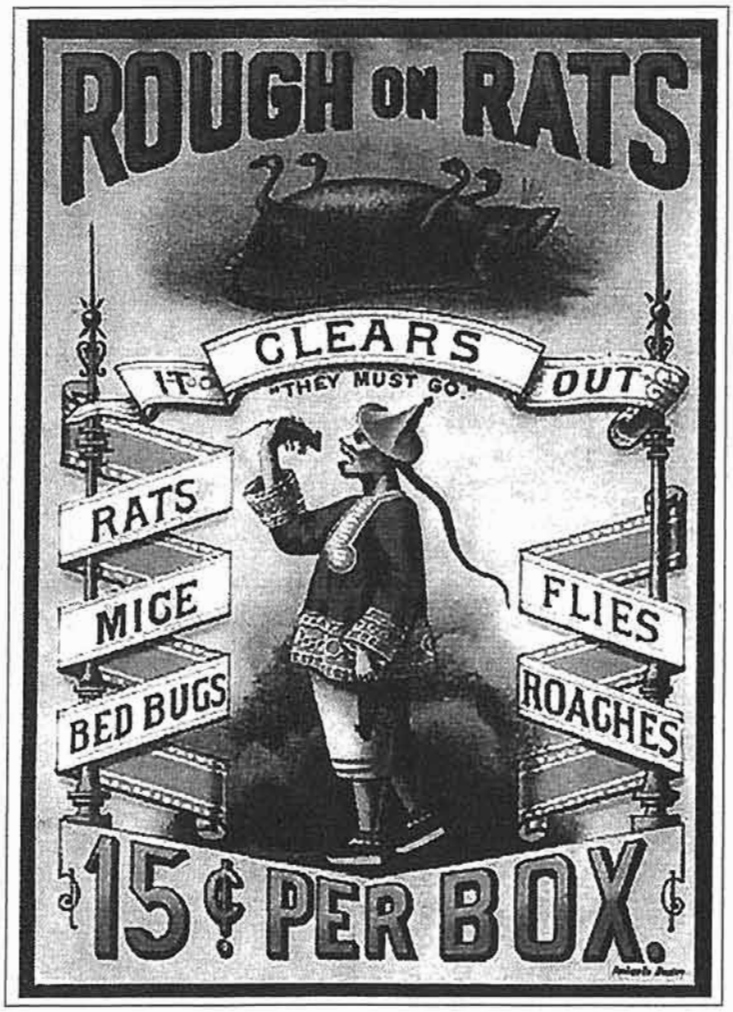
- **Vacor 1080 (Sodium fluoroacetate) is an extremely powerful single-dose rodenticide.** Death normally occurs 4 to 8 hours after ingestion, and little or no bait shyness develops since death generally follows ingestion. Vacor is available in a formulated ready-to-use bait mixture for licensed professional rat exterminators, but is generally not available to the lay public as it is a poison that is powerful enough to kill almost anything else that ingests the bait.

- **Sodium Fluoroacetate or 1081, is one of the most effective rodenticides known.** It is virtually tasteless and odorless and kills in 1 to 8 hours. No tolerance or bait shyness develops. The drawbacks are that it is highly toxic to all animals, has no antidote, and has a high degree of secondary poisoning for animals eating rats or mice killed by the 1080 poison. As a result, 1080 is classified as extremely hazardous and is

available for use only by licensed professional applicators. Cause of death is heart paralysis.

- **Fumigants such as methyl bromide and hydro-cyanide gas** are fast and effective controls for rats and mice in burrows or tightly closed buildings but should never be used by anyone other than licensed and trained professionals.

- **Other Poisons No Longer Used.** These poisons are more dangerous and not as effective as their readily-available alternatives: Barium carbonate, phosphorous paste (pictured in the French rat poison bottle at the top of this page), and Thallium sulfate.



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Common pesticides use fluoridation chemicals as their main ingredient

Fluoridation chemicals are used in pesticides and in toothpaste! Here's a few examples.

☐ Product: **REXCO ROACH DEATH**
EPA Registration Number: 03025708880
This pesticide is used as a: Insecticide and Miticide
This pesticide's toxicity code is 1, which corresponds to a toxicity category of Danger.
Active Ingredient: SODIUM FLUORIDE 40% (% by mass)

☐ Product: **ROACHES LAST MEAL**
EPA Registration Number: 01080000001
This pesticide is used as a: Insecticide and Miticide
Active Ingredients: SODIUM FLUORIDE 40%

☐ Product: **BUG-NO-MOR** (POWDER)
EPA Registration Number: 02524207637
This pesticide is used as a: Insecticide and Miticide
Active Ingredients: PYRETHRUM 50%, **SODIUM FLUORIDE** 45%

Product: **OSMOPLASTIC WOOD PRESERVING COMPOUND**
EPA Registration Number: 00300800056
This pesticide is used as a: Fungicide
Active Ingredients: SODIUM FLUORIDE 44.42%, CREOSOTES 45.62%

The following is am abbreviated list of additional pesticides which also contain SODIUM FLUORIDE. The numbers indicate the percentage of SODIUM FLUORIDE in the products.

ADZ-PAD 52.2%
BUG-NO-MOR (POWDER) 45%
OSMOPLASTIC-D WOOD PRESERVING COMPOUND 44.42%
OSMOPLASTIC SD WOOD PRESERVING COMPOUND 44.42%
COP-R-PLASTIC WOOD PRESERVING COMPOUND 44.4%
TIMPREG I 40.74%
KNOX-EM ROACH POWDER 40%
NOXX ROACH POWDER 40%
REXCO ROACH DEATH 40%
PROFESSIONAL ROACH POWDER 40%
RED WING PEST KIT ROACH POWDER 40%
TRITOX PRESERVATIVE PASTE 40%
PERKERSON'S - KILL ALL ROACHES & WATER BUGS 40%
PEST HOUSE ROACH POWDER 40%
JANO ROACH POWDER "KILLS-EM QUICK" 40%
MOMAR FORMULA 357 ROACH POWDER CONCENTRATE 40%
RHODO ROACH RIDDER 40%
KILL KOTE SPECIAL ROACH POWDER 40%
ELCO ROACH AND ANT POWDER 40%

SWEENEY'S SODIUM FLUORIDE ROACH KILLER 40%
KILL-KO ROACH POWDER 40%
ROACH POWDER E227 40%
NOTT'S ROACH POWDER 40%
TRIPLE-X ANT ROACH AND WATERBUG POWDER 40%
HUMCO BRAND SODIUM FLUORIDE (TINTED) 40%
FLIPO SPECIAL ROACH AND WATERBUG KILLER 40%
HUB STATES READY-KILL ROACH POWDER 40%
POLE-TOX WOOD PRESERVING COMPOUND 40%
RITTER'S ROACH POWDER 40%
ROACHES LAST MEAL 40%
SCRAMO 39.5%
ERADICO ROACHPOWDER 39.5%
GETEM ROACH POWDER 39.2%
RED SEAL ROACH POWDER 39%
SODIUM FLUORIDE 40 39%
ROCHEK ROACH POWDER 39%
DRO SODIUM FLOURIDE KILLS ROACHES 39%
CERTOX ROACH POWDER CODE NO. FP-1 39%
DUNCAN'S ROACH AND ANT DESTROYER 39%
DAND L TINTED BLUE-AN INSECTICIDE 39%
SODIUM FLUORIDE 40 39%
SPRAYALL INSECTICIDE POWDER 39%
REDWOOD ROACH POWDER 39%
HUB STATES SODIUM FLUORIDE 39 39%
OKAY SPECIAL ROACH POWDER 38.8%
CE CO INSECT POWDER 38.8%
FLUO-PYRE ROACH POWDER 38.8%
RESIDEX ROACH POWDER 38.8%
CENOL WATERBUG AND ROACH POWDER 38.8%
PYRETHRUM - SODIUM FLUORIDE POWDER SPECIAL #2 38.8%
PRENTOX BLUE POWDER 38.8%
PYRETHRINS SODIUM FLUORIDE POWDER 38.8%
CERTOX FP-11, ROACH POWDER 38.8%
ANDEX INSECT POWDER 38.8%
FORMULA A-1 ROACH POWDER 38.8%
FLUO-PYRE ROACH POWDER 38.8%
TORNADO ROACH POWDER 38.8%
CHEM-TOX WATERBUG & ROACH KILLER 38.8%
SHUR-DETH 38.8%
OKAY SPECIAL ROACH POWDER 38.8%
MINIMAX ROACH POWDER 38%
PATOX POLE TREATING BANDAGE I 37.9%
PATOX POLE TREATING BANDAGE I 37.9%
PROFESSIONAL ROACH CONTROL 35%
COUNTY PEST CONTROL ROACH POWDER 35%
SODIUM FLOURIDE POWDER 35%
STEPHENSON CHEMICALS SPECIAL RESISTANT ROACH POWER 35%
COOK'S RESISTANT INSECT POWDER 35%
SODIUM FLOURIDE POWDER 35%
PROFESSIONAL ORKIN SPECIAL FORMULA R-333 DD GRADE
INSECT POWDER 33.33%
PROFESSIONAL ORKIN SPECIAL FORMULA PFT 33.33%
ORIGINAL PROFESSIONAL DO IT YOURSELF EXTERMINATOR'S
KIT FORMULA 401 31.8%
OSMOPLASTIC-F 30%
WIL-KIL SILVER FISH BAIT 25%
ROBINSON ROACH DESTROYER 20%
OSMOBAND WOOD PRESERVATIVE BANDAGE 20%
TIMPREG PAK 15%
POLE-LIFE PRESRVATIVE PASTE TF 15%
C WOOD PRESERVATIVE AT-8242 15%
TIMPREG PAK POL-NU TYPE 15%
HOLLOW HEART CONCENTRATE 10.9%

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Product: ROACHES LAST MEAL
EPA Registration Number: 01080000001

This pesticide is used as a:

- INSECTICIDE
- MITICIDE

This pesticide is registered for unrestricted use.

This pesticide's toxicity code is 2, which corresponds to a toxicity category of Warning.

Active Ingredients in this Product	Percentage by Mass
SODIUM FLUORIDE	40%

Questions or Comments? Email:
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CHEMICAL PROFILES | Product Profile

Product: REXCO ROACH DEATH
EPA Registration Number: 03025708880

This pesticide is used as a:

- INSECTICIDE
- MITICIDE

This pesticide is registered for unrestricted use.

This pesticide's toxicity code is 1, which corresponds to a toxicity category of Danger.

Active Ingredients in this Product	Percentage by Mass
SODIUM FLUORIDE	40%
PYRETHRUM	.16%

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Product: RED WING PEST KIT ROACH POWDER
EPA Registration Number: 00672300005

This pesticide is used as a:

- INSECTICIDE
- MITICIDE

This pesticide is registered for unrestricted use.

This pesticide's toxicity code is 2, which corresponds to a toxicity category of Warning.

Active Ingredients in this Product	Percentage by Mass
SODIUM FLUORIDE	40%
MALATHION	4%
PYRETHRUM	.05%

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Product: REXCO ROACH DEATH
EPA Registration Number: 03025708880

This pesticide is used as a:

- INSECTICIDE
- MITICIDE

This pesticide is registered for unrestricted use.

This pesticide's toxicity code is 1, which corresponds to a toxicity category of Danger.

Active Ingredients in this Product	Percentage by Mass
SODIUM FLUORIDE	40%
PYRETHRUM	.16%

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CHEMICAL PROFILES | Product Profile

Product: ROACHES LAST MEAL
EPA Registration Number: 01080000001

This pesticide is used as a:

- INSECTICIDE
- MITICIDE

This pesticide is registered for unrestricted use.

This pesticide's toxicity code is 2, which corresponds to a toxicity category of Warning.

Active Ingredients in this Product	Percentage by Mass
SODIUM FLUORIDE	40%

Questions or Comments? Email:
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Fluoride overdose

Fluoride is a chemical commonly used to prevent tooth decay. Fluoride overdose occurs when someone accidentally or intentionally takes more than the normal or recommended amount of this substance.

This is for information only and not for use in the treatment or management of an actual poison exposure. If you have an exposure, you should call your local emergency number (such as 911) or the National Poison Control Center at 1-800-222-1222.

See also: Fluoride in diet

Poisonous Ingredient

- Fluoride

Where Found

Fluoride is found in many over-the-counter and prescription products, including:

- Certain mouthwashes and toothpastes
- Certain vitamins (Tri-Vi-Flor, Poly-Vi-Flor, Vi-Daylin F)
- Fluoridated water
- Sodium fluoride liquid and tablets

Fluoride may also be found in other household items, including

- Etching cream
- Roach powders

Note: This list may not be all-inclusive.

Symptoms

- Abdominal pain
- Abnormal taste (salty or soapy taste)
- Convulsions
- Diarrhea
- Drooling
- Headache
- Heart attack
- Irregular heartbeat
- Nausea
- Shallow breathing
- Slow heartbeat

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- Tremors
- Vomiting
- Weakness

Before Calling Emergency

Determine the following information:

- Patient's age, weight, and condition (for example, is the person awake or alert?)
- Name of the product (ingredients and strengths, if known)
- Time it was swallowed
- Amount swallowed

However, do NOT delay calling for help if this information is not immediately available.

Poison Control

The National Poison Control Center (1-800-222-1222) can be called from anywhere in the United States. This national hotline number will let you talk to experts in poisoning. They will give you further instructions.

This is a free and confidential service. All local poison control centers in the United States use this national number. You should call if you have any questions about poisoning or poison prevention. It does NOT need to be an emergency. You can call for any reason, 24 hours a day, 7 days a week.

See: Poison control center - emergency number

What to Expect at the Emergency Room

The health care provider will measure and monitor the patient's vital signs, including temperature, pulse, breathing rate, and blood pressure. Symptoms will be treated as appropriate. The patient may receive:

- Calcium or milk
- Methods or medicines to cause vomiting
- Tube through the mouth into the stomach to wash out the stomach (gastric lavage)

Outlook (Prognosis)

How well a patient does depends on the amount of poison swallowed and how quickly treatment was received. The faster a patient gets medical help, the better the chance for recovery.

The amount of flouride found in toothpaste is usually not swallowed in large enough amounts to cause harm.

References

Scalzo AJ, Blume-Odom CM. Hydrofluoric acid and other fluorides. In: Shannon MW, Borron SW, Burns MJ, eds. *Haddad and Winchester's Clinical Management of Poisoning and Drug Overdose*. 4th ed. Philadelphia, Pa: Saunders Elsevier; 2007:chap 90.

Update Date: 1/20/2010

Updated by: Jacob L. Heller, MD, MHA, Emergency Medicine, Virginia Mason Medical Center, Seattle, Washington. Also reviewed by David Zieve, MD, MHA, Medical Director, A.D.A.M., Inc.

FLUORIDE DOSAGE TOOTHPASTE VS. WATER

JADA Study Proves Fluoridation is Money down the Drain

NEW YORK, Sept. 29 /PRNewswire-USNewswire/ -- Children's cavity rates are similar whether water is fluoridated or not, according to data published in the July 2009 *Journal of the American Dental Association* by dentist J.V. Kumar of the NY State Health Department(1), reports NYSCOF.

In 2008, New York City spent approximately $24 million on water fluoridation ($5 million on fluoride chemicals)(1a). In 2010, NYC's fluoride chemicals will cost $9 million(1b).

Fluoride in water at "optimal" levels (0.7 - 1.2 mg/L) is supposed to reduce tooth decay without creating excessive fluorosis (fluoride-discolored and/or damaged teeth). Yet cavities are rampant in NY's fluoridated populations(1c).

Attempting to prove that fluorosed teeth have fewer cavities, Kumar uses 1986-1987 National Institute of Dental Research (NIDR) data which, upon analysis, shows that 7- to 17-year-olds have similar cavity rates in their permanent teeth whether their water supply is fluoridated or not (Table 1).

In 1990, using the same NIDR data, Dr. John Yiamouyiannis published equally surprising results in a peer-reviewed journal. He concluded, "No statistically significant differences were found in the decay rates of permanent teeth or the percentages of decay-free children in the F [fluoridated], NF [non-fluoridated], and PF [partially fluoridated] areas."(2).

Kumar divided children into four groups based on their community's water fluoride levels:

Less than 0.3 mg/L where 55.5% had cavities

From 0.3 to 0.7 mg/L where 54.6% had cavities

Optimal 0.7 to 1.2 mg/L where 54.4% had cavities

Over 1.2 mg/L where 56.4% had cavities

"Dr. Kumar's published data exposes more evidence that fluoridation doesn't reduce tooth decay," says attorney Paul Beeber, President, New York State Coalition Opposed to Fluoridation.

"It's criminal to waste taxpayers' money on fluoridation, while exposing entire populations unnecessarily to fluoride's health risks, especially when local and state governments are attempting to balance budgets by cutting essential services," says Beeber.

Analysis of Kumar's data: http://tinyurl.com/MoneyDownTheDrain

More information about fluoride and tooth decay:

http://www.fluoridealert.org/health/teeth/caries/fluoridation.html#surveys

References:

1) "The Association Between Enamel Fluorosis and Dental Caries in U.S. Schoolchildren," Kumar & Iida *Journal of the American Dental Association*, July 2009 (Table 1)

1a) http://www.scribd.com/doc/18235930/NYC-Fluoridation-Costs-2008-Feb-2-2009-Letter-Page-1

1b) http://www.council.nyc.gov/html/budget/PDFs/fy_10_exec_budget_dept_enviro_protection.pdf

1c) http://www.freewebs.com/fluoridation/fluoridationfailsnewyork.htm

2) *Fluoride: Journal of the International Society for Fluoride Research*
April 1990 (Volume 23, Issue 2, Pages 55-67) "Water Fluoridation & Tooth Decay: Results from the 1986-1987 National Survey of US Schoolchildren," by John A. Yiamouyiannis, Ph.D.

Contact: Paul Beeber, Esq 516-433-8882 nyscof@aol.com

http://www.orgsites.com/ny/nyscof

http://www.FluorideAction.net

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Study Proves Fluoridation is Money Down the Drain

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New York – Sept 2009 – Children's cavity rates are similar whether water is fluoridated or not, according to data published in the July 2009 Journal of the American Dental Association by dentist J.V. Kumar of the NY State Health Department (1).

In 2008, New York City spent approximately $24 million on water fluoridation ($5 million on fluoride chemicals) (1a). In 2010, NYC's fluoride chemicals will cost $9 million (1b).

Fluoride in water at "optimal" levels (0.7 – 1.2 mg/L) is supposed to reduce tooth decay without creating excessive fluorosis (fluoride-discolored and/or damaged teeth). Yet cavities are rampant in NY's fluoridated populations (1c).

Attempting to prove that fluorosed teeth have fewer cavities, Kumar uses 1986-1987 National Institute of Dental Research (NIDR) data which, upon analysis, shows that 7- to 17-year-olds have similar cavity rates in their permanent teeth whether their water supply is fluoridated or not (Table 1).

In 1990, using the same NIDR data, Dr. John Yiamouyiannis published equally surprising results in a peer-reviewed journal. He concluded, "No statistically significant differences were found in the decay rates of permanent teeth or the percentages of decay-free children in the F [fluoridated], NF [non-fluoridated], and PF [partially fluoridated] areas." (2).

Kumar divided children into four groups based on their community's water fluoride levels:

Less than 0.3 mg/L where 55.5% had cavities
From 0.3 to 0.7 mg/L where 54.6% had cavities
Optimal 0.7 to 1.2 mg/L where 54.4% had cavities
Over 1.2 mg/L where 56.4% had cavities

"Dr. Kumar's published data exposes more evidence that fluoridation doesn't reduce tooth decay," says attorney Paul Beeber, President, New York State Coalition Opposed to Fluoridation.

"It's criminal to waste taxpayers' money on fluoridation, while exposing entire populations unnecessarily to fluoride's health risks,

especially when local and state governments are attempting to balance budgets by cutting essential services," says Beeber.

More information about fluoride and tooth decay:
http://www.fluoridealert.org/health/teeth/caries/fluoridation.html#su...

References:

1) "The Association Between Enamel Fluorosis and Dental Caries in U.S. Schoolchildren," Kumar & Iida Journal of the American Dental Association, July 2009

1a) http://www.scribd.com/doc/18235930/NYC-Fluoridation-Costs-2008-Feb-2-...

1b)
http://www.council.nyc.gov/html/budget/PDFs/fy_10_exec_budget_dept_en...

1c) http://www.freewebs.com/fluoridation/fluoridationfailsnewyork.htm

2) Fluoride: Journal of the International Society for Fluoride Research
April 1990 (Volume 23, Issue 2, Pages 55-67) "Water Fluoridation & Tooth Decay: Results from the 1986-1987 National Survey of US Schoolchildren," by John A. Yiamouyiannis, Ph.D.

Contact: Paul Beeber, Esq nys...@aol.com
http://www.orgsites.com/ny/nyscof
http://www.FluorideAction.net

STATISTICAL ANALYSIS OF KUMAR's DATA:

Kumar's data: 6,720 children came from communities with water fluoride levels less than 0.3 mg/L

54.9% of the 3,921 children without fluorosis had tooth decay = 2,153
60.1% of 1,818 children with questionable fluorosis had tooth decay = 1,093
49.3% of 981 with fluorosis present had cavities = 484

TOTAL 6,720 TOTAL WITH CAVITIES 3,730

3,730 is 55.5% of 6,720 Therefore, 55.5% had cavities.

***

1,979 children tested lived in communities with from 0.3 to 0.7 mg/L Fluoride

54.7% of 1,084 children with normal teeth have cavities = 593
59% of 507 children with questionable fluorosis have cavities = 299
48.4% of 388 children with fluorosis present had cavities = 188

TOTAL 1,979 TOTAL WITH CAVITIES 1,080

1,080 is 54.6% of 1,979. Therefore, 54.6% of children living in 0.3 to 0.7 mg/L fluoridated communities have tooth decay

***

7,177 lived in optimally fluoridated areas (0.7 – 1.2 mg/L)

54.1% of the 2875 children without fluorosis had tooth decay = 1,555
53.8% 2493 children with questionable fluorosis had tooth decay = 1,341
55.7% 1809 with fluorosis present had cavities = 1,008

TOTAL 7,177 TOTAL WITH CAVITIES 3,904

3914 is 54.4% of 7177. Therefore 54.4% of children living in optimally fluoridated areas had cavities

***

813 children living in areas with greater than 1.2 mg/L Fluoride in their water

52.3% of 248 children with normal teeth had cavities = 130
64.1% of 236 children with questionable fluorosis had cavities = 151
54% of 329 children with fluorosis had cavities = 178

TOTAL 813 TOTAL WITH CAVITIES 459

56.4% of 813 children is 459 . Therefore 56.4% of children living in areas with more than 1.2 mg/L fluoride in the water have cavities

ADDITIONAL SCIENTIFIC EVIDENCE THAT FLUORIDATION FAILS TO REDUCE TOOTH DECAY

-- Achieving cavity-free status has little to do with fluoride intake, reports a study in the Fall 2008 Journal of Public Health Dentistry. Researchers explain that when fluoridation began in the 1940's, "it was believed that fluoride needed to be ingested early in life to provide [cavity] prevention...Today, evidence suggests that...the benefits of fluoride are mostly topical." (A)

-- Researchers reporting in the Oct 6 2007 British Medical Journal indicate that fluoridation, touted as a safe cavity preventive, never was proven safe or effective and may be unethical. (B)

-- Even though fluoridated water is the most consumed item in Detroit Michigan, cavities are extensive, according to Caries Research. (C)

-- Fluoridation is damaging teeth with little cavity reduction, according to a review of studies reported in Clinical Oral Investigations. (D)

-- After 50+ years of water fluoridation, Newburgh NY children have more cavities and more fluoride-caused discolored teeth (dental fluorosis) than children in never-fluoridated Kingston NY, according to a 1998 New York State Department of Health study. (E)

-- "It may...be that fluoridation of drinking water does not have a strong protective effect against early childhood caries (ECC)," reports dentist Howard Pollick, University of California, and colleagues, in the Winter 2003 Journal of Public Health Dentistry (F)

--Cavity rates declined in several cities that stopped water fluoridation, several studies report (G)

-- Despite living without fluoridated water, rural children's cavity rates equal those of urban children, who are more likely to drink fluoridated water, according to a large national government study of over 24,000 U.S. children, ages 2- to 17-year-old.(H)

-- Dental examinations of 4800 South Australian ten- to fifteen-year-olds' permanent teeth reveal unexpected results - similar cavity rates whether they drink fluoridated water or not, reports Armfield and Spencer in the August 2004 "Community Dentistry and Oral Epidemiology"(I).

-- Fluoridation is based more on unproven theories than scientific evidence, according to a revised dental textbook by leaders in the field. (J)

-- Current evidence strongly suggests that fluorides work primarily by topical means through direct action on the teeth and dental plaque. Thus ingestion of fluoride is not essential for caries (cavity) prevention," report Warren and Levy in Dental Clinics of North America, April 2003.(K)

References:

(A) Journal of Public Health Dentistry, Fall 2008, "Considerations on Optimal Fluoride Intake Using Dental Fluorosis and Dental Caries Outcomes – A Longitudinal Study," by Warren, et al.

(B) "Adding fluoride to water supplies," British Medical Journal, KK Cheng, Iain Chalmers, Trevor A. Sheldon, October 6, 2007

(C) "Dietary Patterns Related to Caries in a Low-Income Adult Population, Burt, et al., Caries Research 2006:40:473-480

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(E) Figure 1, Page 41, "Recommendations for Fluoride Use in children" NYS Dental Journal, February 1998

(F) "The Association of Early Childhood Caries and Race/Ethnicity among California Preschool Children, by Shiboski, Gansky, Ramos-Gomez, Ngo, Isman, Pollick, Journal of Public Health Dentistry, Winter 2003, pages 38-46

(G) http://groups.google.com/group/fluoridation-news-releases/browse_thre...

(H) Journal of Rural Health, Summer 2003, "Oral Health Status of Children and Adolescents by Rural Residence, United States." by Clemencia M. Vargas, DDS, PhD; Cynthia R. Ronzio, PhD; and Kathy L. Hayes, DMD, MPH

(I) Community Dentistry and Oral Epidemiology, August 2004 Consumption of nonpublic water: implications for children's caries experience, byArmfield JM, Spencer AJ.

(J) "Dentist, Dental Practice, and the Community,"
1999, by prominent researchers and dental university professors,
Burt,
Eklund, et al.

(K) Warren JJ, Levy SM. (2003). Current and future role of fluoride in nutrition. Dental Clinics of North America 47: 225-43

End of messages

« Back to Discussions « Newer topic Older topic »

Is There More Fluoride In a Pea-Sized Amount of Toothpaste or a Liter of Water?

Posted by **Tom** • 20 April 2011 • Category: **Fluoride** • **Printer-friendly**

Jake (whom I assume is a dentist) **left an interesting comment about fluoride on Sunday**. He said:

> *I had an anti-fluoride patient the other day that was saying he read somewhere that a pea-sized amount of toothpaste contains the same amount of fluoride in 1 liter of tap water (1 ppm). His argument was that the toothpaste labels says to call poison control if more than a pea-sized amount is swallowed (which it doesn't), and the same amount is in 1 liter of water. So he was wondering if he should call poison control every time he drinks more than a liter of water. It sounded ludicrous, but how much fluoride is actually in a pea-sized amount of toothpaste in comparison to 1 liter of water?*

Fluoride Warnings on Toothpaste (Click to enlarge)

I enjoy talking about water fluoridation. Looking back, I've actually written **15 different posts about fluoride**!

Jake's comment really got me wondering about how the fluoride levels compare between fluoridated water and toothpaste.

Do Toothpastes Contain a Warning Telling You to Call Poison Control?

First, let's take a look at the common anti-fluoride claim that fluoride is poison. I took a picture of the back of three different brands of toothpastes: Colgate, Aquafresh, and Crest. If you click on the picture, you can view a large size that will let you read the warning. Each tube has a similar warning. The back of the Colgate Total toothpaste box states:

If more than used for brushing is accidentally swallowed, get medical help or contact a Poison Control Center right away.

But how much do people really use for brushing? There's the ultra-conservative pea size, and then there's the large stripe that toothpaste manufacturers want us to use so that we buy lots of toothpaste!

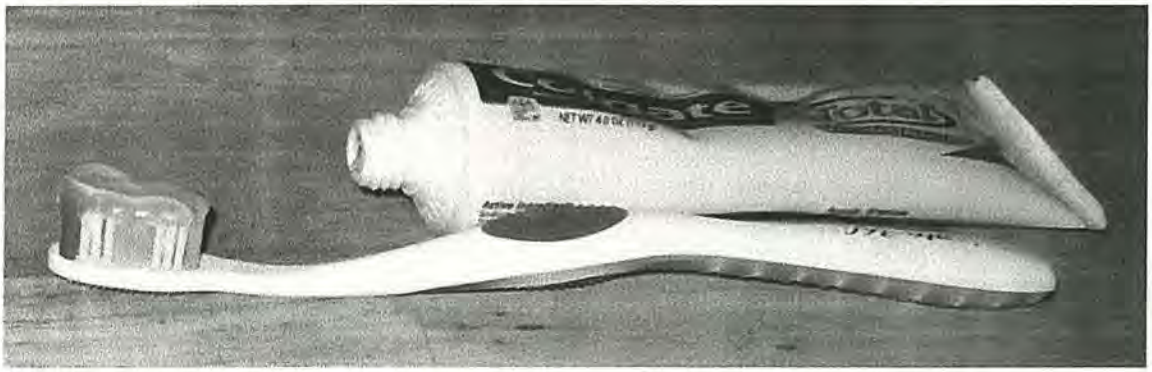
I decided to find out how much toothpaste is in a large stripe by conducting a two-part experiment.

My Toothpaste Experiment

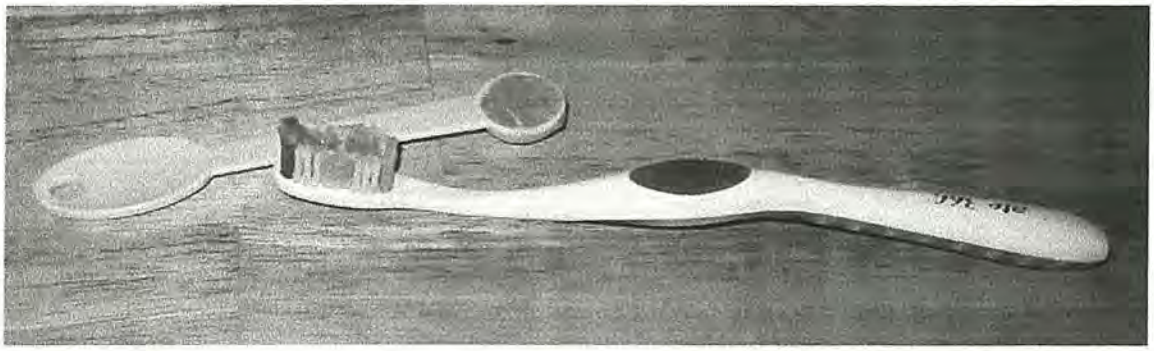
On the back of the toothpaste tube, it states that you should call the poison control center *if you swallow more than is used for brushing.* This is what the toothpaste manufacturers write. I took the liberty of assuming that a *normal* amount of toothpaste for them is a thick stripe on a manual toothbrush (like they show in their commercials).

I decided to find out exactly how much toothpaste is in a big stripe so that I could figure out how much fluoride it has. I got carried away and tried two different brands.

Here's the large stripe of Colgate Total that I put on my wife's toothbrush (are your toothbrush bristles as straight as hers? If not, it may be **time to get a new toothbrush**):



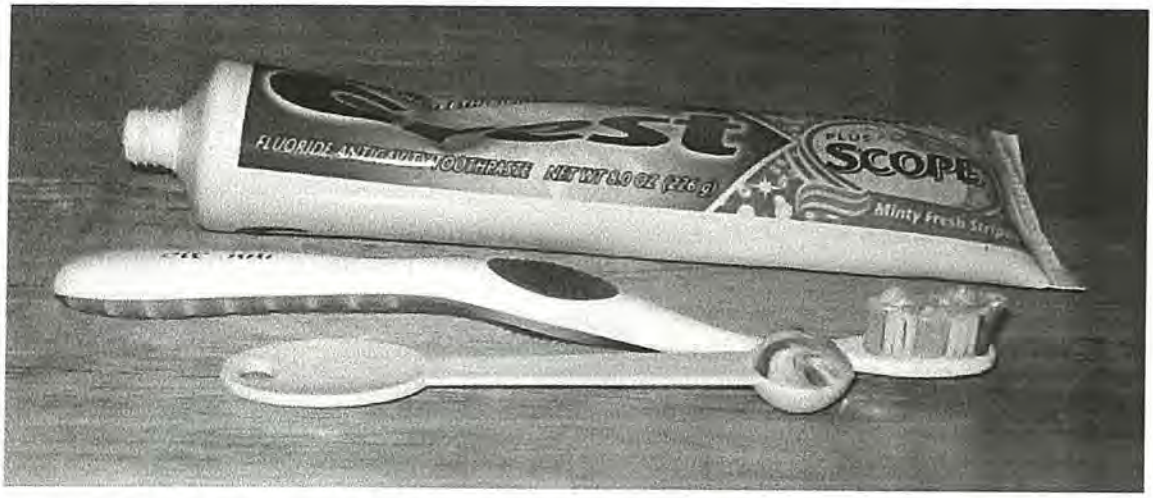
I measured the toothpaste and found that it filled the 1/4 teaspoon - giving us 1.25 ml of toothpaste:



Out of curiosity (and because it seemed like a fun idea after taking two finals over the past 36 hours), I measured the Crest Toothpaste as well. I was able to get a slightly bigger stripe on the brush this time. Unfortunately, the stripe I created just wasn't as good looking as it is on the toothpaste commercials!

However, if you want to practice making a beautiful stripe of toothpaste on your brush, I have to recommend the Crest since it is much thicker.

This large stripe of Crest ended up overflowing the 1/4 teaspoon, giving us about 1.75 ml of toothpaste:



I decided to take the average of my two "large stripes" to use as the baseline amount of toothpaste you can swallow and still be safe (according to the toothpaste manufacturers) - **which appears to be 1.5 ml from my unscientific experiment.**

Contrast this with a pea-size amount of toothpaste which is only 0.2 ml. Who would've guessed that the **average pea only takes up a volume of 0.2 ml**?

Now that we know how much toothpaste we use, we can figure out how much fluoride we would ingest if we swallowed a large stripe of toothpaste.

How Much Fluoride is in Toothpaste?

A majority of toothpastes on the market contain about 0.15% fluoride ion, which comes out to 1500 **ppm (parts per million.)**

In 1.5 ml of toothpaste (the large stripe pictured above) you would find **2.25 mg of fluoride.**

In a pea sized amount of toothpaste, you would only find **0.3 mg of fluoride.**

How Much Fluoride is in Fluoridated Water?

Most fluoridated water contains about 1.0 ppm. That means that in 1 liter of water, you would find about **1 mg of fluoride**.

Not sure how much fluoride is in your water? Then **find out how much fluoride is in your tap water!**

Comparing the Amount of Fluoride In Water with the Amount of Fluoride in Toothpaste

As you can see, you would have to drink over 2 liters of water to get the same amount of fluoride that you would get by swallowing a large stripe of toothpaste. You would only have to drink 300 ml of water (a little less than a 12 oz. can of soda) to get the same amount of fluoride you would get by swallowing a pea size amount of toothpaste.

You Don't Need to Call Poison Control When You Drink Fluoridated Water!

I'm sure Jake's patient was just trying to make a point. Point taken! However, according to the American Dental Association (**Page 31 in their Fluoridation Facts PDF**), it would take 5-10 grams of fluoride to cause fluoride toxicity in an average 155-pound man. That means that a 155-pound man would need to drink 5,000 liters of water (over 1300 gallons!) in order to get a toxic dose of fluoride.

The water would kill you (**as this tragic story illustrates**) long before the fluoride would have any toxic effect.

Conclusion

Interestingly, there is more fluoride in a liter of water than in a pea-sized amount of toothpaste, but more fluoride in a large stripe of toothpaste than in a liter of water. Here's what I found:

- In a pea size amount of toothpaste, there's 0.3 mg of fluoride.
- In a large stripe of toothpaste, there's 2.25 mg of fluoride.
- In one liter of fluoridated water, you'll find 1 mg of fluoride.

Although fluoride is great for your teeth, too much of it during development of the teeth can cause **dental fluorosis**.

Do you have any questions about toothpaste fluoride content or water fluoride content? I'd love to hear what you have to say in the comments section below. Thanks for reading!

If You Liked This Article, Chances Are That You'll Like These:

1. Find Out How Much Fluoride Is In Your Tap Water
2. Is Water Really Fluoridated With Toxic Waste Fluoride?
3. Is Water Fluoridation A Conspiracy for Businesses to Make Money Selling Fluoride?
4. How Much Fluoride Is In a PPM (Part Per Million)?
5. Toothpaste Ingredients: The 10 Main Ingredients In Toothpaste

9 Comments | Leave A Comment

1.

Jake
21 April 2011 • 3:59 AM

Excellent response! Thank you. Yes, the patient did have a point. I told him that I was aware of the dangers of excess fluoride. It looks like we have differing definitions of excess fluoride, and his doesn't line up with the definition given by the ADA. He showed me and mentioned that he gets most of his information from fluoridealert.org. So, probably some anit-fluoride dentist on that website was making that argument, and it seems credible.

You mentioned that the lethal dose of fluoride is 5-10 grams. I was curious to know what an acute toxic dose was, so I did a little research. Oddly enough, I got my information from an anti-fluoride website. They mention research that shows an acute toxic dose around 5 mg/kg. So, in your 155 lb man, he would need at least 350 mg, or 350 liters of water, or over 1000 pea-sized amounts of toothpaste. They also used a case report that happened in Alaska where several hundred individuals got sick from a malfunction that put the fluoride levels at 150 ppm. The investigation concluded that the acute toxic dose was 0.3mg/kg. Much lower than 5mg, but still...do the math. Your 155 lb man would need 21mg, or 21 liters of water, or 70 pea-sized amounts of toothpaste. If you drank 21 liters of water (5.5 gallons) you'd die of hyponatremia before you even got sick from the fluoride.

Having said all of that, you still have to be safe, especially with kids in the house. A little 1-year old who's toddling all over and stumbles upon a tube of toothpaste could easily get sick if he ingests too much. However, the anti-fluoride hype is a bit irrational and over the top in my opinion.

Reply

Tom
21 April 2011 • 12:55 PM

Hi Jake - Thanks for running all the numbers above!

It seems like the ADA and anti-fluoride sites do end up disagreeing on the level of toxicity. When I was preparing a debate on the water fluoridation issue, I turned to a more neutral source, which is the book **Fluoride In Dentistry by Fejerskov**. The authors state, "Because there are several variables that can affect the outcome of acute fluoride poisoning, it is not surprising that the fatal dose is uncertain. In cases of human poisonings the uncertainty is amplified because, in most instances, the exact doses involved is not precisely known. **Dreisbach stated** that the acute lethal dose of fluoride for humans is 6-9 mg F/kg while **the data of Lidbeck suggested** that it is over 100 mg F/kg. The most frequently cited range for the certainly lethal dose of sodium fluoride was offered by Hodge & Smith (Hodge HC, Smith FA. Biological properties of inorganic fluorides. In: Fluorine chemistry. Simons HH, ed. New York: Academic Press;1965:1-42.) After reviewing case reports, they concluded that 5-10 g of sodium fluoride would certainly be fatal for a person with a body weight of 70 kg [which makes] the dose range for adults would be 32-64 mg F/kg."

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Original article

Neurodegenerative changes in different regions of brain, spinal cord and sciatic nerve of rats treated with sodium fluoride

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Abstract

Fluoride is known to cross the blood-brain barrier and alter the structure and function of neural tissue. There are few authoritative reports on neurodegenerative changes in hippocampus, neocortex, cerebellum, spinal cord and sciatic nerve in fluoride intoxication. We report the alterations in the structure of neuronal tissue after chronic administration of sodium fluoride (for 60days) to rats. Twelve male Wistar rats were divided equally into two groups: one group received 20 ppm of sodium fluoride (NaF) and the other group (which served as a control) received tap water for 60days.

The body weights and organic somatic index of brain in the sodium fluoride treated animals were significantly reduced, relative to the control group. Tissue fluoride levels of hippocampus, neocortex, cerebellum, spinal cord and sciatic nerve, all increased significantly in fluoride treated rats. Electron microscopy of the hippocampus, neocortex, cerebellum, spinal cord and sciatic nerve showed neurodegenerative changes in the NaF treated group compared to controls. Axon deterioration, myelin sheath degeneration and dark cells with scanty cytoplasm were observed in spinal cord and sciatic nerve in the treated group. Other distinctive morphological alterations observed were: vacuolated swollen mitochondria in neocortex, hippocampus and cerebellum; myelinated fibers with breaks in continuity (axon partly preserved and partly vacuolated) in hippocampus; myelin splitting and vacuolated schwann cell within the cerebellum and sciatic nerve respectively. Thus, neurodegeneration was clearly evident in the hippocampus, neocortex, cerebellum, spinal cord and sciatic nerve on fluoride exposure.

Key words: sciatic nerve, cerebellum, sodium fluoride, hippocampus, transmission electron microscope

Fluorosis is a well-defined clinical entity characterized by the toxicity of elevated fluoride intake on teeth, bones and soft tissues[1,2]. Fluoride-exposed rat pups show mild degeneration of nerve cells[3]. Fluoride-induced morphological alterations in liver were reported with transmission

electron microscopy[4]. Cell lysis, mitochondria vacuolation, crenulations of nuclear membrane and cell shrinkage has been observed in renal cells of young pigs treated with fluoride[5]. The molecular basis of fluoride action is mainly concerned with cellular enzymes, especially antioxidant enzymes. High levels of fluoride in drinking water (1-12ppm) affect central nervous system directly without first causing the physical deformities of skeletal fluorosis[6,7]. According to Mullenix et al[8] hyperactivity and cognitive deficits can be correlated with hippocampus damage induced by sodium fluoride (NaF). Distinctive alterations in the brain have also been observed with the chronic administration of aluminium Fluoride (AlF_3) and NaF[9]. Histological changes in the brain of young, fluoride-intoxicated rats have been reported by Shivarajashankara[10]. However most of the studies have so far been confined to the whole brain. This study reports the neural changes with respect to the different regions of the brain with emphasis on hippocampus, neocortex and cerebellum of the brain, spinal cord and sciatic nerve by using transmission electron microscope (TEM), in rats administered with 20 ppm NaF for 60 days.

Materials and methods

Male Wistar rats weighing 180 ± 20gm were used in this experiment. They were housed in polycarbonated cages bedded with paddy husk; commercial pellet diet (Hindustan Lever Limited, Bangalore, India) and water were provided *ad libitum*. The animals were then divided into two groups, "control" and "fluoride" groups (n=6) respectively. The control group was given ordinary tap water, while the fluoride group received 20ppm concentration of fluoride through gavage feeding for two months. Following the treatment period the rats were euthanized and the brain (further dissected into cerebellum, neocortex and hippocampus) spinal cord and sciatic nerve were removed for TEM studies. Fluoride levels in the brain and spinal cord were determined with fluoride specific ionic electrode (Orion R 96-090).

For TEM studies, samples were transferred to vials and fixed in 2.5% glutaraldehyde in 0.5 M phosphate buffer pH 7.2 for 24 hrs at 4°C and post-fixed in 0.5% aqueous osmium tetroxide in the same buffer. After the post-fixation, the samples were dehydrated in a series of graded alcohol, infiltrated and embedded in spurs resin[11]. Both semi-thin and ultra-thin sections were cut with a glass knife on a leica ultra cut UCT-GA-D-E1-00 ultra microtome. Semi-thin sections of 200-300nm thickness were stained with toludine blue and ultra-thin section 50-70nm thickness were mounted on grids, stained with saturated aqueous uranyl acetate and counter stained with 4% lead citrate. sections were then examined at various magnifications under TEM (Hitachi, H-7500) at Ruska Laboratory, College of Veterinary Science, N.G Ranga Agricultural University, Hyderabad, India.

Results

The results revealed a significantly ($p<0.05$) higher mean value of fluoride in the neural tissue of the fluoride group compared to the control group. The mean body weight and relative organ body weight of brain was found to be relatively low in fluoride-treated group compared to control (Table I).

Neurodegenerative changes were observed in different regions of brain (neocortex, hippocampus, cerebellum), spinal cord and sciatic nerve of fluoride exposed group under different magnifications (Fig 1-16). The sciatic nerve showed normal microscopic features like oval nuclear membrane, normal electro-density and empty appearing axons in control group (Fig 1) while in fluoride group vacuolation of Schwann cells with enlarged axons and disrupted myelin sheaths were clearly observed within the sciatic nerve (Fig 2). As seen in Fig 3 normal nuclei and nucleoli were observed in the spinal cord in the control group, while the fluoride group (Fig 4, 5 & 6) showed irregular nuclei with normal nucleoli, vacuolated cytosol and axons with split myelin. In the cerebellar tissue of the control group normal nuclei and myelinated fibers with empty appearing axons, were seen (Fig 7).

Table I. The body weight, somatic index of brain and accumulation of fluoride in brain of rat after sodium fluoride treatment

Group	Fluoride levels (μg/gram tissue)	Body weight (grams)	Organ somatic index
Control	0.2452 ± 0.013	111.2 ± 2.662	2.072 ± 0.04
Fluoride	0.864 ± 0.014	92.888 ± 2.621	1.3464 ± 0.137

Values represent mean ± standard deviation. The values are significant at $p<0.05$

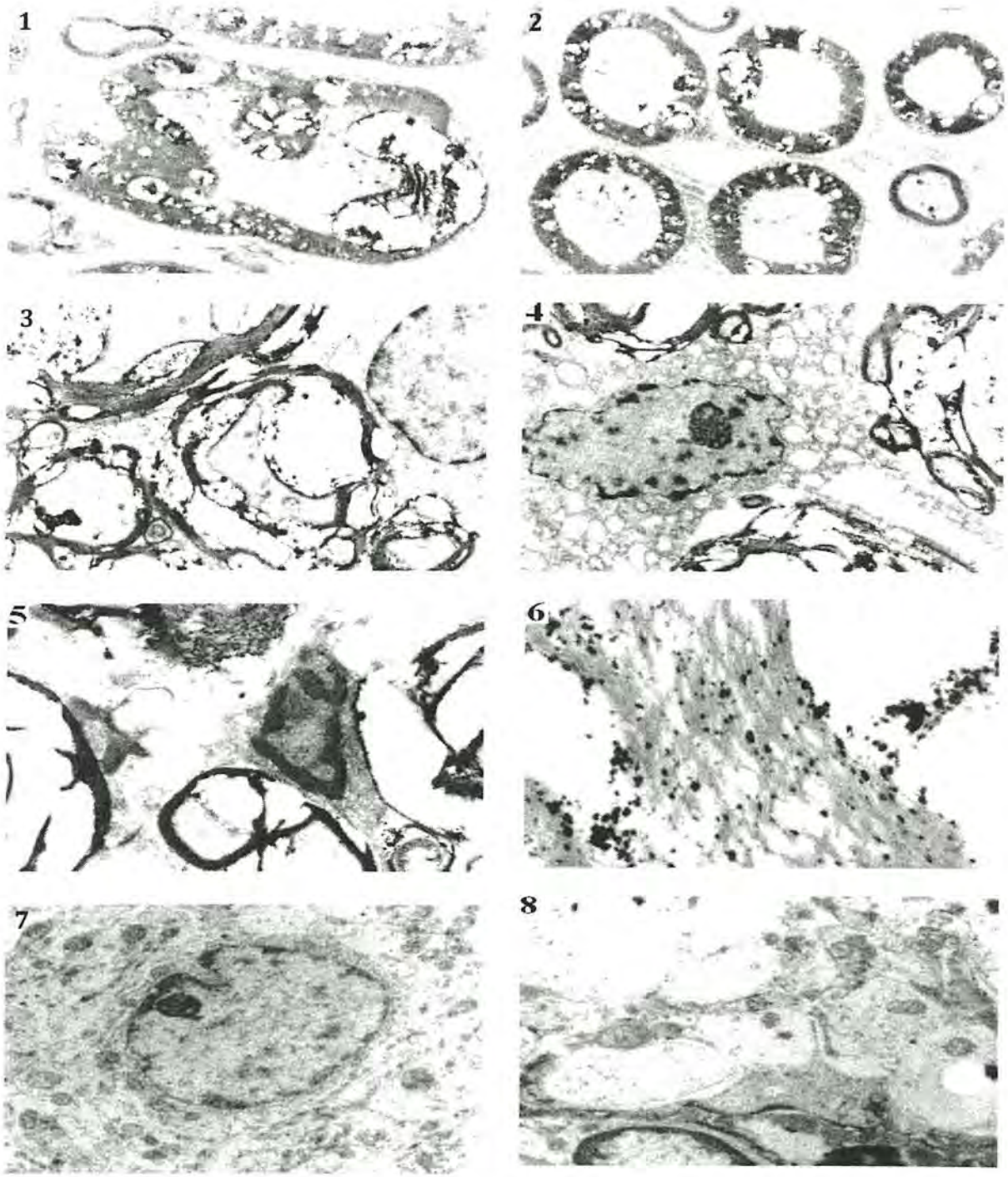

Fig 1. Sciatic nerve in control rat (magnification 3k). Electro density normal, myelin, debris cytoplasm, empty appearing axons (EA); **Fig 2.** Sciatic nerve in fluoride treated rat (magnification 3k). Vacuolation of schwann cells (VS)-cytoplasm appearing enlarged axons (CAE), disrupted myelin sheaths (DMS), fiber density normal; **Fig 3.** Spinal cord in control rat (magnification 7k). Normal dense myelin in cross section (NDM), normal nucleus with nucleolus, organelle contents normal; **Fig 4, 5 & 6.** Spinal cord in fluoride treated rat (magnification 3.5k, 4k and 3k respectively). Irregular nucleus (IN) with normal nucleolus, vacuolated cytosol, myelinated axons normal, disrupted myelin sheath (DMS); **Fig 7.** Cerebellum in control rat (magnification 3k). Normal oval mitochondria (NOM), neuropile normal, nuclei appear normal, myelinated fibers noted, empty appearing axons, organelle contents normal, nucleus normal (NN); **Fig 8 & 9.** Cerebellum in fluoride treated rat (magnification 3k). Predominantly blood vessels (PBV) appear normal, astrocytes normal, swollen mitochondria (SM), crenulated nuclear membrane (CNM), dumbbell shaped mitochondria.

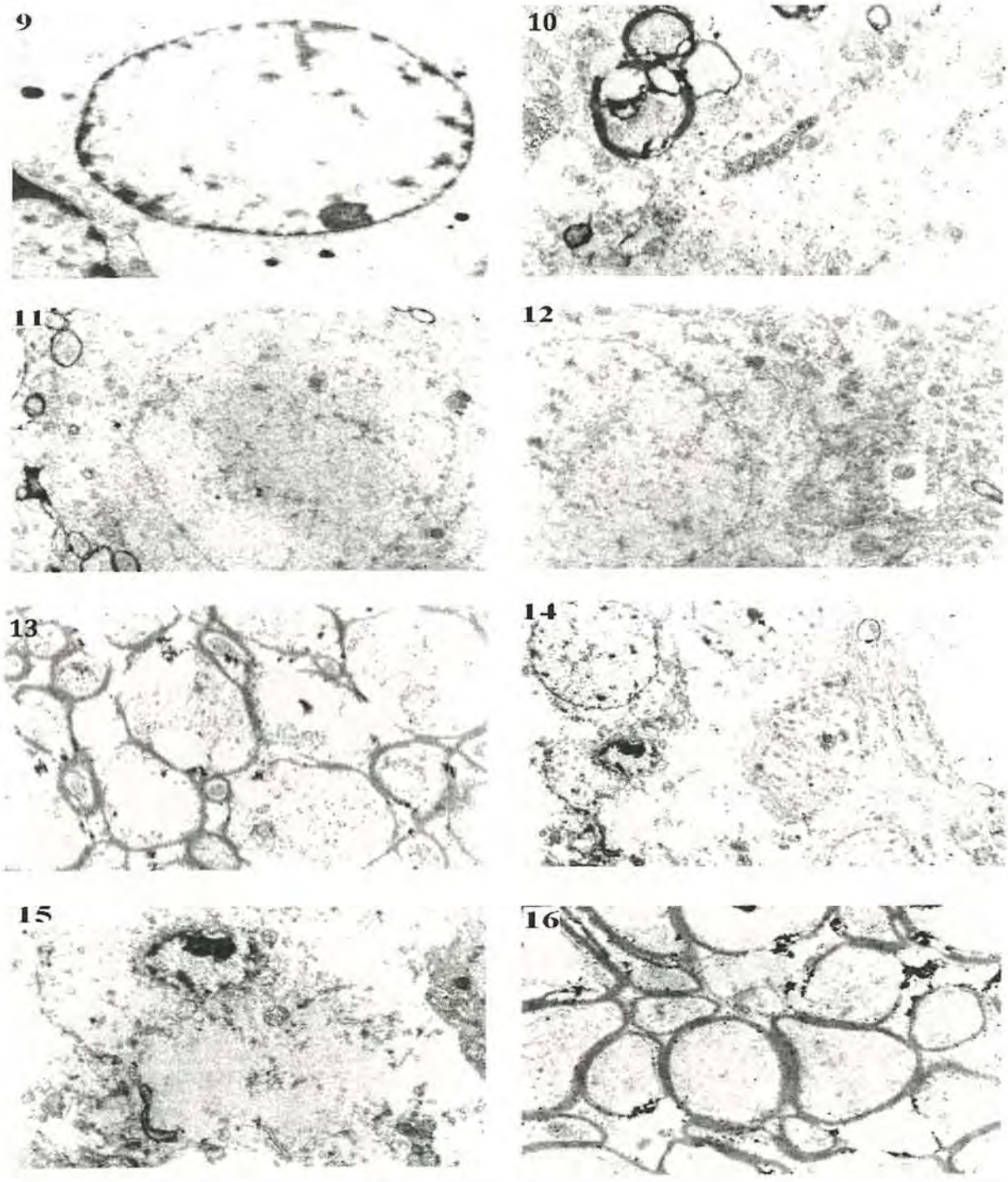

Fig 10. Neocortex in control rat (magnification 3.5k). Oligodendrocyte nucleus appears normal. Few myelinated fibers noted, few mitochondria appear normal (MA), few show vacuolated changes; **Fig 11 & 12.** Neocortex in fluoride treated rat (magnification 4k and 7k respectively). Oligodendrocyte nucleus (ON) normal, myelinated fibers shows myelin splitting (MS). Few axons shows thin myelin (TM), axon preserved, stain precipitate; **Fig 13.** Hippocampus in control rat (magnification 8k). Myelinated fibers (MF) empty axons, stain precipitates; **Fig 14, 15 & 16.** Hippocampus in fluoride treated rat (magnification 8k, 2k and 4k respectively). Disrupted myelin fibers (DMF), rough endoplasmic reticulum strands, organellae preserved, mitochondria show vacuolation. Cell loss more vascular inclusion (CMV), compressed golgi cisternae (CGC), granulated mitochondria (GM).

The cerebellar tissue of the fluoride treated rats (Fig 8 & 9) has shown predominantly normal blood vessels and normal astrocytes, however the mitochondrial morphology became dumbbell shaped and nuclear membrane crenulated. The neocortex of brain in control animals (Fig 10) showed normal oligodendrocytes with normal-appearing nuclei, axons that showed thin myelin and normal-appearing mitochondria. Significant changes in the cell pattern were observed in the neocortex of the brain on fluoride treatment; myelinated fibers with myelin splitting, vacuolated mitochondria, normal oligodendrocyte with slight indentation of nucleus and few axons with thin myelin (Fig 11 & 12). The cytoarchitecture of hippocampus of brain in the fluoride group revealed degrees of alteration in structure which included degenerated cell bodies, granulated mitochondria, vacuolation in cytosol, compressed golgi cisternae and scattered rough endoplasmic reticulum (Fig 14, 15 & 16). Thin broken myelinated fibers, axon partly preserved and partly vacuolated, in hippocampus were observed in the fluoride exposed group (Fig 13). This contrasts with the normal microscopic features like myelinated fibers, empty axons and unchanged nuclear morphology in control animals.

Discussion

In previous reports neurological changes associated with skeletal fluoride have been attributed to compression radiculomyelopathy[12]. The central and peripheral nerve damage has been ascribed to a direct toxic effect of fluoride whereas the loss of function in the motor neuron was attributed to osteoproliferation of vertebrae. Many reports have revealed that excessive fluoride treatment induces extensive damage to the nervous system[8,13].

In our earlier laboratory studies[14] we demonstrated the suppression of both antioxidant enzymes and energy-generating enzymes in female mice treated with 20 mg/kg body weight of NaF for 14 days. The Fluoride induced changes within neuronal cells included scattered and low RER, swollen mitochondria, compressed golgi cisternae in the previous studies[5,15]. It appears that fluoride in a concentration of 20 ppm extensively damages neurons in the brain, spinal cord and sciatic nerve of rats leading to paralysis and brain dysfunction. Varner et al[16] reported that the chronic administration of drinking water containing aluminium fluoride and sodium fluoride to rats resulted in distinctive morphological alteration in specific regions of the brain. In our experiment we found the vacuolation of schwann cell with enlarged and disrupted myelin sheath in the sciatic nerve of the fluoride group. Beside these changes we have also observed the significant changes like crenulated nucleus, vacuolated cytosol at different magnification in the spinal cord of fluoride group compared to control group.

Free radicals and lipid peroxidation products generated by excitotoxicity have been shown to damage dendrites and synaptic connection and, if unrelieved can lead to neuronal destruction[17]. Fluoride is known to accumulate within various parts of rat brain especially in hippocampus[13,18]. Fluoride intoxication decreases the synthesis of cholesterol, free fatty acid, proteins amino acids and RNA in the brain of rabbits[19]. The possible mechanisms for the neurodegenerative effects of fluoride are likely related to excitotoxicity by free radical and lipid peroxidation which impairs the glutamate removal and by activating microglia which contain abundant stores of glutamate[18,20,21]. It has been shown that one of the lipid peroxidation products, 4-hydeoxynonenal (4-HNE), specifically impairs synaptic functions and inhibits glutamate removal by the glutamate transport protein[22]. It was also observed that NaF increased nitric oxide synthase activity plays a major role in all neurodegenerative diseases, primarily by damaging mitochondrial energy production, Inhibiting glutamate reuptake and stimulating lipid peroxidation[23,24,25].

In conclusion, electron microscopic observations comprehensively defined the structural alteration in the specific regions of the brain like the neocortex, hippocampus, cerebellum, spinal cord and sciatic nerve, secondary to fluoride exposure in rats. Further studies are required to unravel the molecular and cellular mechanisms responsible for neurodegenerative changes in cytoarchitecture of the specific structures of the nervous system. This is the first report on ultrastructural changes in the neuronal cells in fluoride-treated rats using TEM.

Conflict of interest: None

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HEALTH RELATED RESEARCH STUDIES

Excerpts from:

Fluoride: The Aging Factor (2nd Edition)
Author: Dr. John Yiamouyiannis
Published: Health Action Press
Year: 1986 (first edition 1983, third edition 1993)
ISBN: 0-913571-01-6

About the Author: Dr. John Yiamouyiannis was, until his death in the fall of 2000, the world's leading authority on the biological effects of fluoride. His formal education included a B.S. in biochemistry from the University of Chicago and a Ph.D in biochemistry from the University of Rhode Island. After a year of postdoctoral research at Western Reserve University Medical School, Yiamouyiannis went on to become biochemical editor at Chemical Abstracts Service, the world's largest chemical information center. It was at Chemical Abstracts Service, where Yiamouyiannis became interested in the damaging effects of fluoride.

Chapter 4 - Breaking Down the Body's Glue

All animals, including humans, are made up of cells. The cell, the basic unit of life, can be identified under a microscope by its outer membrane and a nucleus within the membrane.

Some cells are able to produce a protein called collagen. In this book, the term "collagen" refers to collagen as well as collagen-like proteins. This process occurs inside the cell. Little globules called vesicles carry the collagen from the inside of the cell to the cell membrane where it is released to the outside of the cell. There, the collagen thickens into fibers.

The five different types of cells capable of producing and releasing collagen in this way are:

- fibroblasts, which produce collagen for the structural support of skin, tendons, ligaments and muscle;
- chondroblasts, which produce collagen for the structual support of cartilage;
- osteoblasts, which produce collagen for the structual foundation and framework upon which calcium and phosphate are deposited, giving rise to bone;
- ameloblasts, which produce collagen for the structural foundation and framework upon which calcium and phosphate are deposited, giving rise to tooth enamel.
- odontoblasts, which produce collagen for the structual foundation and framework upon which calcium and phosphate are deposited, giving rise to the inner part of the tooth. This material is called dentin.

Like other proteins, collagen is composed of amino acids linked together in a chain. However, collagen contains two additional amino acids, hydroxyproline and hydroxylysine, not found in other proteins. Thus when collagen breaks down, the hydroxyproline and hydroxylysine levels in the blood and urine increase.

Researchers from Harvard University and the National Institutes of Health knew in the 1960s that fluoride disrupted collagen synthesis. It was not until 1979-1981, however, that a new flurry of

research activity in this area began.

In 1981, Dr. Kakuya Ishida of the Kanagawa Dental University in Japan reported the results of studies in which he fed laboratory animals 1 part per million fluoride in their drinking water and analyzed the urine for hydroxyproline. He found that urinary hydroxyproline levels increased in those animals. This indicates that as little as 1 part per million fluoride interferes with collagen metabolism and leads to its breakdown.

Dr. Marian Drozdz and co-workers from the Institute of Bioanalytical and Environmental Studies in Katowice, Poland found increased hydroxyproline and hydroxylysine levels in the blood and urine as well as a decrease in skin and lung collagen levels in rats fed 1 part per million fluoride in their drinking water.

Dr. Anna Put and co-workers from the Department of Pharmacology of the Pomorska Akademy of Medicine in Szczecin, Poland also found that fluoride increased hydroxyproline levels in urine.

Drs. A.K. Susheela, Y.D. Sharma and co-workers from the All-India Institute of Medical Sciences found that fluoride exposure disrupts the synthesis of collagen and leads to the breakdown of collagen in bone, tendon, muscle, skin, cartilage, lung, kidney, and trachea.

As already noted, small vesicles transport collagen from the inside of the cell to the outside of the cell. Drs. Harold Fleming and Val Greenfield of Yale University School of Medicine found a larger number of these vesicles in collagen forming cells (ameloblasts) in animals exposed to fluoride. This work was recently confirmed by S. Chen and D. Eisenmann of the University of Illinois, who also found a fluoride-induced increase of these granules in ameloblasts.

It appears that fluoride disruption of collagen synthesis in cells responsible for laying down collagen leads these cells to try to compensate for their inability to put out intact collagen by producing larger quantities of imperfect collagen and/or noncollagenous protein.

In 1983, Dr. John R. Farley and co-workers from Loma Linda University showed that treatment of bone cells with less than 1 part per million fluoride increased collagen formation by 50 percent. One year later, Dr. J.R. Smid and co-workers from the Department of Oral Biology at the University of Queensland in Australia found that fluoride ingestion led to an increase of noncollagen proteins as well as collagen proteins.

This is supported by the works of Drs. J.H. Bowes and M.M. Murray, Dr. Kh.A. Abishev and co-workers, and Dr. B.R. Bhussry who report a vastly higher protein content in teeth and bone damaged by fluoride. Clinical findings also show that new irregular bone growth is stimulated by fluoride.

The drawings below illustrate the effect of fluoride on collagen metabolism.

While collagen is made by many different types of cells and, under normal circumstances, is only mineralized in teeth and bones, the body obviously has some mechanism to mineralize the collagen of some tissues while leaving the collagen of other tissues, such as skin, ligaments, tendons, etc., unmineralized.

During the aging process, the body loses its ability to discriminate between which tissues should be mineralized and which tissues should not. As will be shown, consumption of fluoride results in the same loss of the body's ability to discriminate. In other words, mineralization of tissue, such as bone, which should be mineralized, is disrupted, and tendons, ligaments, muscles, and other soft tissue which should not be mineralized start to become mineralized as a result of fluoride exposure.

By interfering with collagen production, fluoride leads to the production of larger quantites of imperfect collagen and/or other types of protein and thus interferes with the body's normal regulation of collagen mineralization.

The type and array of collagen and collagen-related proteins made by the various collagen-producing cells determine whether or not the collagen framework will be mineralized. During the aging process, cumulative damage to these cells leads to the diseases attributed to "old age" - arthritis, arteriosclerosis, brittle bones, wrinkled skin, etc. Consumption of fluoride produces the same effects and results in the same diseases.

Fluoride probably acts by interfering with enzymes essential for setting up the proper conditions for producing intact collagen. Thus, as has already been indicated, larger amounts of imperfect or deformed collagen fibers are formed and the body's ability to regulate collagen formation and mineralization is hindered...

Chapter 6 - Aging the Bone: The Degenerative Effects of Skeletal Fluorosis

Now let's look at the bone. Unlike the ameloblasts, and odontoblasts of teeth whose regenerative activity stops after tooth development, osteoblasts continue to actively lay down collagen, and new bone formation continues to take place.

If a tooth breaks or fractures, you're out of luck. The damage cannot be repaired. However, if a bone breaks or fractures, osteoblasts lay down collagen to produce a framework for new bone formation to repair the damage.

Bone also has the ability to rejuvenate itself. As older bone is removed by bone scavenger cells called osteoclasts, osteoblasts lay down collagen to produce a framework for new bone formation to renew the skeletal structure.

Thus, damage to collagen production in bone can interfere with the normal processes of bone rejuvenation and repair throughout life.

Cartilage

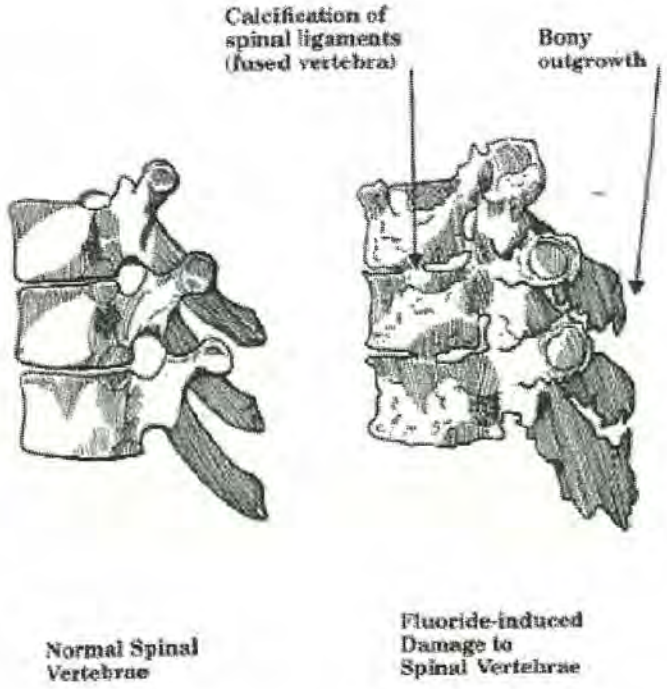
The balls and sockets of bones are lined with a smooth, tough elastic substance called cartilage. Maintaining the integrity of cartilage depends largely upon the ability of cells called chondroblasts to lay down noncalcified collagen which is the major structural component of cartilage.

The Effect of Fluoride on Bone and Cartilage

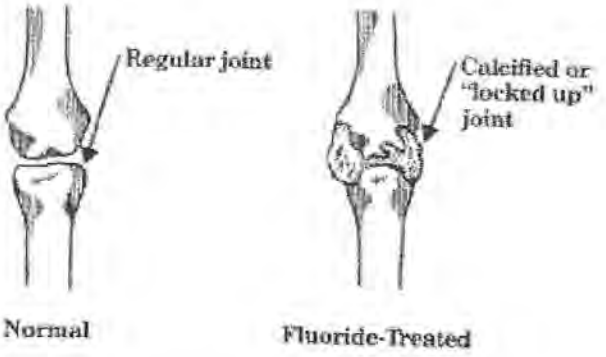
Fluoride has been shown to interfere with collagen formation in osteoblasts and chondroblasts. If, as pointed out, increased production of imperfect collagen or collagen-like protein results in mineralization of tissues which should not be mineralized, and vice versa, one would expect a calcification of ligaments, cartilage, and tendons as well as the formation of poorly and overly mineralized bone. This is exactly what happens after exposure to fluoride.

In discussing their examination of tissues from patients exposed to fluoride, Drs. A. Singh and S.S. Jolly, world-renowned experts on the clinical effects of fluoride on bone, point out that:

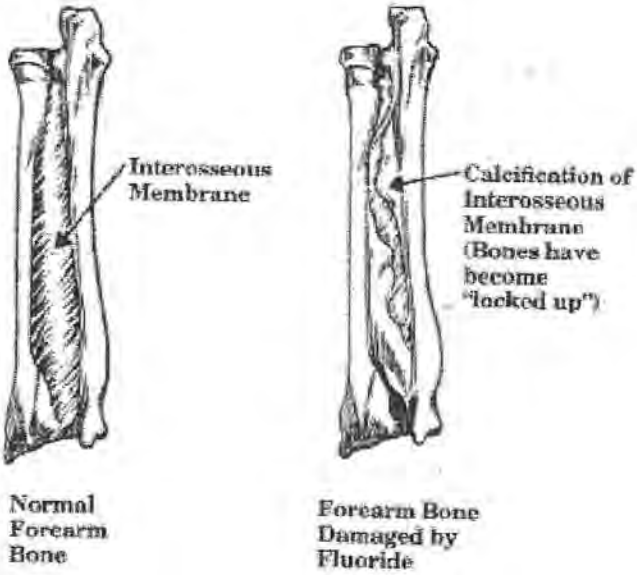
- The most noticeable changes are detected in the spine with calcification of various spinal ligaments, resulting in pronounced bony outgrowths. The other bones show numerous spiky outgrowths especially in tendons (collagen-rich fibrous tissue which attach muscles to bone) and ligaments (collagen rich fibrous tissue which holds bones together). Under careful inspection, the bony outgrowths are found to consist of coarse, woven fibers which are largely uncalcified.



- Irregular bone is also laid down in joint sockets...



and interosseous membranes (membranes between bones in arms and legs).



- In more advanced cases of fluoride exposure, bones become held together by masses of new bone laid down in the joint socket, ligaments and tendons. This results in the locking up of joints and permanent inability of victims to move or flex their joints. Vertebrae become fused at many places. This results in the characteristic "hunch back" symptom of skeletal fluorosis.

- There is a low degree of remineralization of the bone itself, which is partly due to a wide seam of uncalcified osteoid (collagen).

In 1973, Dr. Jolly and co-workers presented radiological evidence of skeletal fluorosis which results in these bone deformities in parts of India where the drinking water contained as little as 0.7 parts per million fluoride, with the occurrence and severity increasing with increasing levels of fluoride in the drinking water.

RADIOLOGICAL EVIDENCE OF SKELETAL FLUOROSIS IN MALES 21 YEARS OF AGE AND OLDER

Village	Water Fluoride (ppm)	Percentage
Mandi Baretta	.7	2.8
Kooriwara	2.3	40.0
Gurnay Kalan	2.4	19.6
Ganza Dhanaula	4.2	26.3
Bajakhana	5.1	46.9
Rajia	5.2	52.2
Village Baretta	5.5	29.6
Rorki	7.0	52.5
Saideke	8.2	52.6
Khara	9.4	80.1

In 1985, Dr. I. Arnala and co-workers of Kuopio University in Finland reported that: *"The upper limit for fluoride concentration in drinking water that does not increase the amount of unmineralized bone is roughly 1.5 parts per million. ...We should however, recognize that it is difficult to give a strict value for a safe concentration in drinking water because individual susceptibility to fluoride varies."*

In addition to fluoride-induced bone irregularities, one could expect that the fluoride-induced irregularities of the joint cartilage (which is normally smooth) would result in the irritation and inflammation commonly referred to as arthritis. One could also expect fluoride to cause an increase in the incidence of fractures and a decrease in the body's ability to heal bone breaks and bone fractures.

Clinical observations show that this is exactly what happens.

Arthritic Changes

Drs. Singh and Jolly point out that early symptoms of fluoride-induced damage to bones and cartilage start with *"vague pains noted most frequently in the small joints of the spine. These cases are frequent in the endemic* [local] *areas and may be misdiagnosed as rheumatoid or osteoarthritis.*

"In later stages, there is an obvious stiffness of the spine with limitation of movements, and still later, the development of kyphosis [hunch back].

"There is difficulty in walking, due partly to stiffness and limitation of the movements of various joints....

"Some patients complain of dyspnea, [difficulty in breathing] *on exertion because of the rigidity of the thoracic cage."*

Dr. Jolly and co-workers reported these symptoms in parts of India where the drinking water contains as little as 0.7 parts per million fluoride, the occurrence and severity increasing as the

fluoride content in the drinking water increased.

In the United States, Dr. George Waldbott also diagnosed some of the early symptoms listed above, including arthritis and joint pains, as being due to the consumption of water fluoridated at 1 part per million. He was able to bring about a reversal in these symptoms by eliminating fluoridated water from the patients'diets. However, if left unattended, the degeneration leads to the advanced stages of arthritis and "old age."

Similar arthritic symptoms have been reported among people exposed to air-borne fluoride in Switzerland, Germany, Britain, United States, Canada, and North Africa. Dr. Yiamouyiannis was contacted by an independent British broadcasting company who consulted him concerning a problem they had found in a brick manufacturing area about 50 miles outside of London where they reported that over 90% of the population was suffering from arthritis induced by air-borne fluoride.

Dr.Waldbott noted the possibility of the age-accelerating effects of fluoride with respect to arthritis and stated:

"Among the elderly, arthritis of the spine is an especially common ailment that is customarily attributed to 'aging.' Since fluoride retention in bones increases as a person grows older, how can we disregard the possibility that this 'old age' disease might be linked with fluoride intake? For example ... [others have] described in detail X-ray changes encountered in skeletal fluorosis in North Africa, that are in every respect identical to those present in the arthritic spine of the elderly."

Breaks and Fractures

In 1978, Dr. J.A. Albright and co-workers from Yale University reported at the Annual Meeting of the Orthopedics Research Society that as little as 1 part per million fluoride decreases bone strength and elasticity.

In 1983, Dr. B. Uslu from Anadelu University School of Medicine in Eskisehir, Turkey reported that addition of fluoride to the drinking water of rats with fractured bones resulted in defective healing of the fracture due to disruption of collagen synthesis.

In 1978, the ***Journal of the American Medical Association*** published an editorial pointing out that *"in several short-term studies, fluoride has been administered for treatment of involutional osteoporosis, alone or with supplemental calcium, vitamin D or both. No studies have demonstrated alleviation of fracture[s]. ... However, studies in humans have shown an increased incidence of... fractures. When high doses of fluorides have been given to animals receiving a diet that was otherwise unchanged, most studies have shown no change or a decrease in the strength of the bone."* They also pointed out that administration of fluoride resulted in nonmineralized seams in bones, resulting in the disease called osteomalacia. These nonmineralized seams imply that breaks and fractures in the patients' bones would tend to heal more slowly.

It is ironic that anyone would ever think of treating osteoporosis (a disease in which the bones lose calcium) with fluoride, a substance which leads to decalcification of bone. In 1977, Dr. Jennifer Jowsey, one of the originators of fluoride therapy for osteoporosis, admitted that fluoride was

leading to a greater degree of osteoporosis (demineralization) in some bones while leading to osteosclerosis (overmineralization) in others. In other words, fluoride treatment of osteoporosis "robs Peter to pay Paul" and leads to a general weakening of the bones.

In 1980, Dr. J.C. Robin and co-workers from the Roswell Park Memorial Institute confirmed the foolishness of using fluoride for the treatment of osteoporosis by publishing their results in the ***Journal of Medicine***. According to the authors, *"fluoride had no preventive effect. In some experiments there was even a deleterious effect of fluoride."* They found fluoride accelerated the process of osteoporosis leading to a loss of calcium from the bone.

Claims that the amount of fluoride found in fluoridated water would help prevent osteoporosis have been studied epidemiologically. Researchers from the U.S. National Center for Health Statistics claimed to find no preventive effect, while researchers from the National Board of Health in Finland claim to find a preventive effect. However, the number of people examined in these two studies was far too small to yield statistically meaningful results. The studies of Drs. Singh and Jolly as well as the studies of Dr. I. Arnala and co-workers who report increases in unmineralized bone, are consistent with the finding of Dr. Robin that fluoride accelerates the process of osteoporosis.

In 1973, a report from the National Institute of Arthritis and Metabolic Diseases found 50 to 100% increases in the incidence of a disease called osteitis fibrosa among patients whose artificial kidney machines were run on fluoridated water. Osteitis fibrosa is a disease characterized by fibrous degeneration of the bone; it results in bone deformities and sometimes in fracture...

Hardening of the Arteries

In a number of areas where people consume water containing 3 parts per million fluoride or more, calcification of the arteries has been clinically correlated with the fluoride-induced bone disorders described in Chapter 6. The indication again is that fibroblasts in the arterial cell walls are producing larger amounts of an imperfect collagen or collagen-like protein, resulting in hardening of the arteries or arteriosclerosis, the leading cause of death in the United States.

During aging, hardening of the arteries is probably due to disruption of collagen production, according to Dr. John Negalesko, director of the first year medical program at the Ohio State University Medical School and an expert in the field. Thus, fluoride, by disrupting the production of collagen and by stimulating the calcification of arteries, has speeded up another phase of the aging process...

Chapter 8: **Fluoride & Genetic Damage**

As pointed out in Chapter 4, all animals, including humans, are made up of cells. Each cell contains a nucleus, which is separated from the remainder of the cell by a nuclear membrane. Within the nucleus exist chromosomes, which contain DNA and protein. DNA is the body's master blueprint material. It is the genetic material that determines how the body is built. DNA specifies traits such as height, hair texture and color, number of fingers on each hand, blood type, and by means of its control of protein and enzyme synthesis, the susceptibility of the individual to

various diseases.

Since maintaining the integrity of this master blueprint is so vital, the cell makes a "photocopy" of the DNA called RNA, so that the risk of damaging the DNA is minimized. This photocopy blueprint is taken to "construction sites" in the cell. These construction sites are called ribosomes. On these ribosomes, the RNA blueprint is used to direct the manufacture of proteins and enzymes, which, in turn, directly determine the structure, traits, and limiting capabilities of the body.

To further insure the integrity of DNA, the cell provides a group of enzymes called the DNA repair enzyme system which repairs DNA when damage is done to it. As people age, their DNA repair enzyme system slows down. This results in DNA damage which goes unrepaired and leads to cell damage or death. Damaged or dead cells may then put out products which in turn damage other cells, leading eventually to massive cell death and the degenerative loss of various tissues and organs in a snowballing cycle of aging > damage > aging ....

Serious consequences can also arise if the unrepaired DNA damage occurs in a cell which gives rise to a sperm or egg cell. In these cases, DNA damage in the defective egg or sperm cell will be replicated in every cell of the offspring's body and will lead to a birth defect. If the child with this birth defect survives to maturity and reproduces, this genetic deformity will be passed on from generation to generation. A decline in DNA repair activity with "age" is one of the reasons why the number of birth defects increases as maternal age increases.

Unrepaired damage of a segment of the DNA responsible for control of cell growth (brought about by a deficient DNA repair enzyme system) can lead to uncontrolled cell growth or tumors. Many tumors stop growing when they are contained by the cells around them. However, in some cases, tumor cells may release an enzyme, or may be induced by additional genetic damage to release an enzyme, which digests the surrounding cells. The result is an invasive or malignant tumor and is more commonly referred to as cancer.

An excellent example of a defective DNA repair enzyme system leading to cancer is provided by victims of a disease called xeroderma pigmentosum. These people suffer from an inherited deficiency of DNA repair enzyme activity and are known to succumb to cancer early in life as a result.

A decline in DNA repair activity with "age" is one of the primary reasons why the incidence of cancer among older people is so much higher than the cancer incidence among younger people. The defective DNA repair enzyme in patients with xeroderma pigmentosum accelerates the aging process to the extent that xeroderma pigmentosum patients in their 20's have the same cancer risk as "normal" people in their 80's.

Dr. Wolfgang Klein and co-workers at the Seibersdorf Research Center in Austria reported that 1 part per million fluoride inhibits DNA repair enzyme activity by 50%. Since fluoride inhibits DNA repair enzyme activity, fluoride should also be expected to lead to an increase in genetic or chromosome damage.

This has indeed been found to occur in numerous studies showing that fluoride in water, even at the concentration of 1 part per million, can cause chromosome damage.

The following table outlines the results of laboratory studies regarding the effect of fluoride on genetic damage in mammals.

Year	Institution	Animal	Findings
1973	Russian Research Institute of Industrial Health & Occupational Diseases (USSR)	rat	fluoride causes genetic damage
1974	Columbia University College of Physicians & Surgeons (USA)	mouse/sheep/cow	fluoride causes genetic damage
1978	Pomeranian Medical Academy (Poland)	human WBCs	fluoride causes genetic damage
1979	National Institute of Dental Research (USA)*	mouse	fluoride does not cause genetic damage*
1981	Institute of Botany, Baku (USSR)	rat 3 studies	fluoride causes genetic damage
1982	University of Missouri, Kansas City (USA)	mouse	fluoride causes genetic damage
1983	Kunming Institute of Zoology, Kunming (Peop. Rep. China)	deer	fluoride causes genetic damage
1983	Kunming Institute of Zoology, Kunming (Peop. Rep. China)	human WBCs	fluoride causes genetic damage
1984	Nippon Dental University, Tokyo (Japan)	hamster embryo cell	fluoride causes genetic damage
1984	Nippon Dental University, Tokyo (Japan)	human cell culture	fluoride causes genetic damage
1985	Medical Research Council, Edinburgh (UK)	human WBCs	fluoride causes genetic damage

**A prepublication copy of this paper was submitted as an exhibit in a court case in Pittsburgh (USA). During trial, it was brought out that the results showed that increasing fluoride contents in drinking water increased genetic damage in mouse testes cells. Before the paper was published these figures were altered so as to destroy the original figures showing a relation between fluoride and genetic damage (see Chapter 16).*

One of the most relevant of these studies are those of Dr. Aly Mohamed, a geneticist at the University of Missouri. They show that one part per million fluoride in the drinking water of mice causes chromosomal damage. These studies also show that as the fluoride content of the water increases the degree of chromosomal damage increases in both testes and bone marrow. The results are presented in the following table:

1973	Texas A&M University (USA)	Barley (2)	fluoride causes genetic damage
1982	Institute of Botany, Baku (USSR)	Onion	fluoride causes genetic damage
1983	Institute of Botany, Baku (USSR)	Onion	fluoride causes genetic damage

Drs. R.N. Mukherjee and F.H. Sobels from the University of Leiden in Holland found that fluoride increased the frequency of genetic damage in sperm cells which were produced by laboratory animals exposed to X-rays. It is evident, from their studies, that fluoride inhibited the repair of DNA damaged X-rays. The authors themselves concluded: *"sodium fluoride resulted in a consistent and highly significant increase of the mutation* [i.e. genetic damage] *frequency. This effect is thought to result from interference with a repair process."*

In agreement with Drs. Mukheijee and Sobels were Dr. S.I. Voroshilin and co-workers from the Russian Research Institute of Industrial Health and Occupational Diseases. From their studies they concluded: *"It would seem to us that fluoride could cause some kind of disturbance in the enzymes that are related to the mechanisms of DNA repair and synthesis."*

In 1981, Dr. A. Iarez and co-workers from the Department of Toxicology from Central University ofVenezuela in Caracas, reported that fluoride added to the drinking water of female rats produced birth defects in their offspring. Just one year later Drs. Rhuitao Zhang and Shunguang Zhang of the Changjian Institute of Marine Products found that fluoride caused birth defects in fish.

According to the June 16, 1976 issue of the San Diego Union, an experiment showed that 10% of the litters of female mice drinking tap water from Durham, North Carolina (fluoridated in 1962) contained at least one malformed baby. No birth defects were observed in mice drinking purified water. While this study in itself does not prove that fluoride was the cause, the effects of fluoride as determined by the investigators mentioned above certainly make fluoride a prime suspect.

Fluoride-Induced Cancer

The ability of fluoride to cause genetic damage is so well recognized that investigators are now trying to find ways to counteract its genetic damaging effects.

Substances like fluoride which cause genetic damage are called mutagenic substances and it is a well-accepted fact that substances which are mutagenic also tend to be carcinogenic, or cancer producing. In fact, this is exactly what has been found with regard to fluoride.

Dr. Takeki Tsutsui and co-workers of the Nippon Dental College in Japan showed that fluoride not only caused genetic damage but was also capable of transforming normal cells into cancer cells. The levels of fluoride used in this study were the same levels of fluoride that the U.S. National Cancer Institute suggested should be used to determine whether or not fluoridation of public water supplies causes cancer.

They found that cells treated with 34 and 45 parts per million fluoride produced cancer (fibrosarcoma) when injected under the skin of otherwise healthy adult hamsters. In contrast, they

CHROMOSOME DAMAGE CAUSED BY FLUORIDE

Fluoride (parts per million)	PERCENT OF CELLS WITH CHROMOSOMAL DAMAGE			
	Bone Marrow		Testes	
	3 weeks	6 weeks	3 weeks	6 weeks
0	18.4	19.3	16.0	15.8
1	25.7	32.1	21.4	21.1
5	29.9	41.3	23.2	22.8
10	35.5	46.0	30.5	29.7
50	44.6	47.1	34.3	41.3
100	47.5	47.9	40.3	48.2
200	45.6	49.2	42.5	50.3

(Click to enlarge table)

Chromosomes (and thus any chromosomal abnormalities that may occur) are only visible while the cell is dividing. Therefore, Dr. Mohamed studied bone marrow and testes cells since these cells divide rapidly.

Since the testes cells observed by Dr. Mohamed give rise to sperm cells which are passed on to future generations, genetic damage to these testes cells can lead to birth defects and other metabolic disorders which can be passed on from generation to generation.

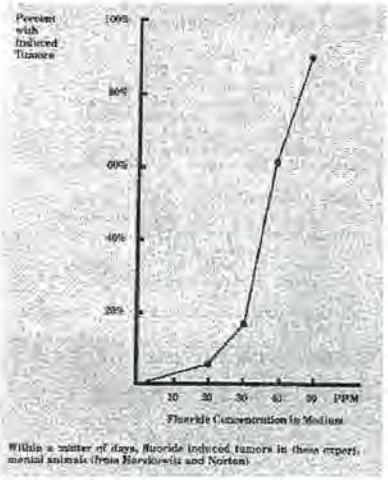
Early studies regarding the ability of fluoride to cause chromosome damage were done on plants and insects and as a result drew little attention. However, since the basic structure, function, and repair of chromosomes is similar in plants, insects, and animals, substances like fluoride which cause genetic damage in plants and insects, will most likely cause genetic damage in animals-including man.

The following table outlines the results of laboratory studies regarding the effect of fluoride on genetic damage in plants and insects.

Year	Institution	Plant or Insect Used	Findings
1966	Texas A&M University (USA)	Onion	fluoride causes genetic damage
1966	Texas A&M University (USA)	Tomato	fluoride causes genetic damage
1968	University of Missouri, Kansas City (USA)	Tomato	fluoride causes genetic damage
1970	University of Missouri, Kansas City (USA)	Maize	fluoride causes genetic damage
1970	University of Missouri, Kansas City (USA)	Fruit Fly	fluoride causes genetic damage
1971	Texas A&M University (USA)	Fruit Fly	fluoride causes genetic damage
1973	Texas A&M University (USA)	Fruit Fly	fluoride causes genetic damage
1973	Central Laboratory for Mutagen Testing (W. Germany)	Fruit Fly	fluoride causes genetic damage

found that cells that were not treated with fluoride did not produce cancer.

This confirms the earlier U.S. National Cancer Institute sponsored studies done by Drs. Irwin Herskowitz and Isabel Norton. In 1963, these St. Louis University scientists showed that low levels of fluoride increased the incidence of melanotic tumors in fruit flies by 12 to 100% (see the following figure).



Within a matter of days, fluoride induced tumors in these experimental animals (from Herskowitz and Norton)

(Click here to enlarge)

Similar types of transformations of normal cells to potentially cancerous cells have been observed in humans.

Dr. Danuta Jachimczak and co-workers from the Pomeranian Medical Academy in Poland reported that as little as 0.6 part per million fluoride produces chromosomal damage in human white blood cells. This study has received support from two other studies by Dr. R. Lin and co-workers from the Kumming Institute of Zoology and Dr. E.J. Thomson and co-workers from the Medical Research Council in Edinburgh, Scotland, who showed a 2-fold to 15-fold increase in chromosomal aberration rates at levels of 1.5 to 60 parts per million fluoride. The Thomson study suffers from the fact that the investigators administered another mutagenic substance to all the cells tested to measure other indexes of chromosomal activity.

Dr. Stephen Greenberg from the Chicago Medical School observed a disturbance of the DNA in white blood cells of animals treated with 5-10 ppm fluoride and observed other changes which he maintained were characteristic of cancer cells. In humans, Dr. Paul H. Duffey and co-workers from the Tucson Medical Center also found that fluoride transforms certain white blood cells into cells which appeared to be cancerous.

It is quite clear that fluoride causes genetic damage. The mechanism of action of fluoride cannot be exactly pinpointed because fluoride interferes with a number of physiological processes. Most evidence indicates that fluoride acts on the DNA repair enzyme system. This does not rule out the possibility that fluoride also interferes with DNA synthesis or that it may even act directly on the DNA itself. DNA is composed of two molecular strands held together by hydrogen bonds and fluoride is capable of disrupting these bonds. Such disruption would be expected to result in genetic damage directly and/or interference with DNA synthesis and DNA repair.

Furthermore, fluoride-induced genetic damage may also result from the general metabolic imbalance caused by fluoride selectively inhibiting certain enzymes.

The fact that fluoride has also been shown to cause cancer should not be surprising since it is almost universally accepted that cancer results from genetic damage.

In any event, the fact that fluoride disrupts DNA repair enzyme activity, the fact that fluoride causes genetic damage, and the fact that fluoride causes cancer shows again that fluoride is directly accelerating the aging process.

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ORIGINAL PAPER

Age-specific fluoride exposure in drinking water and osteosarcoma (United States)

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Abstract

Objective We explored age-specific and gender-specific effects of fluoride level in drinking water and the incidence of osteosarcoma.

Methods We used data from a matched case–control study conducted through 11 hospitals in the United States that included a complete residential history for each patient and type of drinking water (public, private well, bottled) used at each address. Our analysis was limited to cases less than 20 years old. We standardized fluoride exposure estimates based on CDC-recommended target levels that take climate into account. We categorized exposure into three groups (<30%, 30–99%, >99% of target) and used conditional logistic regression to estimate odds ratios.

Results Analysis is based on 103 cases under the age of 20 and 215 matched controls. For males, the unadjusted odds ratios for higher exposures were greater than 1.0 at each exposure age, reaching a peak of 4.07 (95% CI 1.43, 11.56) at age 7 years for the highest exposure. Adjusting for potential confounders produced similar results with an adjusted odds ratio for males of 5.46 (95% CI 1.50, 19.90) at age 7 years. This association was not apparent among females.

Conclusions Our exploratory analysis found an association between fluoride exposure in drinking water during childhood and the incidence of osteosarcoma among males but not consistently among females. Further research is required to confirm or refute this observation.

Keywords Osteosarcoma · Fluoride · Fluoridation · Case–control

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Introduction

Osteosarcoma is a very rare primary malignant tumor of bone. Although uncommon, primary malignant bone tumors comprise the sixth most common group of malignant tumors in children and the third most common malignant tumor for adolescents, with an annual incidence rate of 5.6 per million for Caucasian children under 15 years old [1]. Osteosarcoma is the most common tumor of bone and for patients less than 20 years old more than 80% of these tumors tend to occur in the long bones of the appendicular skeleton which are undergoing rapid growth [2]. The incidence of osteosarcoma is slightly higher in males than females with an annual incidence rate of approximately 3.5 per million for males and 2.9 per million for females under the age of 24 years [3].

The etiology of osteosarcoma is largely unknown [1, 4]. In humans, ionizing radiation is the only environmental agent known to cause bone cancer and is thought to have an effect in approximately 3% of cases from either external high-dose irradiation used in cancer therapy or internal bone-seeking radioisotopes from occupational or medical use [1, 5, 6]. Alkylating agents used in chemotherapy are thought to increase the risk for osteosarcoma and evidence for other etiologic factors including viruses, antecedent trauma, or radium in drinking water has been suggested but inconclusive [1, 5, 7, 8]. Certain pre-existing bone defects including Paget's disease have been found more frequently in patients who subsequently developed bone cancers [1, 4, 5]. Also, a genetic predisposition for osteosarcoma has been described, specifically for patients with a hereditary form of retinoblastoma or those with familial Li-Fraumeni cancer syndrome [1, 5, 6].

The age-incidence distribution of osteosarcoma is bimodal, raising the possibility of different risk factors contributing to the incidence of osteosarcoma at different ages. The first and larger peak in incidence occurs in the second decade of life and a subsequent peak occurs in males in the eighth decade of life [2, 4, 5, 9]. Evidence suggests that osteosarcoma is associated with skeletal growth, particularly for patients diagnosed during adolescence [1, 9–11]. Since fluoride may act as a mitogen (increasing the proliferation of osteoblasts) and its uptake in bone increases when skeletal growth is more rapid [12, 13], it is biologically plausible that fluoride exposure during growth is associated with the subsequent development of osteosarcoma, and fluoride could either increase or decrease the rate of osteosarcoma.

There are conflicting data regarding the association between fluoride exposure and the incidence of osteosarcoma. Several animal studies have been conducted, but only one found evidence that fluoride exposure may increase osteosarcoma formation, specifically in male rats [14]. Human studies also show conflicting results. The majority of epidemiologic studies found no association between fluoride and osteosarcoma [15–21]. However, two studies found evidence of an association in males under age 20, but not in females [22, 23]. Furthermore, prior studies have primarily evaluated fluoride exposure at the time of diagnosis or as an average lifetime exposure and have not evaluated exposure at specific ages during growth and development when cell division is occurring rapidly.

Therefore, we use data from the Harvard Fluoride Osteosarcoma Study [24] to explore age-specific and sex-specific effects and evaluate exposure to fluoride in drinking water from birth through early adolescence. Specifically, based on prior studies suggesting an effect of fluoride limited to males under age 20, we limited our analysis to the first two decades of life and evaluated effects in males and females separately.

Materials and methods

We used data from a hospital-based, matched case–control study which evaluated lifetime exposure to fluoride from drinking water and self-administered fluoride products [24]. Subjects were identified through the orthopedic departments at 11 teaching hospitals across the United States. Cases had histologically confirmed osteosarcoma diagnosed between November 1989 and November 1992. Exclusion criteria were: age 40 years or older, any history of radiation therapy or a history of renal dialysis. Controls were patients of the same hospital's orthopedics department, seen within ±6 months of the case's diagnosis and matched with cases on age (±5 years), gender, distance from hospital, with the same exclusion criteria applied to cases. Telephone interviews were conducted between January 1992 and January 1995 with the parent or subject (over 18 years old) or with a proxy if subjects were deceased or incapacitated. Interviewers collected information which included a complete residential history, use of fluoride supplements and mouth rinses. Study procedures were approved by the Harvard Medical School Committee on Human Studies and each of the participating institutions. Data on a total of 419 subjects, 139 cases and their 280 matched controls, were available based on eligibility criteria, matching criteria and a completed interview. However, we limit the current analysis to 103 cases less than 20 years old and 215 controls matched to these cases.

Fluoride level in drinking water was the primary exposure of interest. The interview obtained the usual type of the subject's drinking water (municipal, private well, bottled) and the subject's age(s) while at each address. From these data, we estimated the level of fluoride in drinking water for each subject at each age, and explored the effects of fluoride during their growth and development. To estimate fluoride concentration for public water supplies, we obtained preliminary data from the 1985 CDC Fluoridation Census [25] and the 1992 CDC Fluoridation Census [26]. We then contacted state agencies (State Dental Director's Office, State EPA Office of Drinking Water, Water Administrators Office) and local sources (county health departments, the town or city clerk's office and specific water systems) to confirm and supplement the CDC data [27]. For subjects who drank well water, a sample was obtained from current or former residents for the specific appropriate addresses. Fluoride concentrations were measured at Harvard School of Dental Medicine using a Colorimeter (Model 41100-21, Hach Company, Ames, IA). Subjects who used bottled water as their usual source of drinking water were identified, but information about specific brand was not collected. We estimated fluoride

levels to be 0.1 parts per million (ppm) in bottled water based on the weighted average of fluoride concentration in leading brands [28]. Since subjects who used bottled water were also likely to consume fluoride from tap water in food and beverage preparation and use outside the home (e.g., school), we used the mean of fluoride estimates for bottled water (i.e., 0.1 ppm) and municipal water for these residences. Since water consumption may vary based on climate, we standardized fluoride exposure estimates based on CDC recommendations for optimal target levels of fluoride [29]. For example, for locations in warmer climates where the target fluoride level is 0.7 ppm, we divided fluoride levels by 0.7, while for locations in colder climates where the target fluoride level is 1.2 ppm we divided by 1.2. The standardization of fluoride exposure was done for all three types of drinking water.

We created a proxy measure for socioeconomic status (SES) by linking zip code at the time of diagnosis with data from the Census Bureau that provide 1989 median family income for each zip code. Median family income was categorized into quartiles based on the distribution for controls. We also used data from the Census Bureau to determine the 1990 population of the county where subjects resided at the time of diagnosis, categorized by approximate tertiles. We examined type of drinking water by including indicators for use of bottled water or well water at any time up to the exposure age. Since age matching allowed for a difference as large as 5 years, we included age (at diagnosis for cases and at time of hospital treatment for controls) as a covariate. Lastly, since information was collected for use of self-administered fluoride products at home or in school-based programs, we included an indicator for any use of these products as an additional covariate.

We used conditional logistic regression to estimate the odds ratio for the association between fluoride exposure and osteosarcoma, taking into account the matching between cases and controls. The dependent variable was an indicator identifying cases and the primary independent variables were measures of fluoride exposure. We fit two basic models. The first model included only the exposure measures as independent variables. The second model also included age, a proxy for SES, county population, use of private well water or bottled water, and any use of fluoride supplements or mouth rinses as covariates. In this analysis, our a priori hypothesis was that fluoride exposure may have sex-specific differential effects on osteosarcoma risk based on age at exposure. The models we employed therefore do not assess the question of average induction time or latency.

We report the mean and standard deviation of fluoride levels in ppm and percent of target for each specific age. To examine the association between osteosarcoma and fluoride exposure at specific ages, we fit separate models for each exposure age up to the age of diagnosis for each case and the same age for the matched controls. Each model included the age-specific fluoride level and a sex–fluoride interaction term. In this analysis we expect substantial correlation in exposure to fluoride in drinking water from year to year, limiting our ability to identify age-specific effects precisely. For our primary analysis we categorized climate-standardized fluoride exposure into three categories (<30%, 30–99%, >99% of target fluoride content) corresponding to approximate tertiles based on the distribution among controls. We plot sex-specific estimates of the odds ratio and 95% confidence intervals as a function of exposure age. We also fit a model using fluoride exposure categorized without standardization by climate into three groups (<0.3, 0.3–0.69, and ≥0.7 ppm).

We performed a sensitivity analysis on our assumption that the fluoride content of bottled water is 0.1 ppm by fitting models using values as high as 0.5 ppm for bottled water (assuming that bottled water and municipal water each contributed half of the consumption for subjects who used bottled water). In addition, we conducted a sensitivity analysis evaluating the age-specific and sex-specific effects of fluoride in drinking water among subjects who reported never having used any fluoride supplements or fluoride mouth rinses.

Results

A total of 157 cases diagnosed before age 20 were identified at the participating hospitals. No interviews were completed for 13 of the cases (did not attempt to contact, could not contact, or respondent refused). Eleven cases used well water for which no sample was obtained and 12 cases lived outside the United States for more than 6 months. An additional 18 cases with interview data were excluded due to lack of appropriately matched controls (nine had no eligible matches identified or successfully interviewed, seven whose only matches used well water for which no sample was obtained and two whose matches lived outside the United States >6 months). Characteristics of the remaining 103 cases and their 215 matched controls are presented in Table 1. Cases were diagnosed at a median age of 14 years (range 6–19, interquartile range 11–17). Residential histories for six participants, five cases and one control, were provided by proxies (grandparents, stepparent, sibling, aunt, neighbor). The 1989 median family income for zip code of residence was lower for cases than controls (P=0.01, Student's t-test) and a larger proportion of controls used bottled water (P=0.002, chi-square test). Table 2 shows the average fluoride level and percent of climate-specific target level in drinking water at each age for cases and controls.

Table 1 Characteristics of study population[a]

	Cases	Controls
Number	103	215
Age (years)	13.7 ± 3.5	14.5 ± 3.9
Gender		
Male	60 (58%)	122 (57%)
Female	43 (42%)	93 (43%)
Self-reported race[b]		
White	81 (79%)	180 (84%)
Black	16 (16%)	23 (11%)
Asian	3 (3%)	2 (1%)
Other	3 (3%)	9 (4%)
Number of residences	2.5 ± 1.7	2.6 ± 1.7
1989 Median family income[c]	$41,458 ± 15,146	$46,841 ± 19,319
County population[c]		
<250,000	37 (37%)	69 (32%)
250,000–999,999	44 (44%)	86 (40%)
1,000,000+	19 (19%)	60 (28%)
Hospital		
MGH	17 (17%)	27 (13%)
CH, Boston	15 (15%)	45 (21%)
Creighton	5 (5%)	11 (5%)
CH, DC	11 (11%)	20 (9%)
MSKCC	7 (7%)	14 (7%)
U Chicago	8 (8%)	16 (7%)
Rush	3 (3%)	6 (3%)
U Florida	12 (12%)	19 (9%)
UCLA	14 (14%)	32 (15%)
Cleveland clinic	8 (8%)	19 (9%)
CWRU	3 (3%)	6 (3%)
Ever well water use	29 (28%)	44 (20%)
Ever bottled water use	8 (8%)	46 (21%)
Fluoride Products		
Rinses	3 (3%)	19 (9%)
School program	17 (17%)	30 (14%)
Tablets	10 (10%)	28 (13%)
Drops	9 (9%)	19 (9%)
Any of above	27 (26%)	77 (36%)

[a] Values reported are mean ± standard deviation or *n* (%)

[b] Race not available for one control

[c] 1989 Median family income and county population data not available for three cases

Figure 1 shows the odds ratio, relative to the lowest exposure group, of osteosarcoma for the climate-standardized fluoride level at each exposure age from 0 to 14 years, estimated using the conditional logistic regression models unadjusted for other covariates. Among males, exposure to fluoride at or above the target level was associated with an increased risk of developing osteosarcoma (Fig. 1a). The association was most apparent between ages 4 and 12 with a peak at 6–8 years of age. The odds ratio for the high exposure group was 4.07 at 7 years of age with a 95% confidence interval of 1.43–11.56. Among females less than 20 years old, no association between fluoride in drinking water and osteosarcoma was apparent at any age (Fig. 1b).

Next we fit models with all the covariates. As an example, Table 3 shows the model for subjects at 7 years

Table 2 Fluoride level for drinking water[a]

	F level in ppm		Percent of target	
	Cases	Controls	Cases	Controls
Age (years)				
0	0.63 ± 0.40	0.60 ± 0.41	66% ± 41%	62% ± 41%
1	0.63 ± 0.40	0.60 ± 0.40	65% ± 41%	61% ± 40%
2	0.64 ± 0.40	0.61 ± 0.40	67% ± 41%	63% ± 40%
3	0.67 ± 0.39	0.63 ± 0.39	69% ± 40%	64% ± 39%
4	0.70 ± 0.40	0.62 ± 0.39	73% ± 41%	63% ± 39%
5	0.69 ± 0.40	0.63 ± 0.39	72% ± 41%	65% ± 38%
6	0.70 ± 0.40	0.62 ± 0.39	74% ± 41%	63% ± 39%
7	0.70 ± 0.38	0.61 ± 0.39	75% ± 40%	63% ± 39%
8	0.69 ± 0.38	0.61 ± 0.39	73% ± 40%	63% ± 38%
9	0.68 ± 0.39	0.63 ± 0.38	73% ± 41%	65% ± 38%
10	0.67 ± 0.39	0.61 ± 0.39	71% ± 41%	63% ± 39%
11	0.70 ± 0.56	0.60 ± 0.39	74% ± 65%	62% ± 39%
12	0.69 ± 0.56	0.59 ± 0.39	75% ± 66%	61% ± 39%
13	0.68 ± 0.39	0.61 ± 0.39	71% ± 41%	62% ± 38%
14	0.65 ± 0.41	0.59 ± 0.39	69% ± 43%	61% ± 38%

[a] When bottled water was used, the estimate was 0.1 ppm for bottled water and it was assumed that bottled water and municipal supply each accounted for 50% of consumption

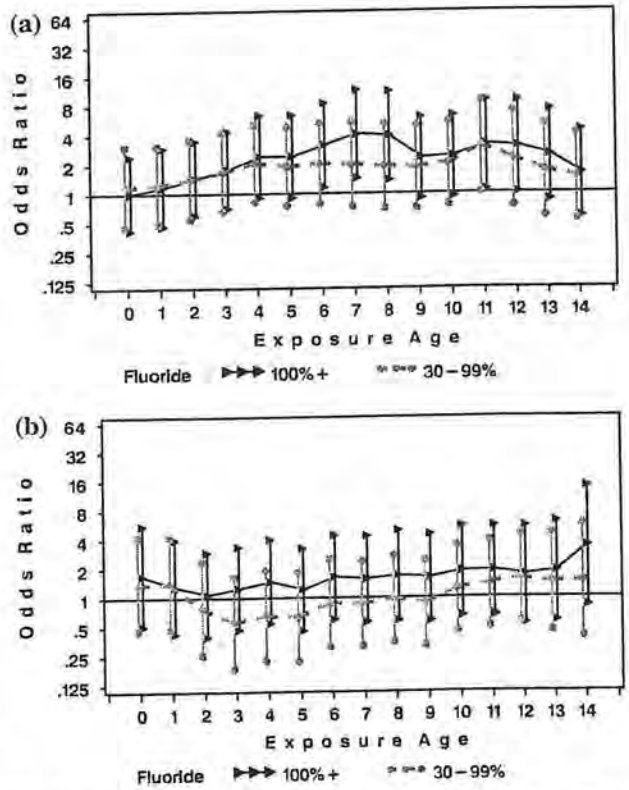

Fig. 1 Odds ratios and 95% confidence intervals relative to fluoride levels less than 30% of target are shown for males (panel a) and for females (panel b). The dashed line shows the odds ratios for the intermediate exposure category (30–99% of target fluoride level) and the solid line shows the odds ratios for the high exposure category (100% of target or greater)

Table 3 Sex-specific associations between fluoride exposure at age 7 years and osteosarcoma, estimated by conditional logistic regression

Fluoride exposure at age 7 years	Odds ratio (95% C.I.)[a]
Males	
Less than 30% of target	1.00
30–99% of target	3.36 (0.99, 11.42)
At least 100% of target	5.46 (1.50, 19.90)
Females	
Less than 30% of target	1.00
30–99% of target	1.39 (0.41, 4.76)
At least 100% of target	1.75 (0.48, 6.35)

[a] Adjusted for age, zip code median income, county population, use of well water by age 7, use of bottled water by age 7, any use of fluoride supplements

of age. Figure 2 shows a similar effect of fluoride level in drinking water after adjusting for income by zip code, county population, ever use of bottled or well water, age, and any use of self-administered fluoride products. For males, the odds ratio for the high exposure group was 5.46 at 7 years of age with a 95% confidence interval of 1.50–19.90. Sensitivity analyses, which assumed that the fluoride

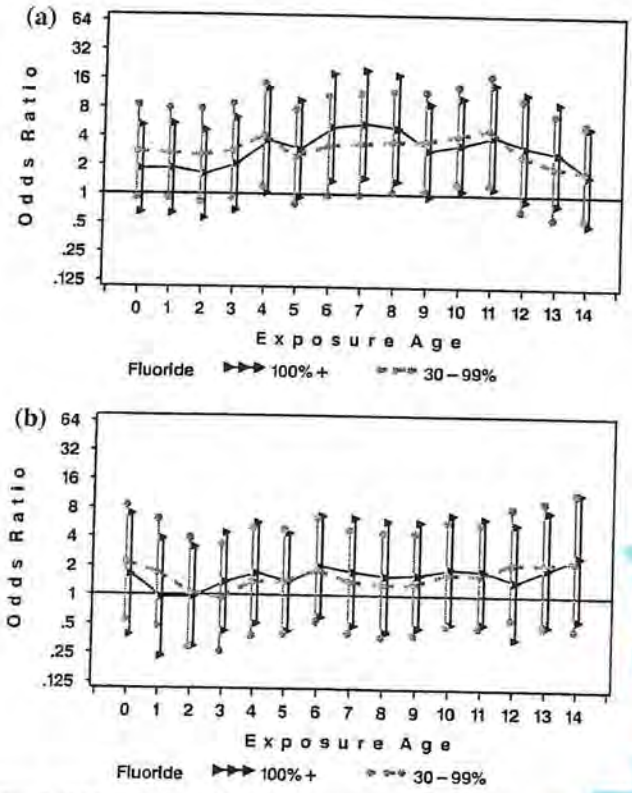

Fig. 2 Odds ratios and 95% confidence intervals relative to fluoride levels less than 30% of target are shown for males (panel a) and for females (panel b). The dashed line shows the odds ratios for the intermediate exposure category (30–99% of target fluoride level) and the solid line shows the odds ratios for the high exposure category (100% of target or greater). Estimates are adjusted for age, zip code median income, county population, prior use of well water, prior use of bottled water, and any use of fluoride supplements

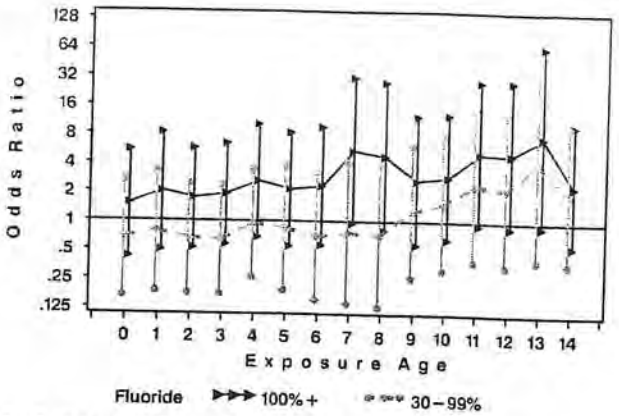

Fig. 3 Odds ratios and 95% confidence intervals relative to fluoride levels less than 30% of target are shown for the subset of male participants who never used fluoride supplements or rinses. The dashed line shows the odds ratios for the intermediate exposure category (30–99% of target fluoride level) and the solid line shows the odds ratios for the high exposure category (100% of target or greater)

content of bottled was as low as 0.1 ppm or as high as 0.5 ppm, yielded essentially identical results. A sensitivity analysis that categorized fluoride exposure based on the absolute fluoride concentration, without standardizing for climate-specific target fluoride level, also showed essentially the same results (unadjusted OR=3.77; 95% CI 1.41, 10.05, and adjusted OR=5.55; 95% CI 1.60, 19.24 for 0.7 ppm or greater relative to less than 0.3 ppm). To avoid potential confounding by fluoride supplementation or fluoride rinses, we conducted a sensitivity analysis restricting our population to subjects who reported that they did not use supplements or rinses. This substantially reduced the sample size limiting us to unadjusted analyses for males. The results were consistent (Fig. 3).

Discussion

Our exploratory analysis described the association of fluoride level in drinking water at specific ages and the incidence of osteosarcoma. We observed that for males diagnosed before the age of 20 years, fluoride level in drinking water during growth was associated with an increased risk of osteosarcoma, demonstrating a peak in the odds ratios from 6 to 8 years of age. All of our models were remarkably robust in showing this effect, which coincides with the mid-childhood growth spurt [30–33]. For females, no clear association between fluoride in drinking water during growth and osteosarcoma emerged.

We found similar effect magnitudes in the intermediate and high exposure levels, as opposed to a dose–response gradient. This may be due to misclassification of the primary exposure for some artificially fluoridated systems.

Reeves [34] reported that only 65% of fluoridated water systems routinely have target levels of fluoride maintained in the drinking water, which may result in our misclassifying up to 35% of the adjusted water systems, categorizing them in the highest group (100% of target or greater) when some truly belong in the middle group (30–99% of target). While non-differential misclassification of exposure results in bias towards the null for a dichotomous exposure, Birkett [35] has shown that with three levels of exposure, the estimated odds ratio for the highest exposure level is biased towards the null, but for the intermediate category the estimate can be biased in either direction. Hence, in our study the misclassification might mask an effect that increases with dose.

Our results are consistent with a pattern seen in the National Toxicology Program (NTP) animal study and two ecological studies. The NTP animal study, which reported "equivocal evidence" for an association between fluoride and osteosarcoma, found a positive association for male rats, but no association for female rats or mice of either gender [14]. Using data from the Surveillance, Epidemiology and End Results (SEER), Hoover et al. found an unexplained increase in osteosarcoma in males less than 20 years of age in fluoridated versus non-fluoridated areas. However, a time-trend analysis which took into account the duration of fluoride exposure failed to demonstrate a higher incidence among males exposed to fluoridated water their entire lives than among those exposed less than half their lives [22]. A similar, but smaller study examining osteosarcoma in New Jersey also showed an increase in incidence rates for males less than 20 years old who lived in fluoridated areas compared to those living in non-fluoridated areas [23].

A number of other case–control studies did not find an association between fluoride in drinking water and osteosarcoma [18–21]. In addition, preliminary analyses of an ongoing case–control study of the determinants of osteosarcoma conducted at the same network of hospitals that participated in the present study and recruited cases during their initial hospitalization, found no overall association between lifetime exposure to fluoride or fluoride content in bone biopsies, a marker of cumulative exposure, and osteosarcoma (personal communication, Chester Douglass, D.M.D., Ph.D.). This lack of agreement may be related to the bimodal age-incidence distribution of osteosarcoma [2, 4, 5, 9]. When there are two distinct peaks in an age-incidence distribution, two distinct sets of component causes should be considered [36]. McGuire et al. [19] and Moss et al. [20] included cases up to age 40 years and 84 years, respectively, and if fluoride exhibits a different effect according to the age-specific distribution, detecting an effect would be unlikely. Operskalski et al. [18] selected friends and neighbors of the cases as controls, which might have been optimal for some exposures of interest, but resulted in inadvertently matching on drinking water fluoride level. The evaluation of age-specific effects distinguishes our study from the other investigations. Rothman [37] has warned that failure to identify the appropriate time window for exposure may result in misclassification which can adversely affect the ability to detect an association. This might explain why the study by Gelberg et al. [21] did not find an association between fluoride in drinking water and osteosarcoma since age-specific effects were not evaluated.

It is biologically plausible that fluoride affects the incidence rate of osteosarcoma, and that this effect would be strongest during periods of growth, particularly in males. First, approximately 99% of fluoride in the human body is contained in the skeleton with about 50% of the daily ingested fluoride being deposited directly into calcified tissue (bone or dentition) [13]. Second, fluoride acts as a mitogen, increasing the proliferation of osteoblasts [12, 38] and its uptake in bone increases during periods of rapid skeletal growth [13]. In the young, the hydroxyapatite structure of bone mineral exists as many extremely small crystals each surrounded by an ion-rich hydration shell, providing a greater surface area for fluoride exchange to occur [39, 13]. Also, osteosarcoma, for the ages we considered, generally originates in the metaphyseal areas of long bones [2] and the pattern of the blood supply to the metaphyses and epiphyses, where growth of long bones takes place, differs from that of the diaphyses because of the special circulation to the epiphyseal growth plate in the young which in turn disappears when growth is complete [40, 41]. Lastly, the amount of fluoride present in bone depends on gender and intake [39] and intake, on average, is greater for males than females for all ages over 1 year [42].

There are several limitations to our study. First, our estimates of fluoride in drinking water at each residence do not reflect actual consumption by subjects and the study did not obtain biologic markers for fluoride uptake in bone. However, dietary sources of fluoride comprise the majority of human exposure [13], and for individuals living in fluoridated communities, the fluoride in drinking water is estimated to contribute two-thirds of the total dietary intake [39]. Also, when we added use of self-administered (home- or school-based) products as a covariate in the model, there was no substantial change in results. The *halo* or *diffusion* effect, described in the dental literature, refers to people in non-fluoridated communities receiving fluoride from food and beverages processed in fluoridated communities and vice versa [43]. We would expect this type of measurement error to result in a bias towards underestimating any true effect that might exist.

Because cases and controls moved rarely up to the age at diagnosis (an average of 1.5 times) leading to essentially

collinear exposure from year to year, we were unable to apply statistical models that assess the effect of age-specific exposure while simultaneously adjusting for exposure at other ages such as distributed lag models. Residential histories were obtained from proxies more often among cases than controls, however the absolute number was small and the proxies were generally close relatives.

The estimation of fluoride concentration at each residence is subject to several sources of measurement error. Monitoring guidelines for fluoridated water systems permit actual fluoride levels to vary. For example, if the target fluoride concentration for a specific water system is 1.0 ppm, guidelines may consider values between 0.8 and 1.3 ppm acceptable. Also, natural fluoride levels may vary over time, but they are unlikely to do so for the length of time subjects lived at their respective address unless the water source changed. For bottled-water users, we did not know the specific brands consumed and a small proportion of brands on the market do have substantial levels of fluoride. However, analysis of the leading national brands makes a value of 0.1 ppm a reasonable estimate [28]. Further, we demonstrated that our findings were not sensitive to this assumption.

The lack of data available for other potential confounders is also a limitation. Fluoride may not be the causative agent; instead there may be another factor in drinking water correlated with the presence of fluoride. Data to assess fluoride exposure in diet, industrial fluoride exposure or other fluoride exposures (e.g., pesticides) were not available. Instead, by including type of drinking water subjects used (ever well, ever bottled) as a covariate, we may have partially controlled for some of the "other unknown factors" such as contaminants or carcinogens subjects might have been exposed to irrespective of fluoride concentration in these natural sources or products.

Another limitation is the possibility of selection bias. In our case–control study, the secondary study base is defined by the cases and in order for the results to be valid the exposure distribution for controls must represent the exposure distribution in this theoretical population. Referral patterns to the participating hospitals may differ for cases and controls because the participating hospitals were primary referral centers for osteosarcoma for large regions but the controls likely represented a more proximate population. Further, for some of the hospitals the referral base for controls could represent different socioeconomic populations than for cases. Distance from hospitals was used as a matching factor, to limit selection bias. This matching factor could also result in some overmatching on exposure, resulting in possible underestimation of the effect. Additionally, we included the 1989 median family income and county population as covariates.

For this study, cases of osteosarcoma that were diagnosed at participating hospitals between November 1989 and November 1992 were identified. However, case and control interviews took place later, between January 1992 and January 1995. Although efforts were made to interview a parent or proxy respondent if the subject was deceased or incapacitated, it is possible that cases with more favorable prognosis may have been over-sampled. If this occurred, an alternative explanation for our observation is that boys exposed to higher levels of fluoride who subsequently develop osteosarcoma have a better prognosis than boys exposed to lower levels. While we cannot rule out this possibility, the magnitude of the protective effect that would be required to explain the observed association is unlikely.

Differential recall of exposure information between cases and controls is unlikely in the current study because respondents did not provide information about the fluoride level in their drinking water but rather a complete residential history. For other covariates, such as date of birth, sex, or zip code at time of diagnosis, information was obtained by medical record review. Reporting of the type of water used or the use of self-administered fluoride products could be affected by recall bias.

In summary, this exploratory analysis found an association between exposure to fluoride in drinking water and the incidence of osteosarcoma, demonstrating a peak in the odds ratio for exposure at ages 6–8 years among males diagnosed less than 20 years old, but no consistent association among females. Future studies would benefit from the inclusion of biomarkers of fluoride exposure and assessment of potential gene–environment interactions. Such studies with larger numbers of osteosarcoma patients, with incidence under age 20, that examine age-specific and sex-specific associations are required to confirm or refute the findings of the current study.

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Fluoride Exposure and Childhood Osteosarcoma: A Case–Control Study

ABSTRACT

Objectives. This study tests the hypothesis that fluoride exposure in a nonoccupational setting is a risk factor for childhood osteosarcoma.

Methods. A population-based case–control study was conducted among residents of New York State, excluding New York City. Case subjects (n = 130) were diagnosed with osteosarcoma between 1978 and 1988, at age 24 years or younger. Control subjects were matched to case subjects on year of birth and sex. Exposure information was obtained by a telephone interview with the subject, parent, or both.

Results. Based on the parents' responses, total lifetime fluoride exposure was not significantly associated with osteosarcoma among all subjects combined or among females. However, a significant protective trend was observed among males. Protective trends were observed for fluoridated toothpaste, fluoride tablets, and dental fluoride treatments among all subjects and among males. Based on the subjects' responses, no significant associations between fluoride exposure and osteosarcoma were observed.

Conclusions. Fluoride exposure does not increase the risk of osteosarcoma and may be protective in males. The protective effect may not be directly due to fluoride exposure but to other factors associated with good dental hygiene. There is also biologic plausibility for a protective effect. (*Am J Public Health.* 1995;85:1678–1683)

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Introduction

Although the benefit to dental health of fluoride exposure has been clearly established, the release of the National Toxicology Program study in which a dose–response relationship for osteosarcoma was indicated for exposure to sodium fluoride among male rats has provoked criticism of water fluoridation programs.[1] In response, the Department of Health and Human Services conducted a review of fluoride's benefits and risks and recommended that analytical epidemiologic studies of osteosarcoma be conducted to determine the risk factors associated with its development.[2]

Osteosarcoma is the fourth most common cancer in persons under 25 years of age[3] occurring most often around puberty.[4] The only known etiological agent is radiation[5]; other suggested risk factors include a rapid rate of bone growth, previous bone trauma, and viruses.[6–8] Persons with the hereditary form of retinoblastoma or with the Li–Fraumeni cancer family syndrome are at high risk for osteosarcoma.[9,10]

Fluoride is deposited directly into the bone, with about 99% of fluoride in the body contained in the skeleton.[1,2] Children, who are actively forming bone, have a higher amount of uptake of fluoride into the bone matrix than adults.[1,2] Fluoride uptake into bone results in an increased rate of osteoblast proliferation and bone formation.[11] Bone in the areas of the knees, ankles, shoulders, and wrists, where childhood osteosarcomas most often occur, shows a high response to fluoride.[12]

Toxicological studies of sodium fluoride have yielded mixed results.[1,13–15] In in vitro studies fluoride appears to be mutagenic and can induce chromosome aberrations, sister chromatid exchanges, cytotoxicity, and neoplastic transformation in cultured mammalian cells.[1,13,14] The recent study conducted by the National Toxicology Program found equivocal evidence for a carcinogenic effect among male F344/N rats, but there was no evidence for carcinogenicity in female F344/N rats, nor in male or female mice.[1] Another study sponsored by the Procter and Gamble company found no carcinogenic evidence in Sprague–Dawley rats.[16]

Ecological studies generally have found no relationship between fluoride levels in drinking water and osteosarcoma and bone cancer incidence or mortality rates.[17–23] Individual exposures were examined in only two small studies.[24,25] One study based on only 20 males found that males under age 20 years who resided in communities with fluoridated water at the time of diagnosis had a higher osteosarcoma rate than those who resided in communities with nonfluoridated water.[24] The other study had only 22 matched case–control pairs and found no associa-

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Note. The views expressed here are the authors' and do not necessarily represent those of the National Cancer Institute.

tion between osteosarcoma and lifetime fluoride exposure from drinking water.[25]

Within New York State, average annual osteosarcoma incidence rates from 1976 to 1987 in fluoridated areas were found not to differ from rates in nonfluoridated areas.[23] To further investigate the potential association of fluoride exposure with childhood osteosarcoma in New York State, excluding New York City, we conducted a population-based case–control study. All sources of fluoride except dietary sources were examined separately and were combined to estimate total lifetime fluoride exposure. Because deleterious effects were limited to male rats in the National Toxicology Program study, additional analyses were conducted by gender.

Methods

Study Population

Cases of osteosarcoma newly diagnosed from January 1978 through December 1988 were identified from the New York State Cancer Registry. Case subjects who were 24 years of age or younger and residing in New York State, excluding New York City, at the time of diagnosis were eligible for inclusion in the study. Case subjects with preexisting cancers were excluded, resulting in a case population of 171.

Control subjects were pair-matched one-to-one to case subjects by year of birth and sex. Potential control subjects were randomly selected from live birth records maintained by the New York State Department of Health. All children born in New York City were excluded. Control subjects were assigned the same age at diagnosis as the corresponding case subjects as a reference date to provide equal time periods at risk. Control subjects had to survive until their matched case subject's age at diagnosis.

Case and control subjects and their parents were traced to determine their vital status and to locate their current address and telephone number. Although it was easier to trace the case subjects than the control subjects because more current information was available, an exhaustive search was made for each potential control subject before another one was selected as a replacement. An average of 2.10 birth certificates were searched before an adequate control subject was located and interviewed.

TABLE 1—Number of Case and Control Subjects, Odds Ratios (ORs), and 95% Confidence Intervals (CIs) for Lifetime Fluoride Exposure Variables (Parents' Data Set)

	No. Subjects				
	Case	Control	OR	95% CI	*P*
Tablets, mg					.03
0	110	104	1.00	...	
1–250	3	4	0.81	0.18, 3.66	
251–550	7	4	1.72	0.50, 5.91	
551–3500	2	10	0.11	0.01, 0.88	
Mouth rinses, mg					.43
0	110	111	1.00	...	
1–7	5	2	4.02	0.44, 36.75	
8–50	4	4	1.03	0.23, 4.57	
51–1005	3	5	0.60	0.14, 2.65	
Toothpaste, mg					.06
0–433	38	23	1.00	...	
434–862	26	35	0.43	0.20, 0.92	
863–1425	30	31	0.54	0.25, 1.19	
1426–2235	28	33	0.49	0.23, 1.06	
Dental treatments, mg					.06
0	88	91	1.00	...	
15	25	17	1.52	0.75, 3.08	
30–60	4	6	0.75	0.21, 2.72	
75–390	5	8	0.64	0.18, 2.21	
Water, mg					.61
0	40	57	1.00	...	
1–1850	32	16	4.13	1.65, 10.35	
1851–3385	26	23	1.84	0.81, 4.20	
3386–6100	24	26	1.40	0.60, 3.29	
Total fluoride, mg					.24
0–1235	31	31	1.00	...	
1236–2161	31	29	1.04	0.50, 2.14	
2162–4101	34	27	1.20	0.56, 2.57	
4102–8433	26	35	0.67	0.29, 1.54	

Note. Odds ratios were estimated from conditional logistic models.

Interviews

A telephone interview was requested from all living study subjects who were at least 18 years of age. Permission to interview the subject's parent was requested during the interview. If a case subject refused the interview, permission was requested to interview the case subject's parent. If permission was not granted, the parent was not interviewed. If the subjects were deceased or too young for an interview (<18 years old), the contact letters were sent directly to the parents. If a control subject did not allow us to contact his or her parents (n = 3), or if the parents refused to be interviewed after the control subject was interviewed (n = 6), the control subject was replaced.

The interview focused on the subjects' sociodemographic, medical, and exposure histories before the date of diagnosis or reference date. Fluoride exposure information was obtained from questions about the use of fluoridated products (toothpastes and mouth rinses) and fluoride supplements (drops, tablets, vitamins, and dental treatments). In addition, a complete residential history from birth until the age of diagnosis or reference age was taken. This history included complete addresses, the years of residence at each address, the water source (public supply or private well) at each address, and whether the water at each address was fluoridated.

There were a total of 130 case–control pairs for which the subject or the parent or both were interviewed for both members of the pair. Sixty-four (49%) interviews were completed for the case subjects, 126 (97%) for the case parents, 119 (92%) for the control subjects, and 126 (97%) for the control parents. Ninety percent of the parents who were interviewed were biologic mothers. The primary reasons for not obtaining interviews

TABLE 2—Number of Case and Control Subjects, Odds Ratios (ORs), and 95% Confidence Intervals (CIs) for Lifetime Fluoride Exposure Variables for Males (Parents' Data Set)

	No. Subjects				
	Case	Control	OR	95% CI	*P*
Tablets, mg					.08
0	73	67	1.00	. . .	
1–250	2	2	1.00	0.14, 7.10	
251–550	1	3	0.33	0.03, 3.21	
551–3500	2	6	0.20	0.02, 1.71	
Mouth rinses, mg					.99
0	73	72	1.00	. . .	
1–7	2	2	0.90	0.05, 17.89	
8–50	2	2	0.81	0.09, 7.52	
51–1005	1	2	0.46	0.04, 5.81	
Toothpaste, mg					.01
0–433	12	27	1.00	. . .	
434–862	23	15	0.23	0.08, 0.70	
863–1425	19	21	0.41	0.14, 1.18	
1426–2235	24	15	0.25	0.09, 0.70	
Dental treatments, mg					.04
0	60	56	1.00	. . .	
15	14	11	1.00	0.39, 2.55	
30–60	1	5	0.20	0.02, 1.80	
75–390	3	6	0.50	0.12, 2.07	
Water, mg					.62
0	27	34	1.00	. . .	
1–1850	20	11	2.81	0.97, 8.09	
1851–3385	15	12	1.67	0.58, 4.77	
3386–6100	16	21	0.93	0.31, 2.83	
Total fluoride, mg					.02
0–1235	17	19	1.00	. . .	
1236–2161	17	23	1.14	0.46, 2.84	
2162–4101	17	19	0.78	0.27, 2.22	
4102–8433	27	17	0.41	0.14, 1.22	

Note. Odds ratios were estimated from conditional logistic models.

were the subject being deceased (42% of the case subjects), inability to locate the subject or parent (8% of the case subjects, 42% of the control subjects), and refusal by the subject or parent to participate in the study. Approximately 6% of the case subjects, control subjects, and control parents refused, and 12% of the case parents refused. Eleven case subjects and their matched control subjects were too young for interviews.

Fluoride Exposure Index

To analyze the relationship between fluoride exposure and osteosarcoma, the lifetime exposure to each source of fluoride was determined, and these were summed into a total lifetime fluoride exposure index. These sources included fluoride drops, tablets, and vitamins, fluoridated mouth rinses and toothpastes, dental fluoride treatments, and fluoride from drinking water and breast milk. It was not possible to measure fluoride from food, which ranges from 6% to 32% of total fluoride intake.[26]

For more than 96% of the addresses identified, the respondent indicated knowledge of whether the water supply was public or private. These data were validated by geocoding all addresses and matching them to census data. There was 96% agreement between the water source according to the 1990 census and the water source reported by interview.

The subjects or parents indicated knowledge of the fluoridation status of their water for only 40% of the addresses. Therefore, instead of relying on the interview information, all of the addresses were further investigated to determine fluoridation status. Natural fluoride levels are relatively low in New York State, so the water was considered not fluoridated for all addresses with private wells in New York. Because fluoridation often follows town boundaries, addresses identified to be within city limits were then compared with a fluoridation census.[27] Telephone calls were made to appropriate agencies to determine the fluoridation status of addresses that could not be classified with the aforementioned method.

The average amount of fluoride ingested by age for each fluoride source was determined from the literature. For example, the dose recommended by the American Dental Association for fluoride drops, tablets, and vitamins was 0.25 mg per day for an infant newborn to 2 years old, 0.50 mg for a 2- to 3-year-old, and 1.0 mg for a child 3 to 13 years of age for the time period of this study.[28–30]

Population-based estimates of tap water intake were used to determine the amount of water ingested by age and sex categories. The estimates were derived from the 1977 and 1978 US Department of Agriculture Nationwide Food Consumption Survey, and the mean estimates for the northeast geographic region (all seasons) were used.[31] The fluoride level in water was assumed to be 1.0 mg/liter for fluoridated areas and 0 mg/liter for nonfluoridated regions.

Cumulative lifetime exposure for each fluoride source was estimated in milligrams by multiplying the amount ingested per exposure by the number of times per day exposed by the total number of days exposed. The lifetime exposures for each fluoride source were then summed to create a total lifetime fluoride exposure index.

Apart from dental fluoride treatments, for which there was a large amount of missing data (approximately 23% of the parents and 8% of the subjects), fewer than 5% of the parents' responses and fewer than 2% of the subjects' responses were missing. The percentage of missing responses did not differ between case and control subjects. A standard set of rules was established to impute values for missing data.

To measure intensity of exposure, each lifetime fluoride exposure variable was divided by the age at diagnosis or reference age to get an average annual exposure. Although matched pairs would still have the same within-pair association because of the matching by age, the relationship among pairs would change with this measure.

Analysis

Because recall could be different between the subjects and parents, separate data sets were created for each of these data sources, maintaining the matching. Sixty-four matched pairs were in-

cluded in the subjects' data set and 122 matched pairs were included in the parents' data set.

EGRET was used to analyze matched observations of each variable against disease status.[32] Odds ratios, 95% confidence intervals, and *P* values were computed by creating conditional logistic models. *P* values for trend were calculated by including the variables in models in their original, noncategorized continuous form. The *P* value for the likelihood ratio statistic reflecting the difference between the model with and the model without the continuous variable was interpreted as the *P* value for trend, which indicated whether the linear component of the trend was statistically significant. The *P* values do not necessarily appear to correspond to the trends of the categorical variables as presented because of the creation of arbitrary cutpoints in the continuous variables for presentation purposes. Extensive subgroup analyses were not conducted due to limitations presented by the relatively small number of subjects in the sample.

Results

Case subjects who were final study subjects (case subject and/or parent was interviewed) were not significantly different from case subjects for whom no interview was obtained (neither case subject nor parent was interviewed) with respect to race, vital status, age at diagnosis, year of diagnosis, stage of tumor, and anatomic location of tumor. However, a statistically significant higher percentage of case subjects not interviewed were male (61% vs 32%). Of the 130 case subjects who were final study subjects, 42 (32%) were male, 51 (39%) were deceased, and 96 (74%) were between ages 10 and 19 years. Eighteen case subjects (14%) but only 4 control subjects (3%) were non-White. This difference was statistically significant ($P = .002$).

The bivariate relationships between osteosarcoma and lifetime exposure to fluoride from tablets, mouth rinses, toothpaste, dental treatments, and drinking water, along with the total lifetime fluoride exposure index, are shown in Table 1 for the parents' data set. Because of the small number of affirmative responses, the fluoride from drops and the fluoride from vitamins were not analyzed separately. Fluoride from toothpaste and total lifetime fluoride exposure were categorized into quartiles. However, because so many individuals did not have exposure to the other fluoride sources, the lowest-level category for these variables included only those individuals with no exposure. Tables 2 and 3 present results for the same lifetime fluoride variables for males and females, respectively.

TABLE 3—Number of Case and Control Subjects, Odds Ratios (ORs), and 95% Confidence Intervals (CIs) for Lifetime Fluoride Exposure Variables for Females (Parents' Data Set)

	No. Subjects				
	Case	Control	OR	95% CI	*P*
Tablets, mg					.20
0	37	37	No convergence		
1–250	1	2			
251–550	6	1			
551–3500	0	4			
Mouth rinses, mg					.30
0	37	39	No convergence		
1–7	3	0			
8–50	2	2			
51–1005	2	3			
Toothpaste, mg					.85
0–433	11	11	1.00	...	
434–862	12	11	0.93	0.31, 2.80	
863–1425	12	9	0.65	0.17, 2.47	
1426–2235	9	13	1.80	0.45, 7.18	
Dental treatments, mg					.70
0	28	35	1.00	...	
15	11	6	2.25	0.69, 7.61	
30–60	3	1	3.00	0.31, 28.84	
75–390	2	2	1.00	0.06, 15.99	
Water, mg					.12
0	13	23	1.00	...	
1–1850	12	5	10.55	1.22, 91.04	
1851–3385	11	11	1.65	0.41, 6.59	
3386–6100	8	5	2.81	0.62, 12.69	
Total fluoride, mg					.24
0–1235	14	12	1.00	...	
1236–2161	12	8	0.74	0.20, 2.69	
2162–4101	10	15	1.81	0.56, 5.82	
4102–8433	8	9	1.34	0.32, 5.57	

Note. Odds ratios were estimated from conditional logistic models.

Total lifetime fluoride exposure was not significantly associated with osteosarcoma among all subjects combined or among females. However, a significant protective trend was observed among males ($P = .02$). With respect to the individual sources of fluoride, a significant trend of decreasing risk with higher exposure was observed among all subjects for tablets ($P = .03$). The trends for toothpaste ($P = .06$) and for dental treatments ($P = .06$) were borderline significant and were also protective. The lowest exposure level for toothpaste was significantly protective for all subjects and for females; and the highest exposure level was significantly protective for tablets, further emphasizing the protective effect. Significant or borderline significant protective trends were also observed for each of these variables among males. The lowest exposure level for water had a significantly elevated odds ratio for all subjects and for females; however, trends were not significant.

The relationships between osteosarcoma and lifetime exposure to fluoride from the various sources, along with the total lifetime fluoride exposure index, are shown in Table 4 for the subjects' data set. Because of the small number of affirmative responses, fluoride from tablets was not analyzed separately. Although there appears to be an increasing risk with exposure, especially for the total fluoride intake, no significant trends were observed and all confidence intervals included 1.0. Models could not be run separately for each sex because of the small number of individuals in this data set.

For both the parents' and subjects' data sets, results of analyses controlling

TABLE 4—Number of Case and Control Subjects, Odds Ratios (ORs), and 95% Confidence Intervals (CIs) for Lifetime Fluoride Exposure Variables (Subjects' Data Set)

	No. Subjects				
	Case	Control	OR	95% CI	P
Mouth rinses, mg					.14
0	55	56	1.00	. . .	
1–35	3	2	1.50	0.25, 8.98	
36–150	2	4	0.55	0.10, 3.06	
151–950	4	2	1.83	0.33, 10.21	
Toothpaste, mg					.22
0–615	15	17	1.00	. . .	
616–1149	14	18	0.89	0.31, 2.61	
1150–1444	15	16	1.03	0.36, 2.97	
1445–3411	20	13	1.93	0.64, 5.84	
Dental treatments, mg					.52
0	45	45	1.00	. . .	
15	8	12	0.77	0.31, 1.96	
30–45	7	2	3.21	0.63, 16.50	
60–300	4	5	0.98	0.25, 3.93	
Water, mg					.48
0	21	29	1.00	. . .	
1–1950	15	11	2.31	0.74, 7.20	
1951–3350	14	12	2.07	0.53, 8.02	
3351–5650	14	12	1.76	0.59, 5.21	
Total fluoride, mg					.25
0–1250	14	18	1.00	. . .	
1251–2338	15	17	1.16	0.44, 3.04	
2339–3987	17	15	1.72	0.55, 5.39	
3988–9291	18	14	1.88	0.64, 5.55	

Note. Odds ratios were estimated from conditional logistic models.

for race and maternal age (which was found to be negatively associated with osteosarcoma in these data) were similar to the bivariate analyses. The results of the analyses for the average annual fluoride exposure variables were essentially the same as the lifetime exposure analyses in both data sets.

Discussion

The total lifetime fluoride exposure index is the most important indicator of whether fluoride is significantly associated with osteosarcoma. In the parents' data set, a significant association was not observed among all subjects, but a significant protective trend was observed among males. Borderline significant or significant protective trends were also observed for lifetime fluoride exposure from tablets, toothpaste, and dental treatments among all subjects and among males only. In the subjects' data set, however, a protective trend was not observed for the total lifetime fluoride exposure index, nor for any of the individual lifetime fluoride exposure variables. Importantly, there was no statistically significant finding from either data set that fluoride exposure increases the risk of childhood osteosarcoma. This result is consistent with previous studies that found no association between fluoride exposure and osteosarcoma.[17–23,25]

The protective effects observed in the parents' data set may be due to concern for personal health and hygiene and not to fluoride exposure. Those individuals who use fluoride tablets, who brush their teeth more often with fluoridated toothpaste, and who receive dental fluoride treatments are possibly more involved with good health practices. The observed protective effects could be the result of healthy behavior practices or of correlates of health behaviors that protect against osteosarcoma, rather than a consequence of fluoride exposure, although it is unusual to find these practices more among boys. The fact that the protective effect was not observed for fluoridated water supports this argument. However, because fluoride is directly deposited into the bone and directly affects the bone structure, it is biologically plausible that the protective effect observed from fluoride exposure could, in fact, be real.

The only demographic variables significantly associated with osteosarcoma in this study were race and maternal age. In general, race is not considered a risk factor for osteosarcoma.[3,33–36] The procedure used to randomly select the control birth certificates did not produce the same percentage of non-White certificates as the percentage from the total live births for upstate New York (7.6% vs 12.8%). Of those certificates obtained for non-White control subjects, a substantially higher proportion did not contain the father's name compared with certificates obtained for White control subjects (33.3% among non-Whites vs 3.9% among Whites), making the non-White control subjects more difficult to trace. Neither race nor maternal age was observed to confound the osteosarcoma–fluoride relationship.

One major limitation in this study was that cases were identified retrospectively for the period 1978 to 1988. Problems with recall became exacerbated because of the long period of time that may have passed since the childhood exposures. Also, only 64 case subjects were directly interviewed because of deaths occurring after diagnosis. Despite this limitation, the case subjects, control subjects, and their parents were equally able to report on the fluoride-related exposures, with a low percentage of missing responses. Because neither the case nor the control group appeared to be more accurate in their reporting of exposures, any misclassification that occurred should be nondifferential and would therefore bias the results toward the null value.

The extent of nondifferential misclassification of fluoride exposure and resultant bias toward the null value are difficult to evaluate. We are confident that the water fluoridation information is accurate. There is no reason to suspect that residential histories were reported inaccurately, the water source information was validated by geocoding, and fluoridation status was objectively determined. However, subjects' and parents' accuracy in reporting exposure to other sources of fluoride could not be assessed.

The low response rate of 48% among control subjects (mainly due to an inability to trace them) was a concern. However, among study subjects, individuals who moved more than two times did not have significantly different total lifetime fluoride exposures than individuals who maintained one or two addresses up to the

diagnosis or reference age. This suggests that the nonparticipant control subjects who could not be traced because they moved often would not have differed from the participant control subjects with respect to total lifetime fluoride exposure.

A strength of this study was the relatively large sample size (122 case–control pairs for the parents' data set and 64 case–control pairs for the subjects' data set) compared with prior studies that examined individual fluoride exposures from drinking water.[27,28] Another advantage of this study was the inclusion of exposures to fluoride from sources other than drinking water.

The differences in results between the parents' and subjects' data sets are probably a reflection of differences in knowledge and recall. Parents were probably more aware of exposures that occurred at young ages, including use of fluoridated drops, tablets, vitamins, and toothpastes and exposure to dental treatments. Subjects probably provided more accurate information for exposures that occurred when they were older, particularly the use of fluoridated toothpastes and mouth rinses. Overall, the parents' data set is probably the more accurate one because of better knowledge of more types of fluoride exposure and when those exposures began.

Conclusion

In conclusion, this study provides no support for the hypothesis that fluoride exposure increases the risk for osteosarcoma. It contributes to the body of evidence that indicates that the public can continue to enjoy the dental health benefits of fluoride with no associated major risks. □

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The Effects Of Fluoride On The Thyroid Gland

By Dr Barry Durrant-Peatfield MBBS LRCP MRCS
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9-9-4

There is a daunting amount of research studies showing that the widely acclaimed benefits on fluoride dental health are more imagined than real. My main concern however, is the effect of sustained fluoride intake on general health. Again, there is a huge body of research literature on this subject, freely available and in the public domain.

But this body of work was not considered by the York Review when their remit was changed from "Studies of the effects of fluoride on health" to "Studies on the effects of fluoridated water on health." It is clearly evident that it was not considered by the BMA (Britsh Medical Association), British Dental Association (BDA), BFS (British Fluoridation Society) and FPHM, (Faculty for Public Health and Medicine) since they all insist, as in the briefing paper to Members of Parliament - that fluoridation is safe and non-injurious to health.

This is a public disgrace, I will now show by reviewing the damaging effects of fluoridation, with special reference to thyroid illness.

It has been known since the latter part of the 19th century that certain communities, notably in Argentina, India and Turkey were chronically ill, with premature ageing, arthritis, mental retardation, and infertility; and high levels of natural fluorides in the water were responsible. Not only was it clear that the fluoride was having a general effect on the health of the community, but in the early 1920s Goldemberg, working in Argentina showed that fluoride was displacing iodine; thus compounding the damage and rendering the community also hypothyroid from iodine deficiency.

Highly damaging to the thyroid gland

This was the basis of the research in the 1930s of May, Litzka, Gorlitzer von Mundy, who used fluoride preparations to treat over-active thyroid illness. Their patients

either drank fluoridated water, swallowed fluoride pills or were bathed in fluoridated bath water; and their thyroid function was as a result, greatly depressed. The use in 1937 of fluorotyrosine for this purpose showed how effective this treatment was; but the effectiveness was difficult to predict and many patients suffered total thyroid loss. So it was given a new role and received a new name, Pardinon. It was marketed not for over-active thyroid disease but as a pesticide. (Note the manufacturer of fluorotyrosine was IG Farben who also made sarin, a gas used in World War II).

This bit of history illustrates the fact that fluorides are dangerous in general and in particular highly damaging to the thyroid gland, a matter to which I shall return shortly. While it is unlikely that it will be disputed that fluorides are toxic - let us be reminded that they are Schedule 2 Poisons under the Poisons Act 1972, the matter in dispute is the level of toxicity attributable to given amounts; in today's context the degree of damage caused by given concentrations in the water supply. While admitting its toxicity, proponents rely on the fact that it is diluted and therefore, it is claimed, unlikely to have deleterious effects.

They could not be more mistaken

It seems to me that we must be aware of how fluoride does its damage. It is an enzyme poison. Enzymes are complex protein compounds that vastly speed up biological chemical reactions while themselves remaining unchanged. As we speak, there occurs in all of us a vast multitude of these reactions to maintain life and produce the energy to sustain it. The chains of amino acids that make up these complex proteins are linked by simple compounds called amides; and it is with these that fluorine molecules react, splitting and distorting them, thus damaging the enzymes and their activity. Let it be said at once, this effect can occur at extraordinary low concentrations; even lower than the one part per million which is the dilution proposed for fluoridation in our water supply.

The body can only eliminate half

Moreover, fluorides are cumulative and build up steadily with ingestion of fluoride from all sources, which include not just water but the air we breathe and the food we eat. The use of fluoride toothpaste in dental hygiene and the coating of teeth are further sources of substantial levels of fluoride intake. The body can only eliminate half of the total intake, which means that the older you are the more fluoride will have accumulated in your body. Inevitably this means the ageing population is particularly targeted. And even worse for the very young there is a major element of risk in baby formula made with fluoridated water. The extreme sensitivity of the very young to fluoride toxicity makes this unacceptable. Since there are so many sources

of fluoride in our everyday living, it will prove impossible to maintain an average level of 1ppm as is suggested.

What is the result of these toxic effects?

First the immune system. The distortion of protein structure causes the immune proteins to fail to recognise body proteins, and so instigate an attack on them, which is Autoimmune Disease. Autoimmune diseases constitute a body of disease processes troubling many thousands of people: Rheumatoid Arthritis, Systemic Lupus Erythematosis, Asthma and Systemic Sclerosis are examples; but in my particular context today, thyroid antibodies will be produced which will cause Thyroiditis resulting in the common hypothyroid disease, Hashimoto's Disease and the hyperthyroidism of Graves' Disease.

Musculo Skeletal damage results further from the enzyme toxic effect; the collagen tissue of which muscles, tendons, ligaments and bones are made, is damaged. Rheumatoid illness, osteoporosis and deformation of bones inevitably follow. This toxic effect extends to the ameloblasts making tooth enamel, which is consequently weakened and then made brittle; and its visible appearance is, of course, dental fluorosis.

The enzyme poison effect extends to our genes; DNA cannot repair itself, and chromosomes are damaged. Work at the University of Missouri showed genital damage, targeting ovaries and testes. Also affected is inter uterine growth and development of the foetus, especially the nervous system. Increased incidence of Down's Syndrome has been documented.
Fluorides are mutagenic. That is, they can cause the uncontrolled proliferation of cells we call cancer. This applies to cancer anywhere in the body; but bones are particularly picked out. The incidence of osteosarcoma in a study reporting in 1991 showed an unbelievable 50% increase. A report in 1955 in the New England Journal of Medicine showed a 400% increase in cancer of the thyroid in San Francisco during the period their water was fluoridated.

My particular concern is the effect of fluorides on the thyroid gland

Perhaps I may remind you about thyroid disease. The thyroid gland produces hormones which control our metabolism - the rate at which we burn our fuel. Deficiency is relatively common, much more than is generally accepted by many medical authorities: a figure of 1:4 or 1:3 by mid life is more likely. The illness is insidious in its onset and progression. People become tired, cold, overweight, depressed, constipated; they suffer arthritis, hair loss, infertility, atherosclerosis and chronic illness. Sadly, it is poorly diagnosed and poorly managed by very many

doctors in this country.
What concerns me so deeply is that in concentrations as low as 1ppm, fluorides damage the thyroid system on 4 levels.

1. The enzyme manufacture of thyroid hormones within the thyroid gland itself. The process by which iodine is attached to the amino acid tyrosine and converted to the two significant thyroid hormones, thyroxine (T4) and liothyronine (T3), is slowed.

2. The stimulation of certain G proteins from the toxic effect of fluoride (whose function is to govern uptake of substances into each of the cells of the body), has the effect of switching off the uptake into the cell of the active thyroid hormone.

3. The thyroid control mechanism is compromised. The thyroid stimulating hormone output from the pituitary gland is inhibited by fluoride, thus reducing thyroid output of thyroid hormones.

4. Fluoride competes for the receptor sites on the thyroid gland which respond to the thyroid stimulating hormone; so that less of this hormone reaches the thyroid gland and so less thyroid hormone is manufactured.
These damaging effects, all of which occur with small concentrations of fluoride, have obvious and easily identifiable effects on thyroid status. The running down of thyroid hormone means a slow slide into hypothyroidism. Already the incidence of hypothyroidism is increasing as a result of other environmental toxins and pollutions together with wide spread nutritional deficiencies.

141 million Europeans are at risk

One further factor should give us deep anxiety. Professor Hume of Dundee, in his paper given earlier this year to the Novartis Foundation, pointed out that iodine deficiency is growing worldwide. There are 141 million Europeans are at risk; only 5 European countries are iodine sufficient. UK now falls into the marginal and focal category. Professor Hume recently produced figures to show that 40% of pregnant women in the Tayside region of Scotland were deficient by at least half of the iodine required for a normal pregnancy. A relatively high level of missing, decayed, filled teeth was noted in this non-fluoridated area, suggesting that the iodine deficiency was causing early hypothyroidism which interferes with the health of teeth. Dare one speculate on the result of now fluoridating the water?

Displaces iodine in the body

These figures would be worrying enough, since they mean that iodine deficiency, which results in hypothyroidism (thyroid hormone cannot be manufactured without

iodine) is likely to affect huge numbers of people. What makes it infinitely worse, is that fluorine, being a halogen (chemically related to iodine), but very much more active, displaces iodine. So that the uptake of iodine is compromised by the ejection, as it were, of the iodine by fluorine. To condemn the entire population, already having marginal levels of iodine, to inevitable progressive failure of their thyroid system by fluoridating the water, borders on criminal lunacy.
I would like to place a scenario in front of those colleagues who favour fluoridation. A new pill is marketed. Some trials not all together satisfactory, nevertheless, show a striking improvement in dental caries. Unfortunately, it has been found to be thyrotoxic, mutagenic, immunosuppressive, cause arthritis and infertility in comparatively small doses over a relatively short period of time.

Do you think it should be marketed?
Fluoridation of the nation's water supply will do little for our dental health; but will have catastrophic effects on our general health. We cannot, must not, dare not, subject our nation to this appalling risk.

Dr Barry Durrant-Peatfield
obtained his Medical degrees in 1960 at Guy's Hospital London. He left the NHS in 1980 to specialise in thyroid illnesses drawing inspiration from the work of infamous Dr Broda Barnes, at the Foundation that bears his name, Connecticut, USA. He has been a medical practitioner for over forty years specialising in metabolic disorders during which time he became a leading authority in the UK for thyroid and adrenal management. For over twenty years he also ran a successful private clinic and became a nation-wide leading authority on thyroid and adrenal dysfunction, but clashed with establishment medicine in the management of thyroid illness. He is the author of The Great Thyroid Scandal (see opposite page), he currently lectures at nutritional colleges in London as well as conducting his own teaching seminars. Barry will shortly be opening a diagnostic clinic in the UK for thyroid and adrenal disorders where he will provide advice on diagnosis and treatment with special interests in nutritional aspects. For further information contact: Dr B Durrant-Peatfield 36A High St, Mersham, Redhill Surrey, RH1 3EA.
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Sarin: (GB: isopropyl methylphosono-fluoridate) is a colorless, odorless volatile liquid, soluble in water, first synthesized at IG Farben in 1938. It kills mainly through inhalation.

Cyclosarin (GF) and Thiosarin are variants. Pennsylvania Department of Health http://www.dsf.health.state.pa.us/health/cwp/view.asp?a=171&q=233740

Sarin: (GB: CH3-P(=O)(-F)(-OCH(CH3)2)
Source: A FOA Briefing Book on Chemical Weapons
http://www.opcw.org/resp/html/nerve.html Gerhard Schrader, a chemist at IG Farben, was given the task of developing a pesticide. Two years later a phosphorus compound with extremely high toxicity was produced for the first time.
IG Farben: "...the board of American IG Farben had three directors from the Federal Reserve Bank of New York, the most influential of the various Federal Reserve Banks. American IG Farben. also had interlocks with Standard Oil of New Jersey, Ford Motor Company, Bank of Manhattan (later to become the Chase Manhattan Bank), and AEG. (German General Electric) Source: Moody's Manual of Investments; 1930, page 2149."

http://reformed-theology.org/html/books/wall_street/chapter_02.htm

At a later date, Namaste will be publishing a more in-depth article outlining the devastating affects that fluoride, aspartame and MSG have on the endocrine system. Dr Durrant-Peatfield wiill be answering frequently asked questions on thyroid illness in Namaste's next issue. Send your questions to us preferably by email to: info@namastepublishing.co.uk

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Nucl Med Commun. 2012 Jan;33(1):14-20.

Association of vascular fluoride uptake with vascular calcification and coronary artery disease.

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Abstract

OBJECTIVE: The feasibility of a fluoride positron emission tomography/computed tomography (PET/CT) scan for imaging atherosclerosis has not been well documented. The purpose of this study was to assess fluoride uptake of vascular calcification in various major arteries, including coronary arteries.

METHODS: We retrospectively reviewed the imaging data and cardiovascular history of 61 patients who received whole-body sodium [^{18}F]fluoride PET/CT studies at our institution from 2009 to 2010. Fluoride uptake and calcification in major arteries, including coronary arteries, were analyzed by both visual assessment and standardized uptake value measurement.

RESULTS: Fluoride uptake in vascular walls was demonstrated in 361 sites of 54 (96%) patients, whereas calcification was observed in 317 sites of 49 (88%) patients. Significant correlation between fluoride uptake and calcification was observed in most of the arterial walls, except in those of the abdominal aorta. Fluoride uptake in coronary arteries was demonstrated in 28 (46%) patients and coronary calcifications were observed in 34 (56%) patients. There was significant correlation between history of cardiovascular events and presence of fluoride uptake in coronary arteries. The coronary fluoride uptake value in patients with cardiovascular events was significantly higher than in patients without cardiovascular events.

CONCLUSION: sodium [^{18}F]fluoride PET/CT might be useful in the evaluation of the atherosclerotic process in major arteries, including coronary arteries. An increased fluoride uptake in coronary arteries may be associated with an increased cardiovascular risk.

PMID:21946616[PubMed - in process]

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ORIGINAL ARTICLE

Acute Fluoride Poisoning from a Public Water System

Bradford D. Gessner, Michael Beller, John P. Middaugh, and Gary M. Whitford

N Engl J Med 1994; 330:95-99 January 13, 1994

Abstract Article References Citing Articles (17)

Since the late 1940s, many communities in the United States have adjusted the fluoride concentration in their water systems to prevent dental caries[1]. Numerous studies attest to the effectiveness and safety of maintaining fluoride concentrations in the range recommended by the Public Health Service -- 0.7 to 1.2 mg per liter[2,3]. As of 1989, a total of 9411 public water systems in the United States provided fluoridated drinking water to 135 million people,[4] yet only six outbreaks of acute fluoride poisoning related to overfluoridation have been reported[5-10]. Acute fluoride poisoning caused the death of one patient in Maryland[8] and, recently, of three patients in Illinois (Flanders R, Illinois Department of Public Health: personal communication), all of whom were undergoing dialysis therapy. In this paper we describe an outbreak of acute fluoride poisoning resulting from overfluoridation of a public water system.

METHODS

Background

Hooper Bay, Alaska, is a small village on the Bering Sea populated predominantly by Alaska Natives. The village has two geographically distinct sections with separate wells and water systems: sections 1 (population, 470) and 2 (population, 375). Neither water system provided running water to individual homes; residents carried water from holding tanks to their homes, where it was stored for domestic use. On May 26, 1992, the Alaska Division of Public Health was notified of an outbreak of acute gastrointestinal illness in the village. The water system in section 1 had been turned off on May 23 after staff members at the Hooper Bay health clinic noted that many residents had become ill shortly after drinking water from that system. Since acute fluoride poisoning produces a clinical syndrome characterized by nausea, vomiting, diarrhea, abdominal pain, and paresthesias and because tests conducted during the six weeks before the outbreak had shown fluoride concentrations in water system 1 of 6.5 and 20.0 mg per liter, acute fluoride poisoning was suspected.

The outbreak resulted in one serious illness and one death. On May 23, a 37-year-old woman with a two-day history of vomiting and diarrhea was evacuated by air to a regional hospital. Her serum calcium concentration was 5.2 mg per deciliter (1.3 mmol per liter), and her serum fluoride concentration was 9.1 mg per liter (480 µmol per liter) (the normal fasting serum fluoride concentration in persons using drinking water containing 1 mg of fluoride per liter is 0.01 to 0.03 mg per liter [0.5 to 1.6 µmol per liter]). She recovered without apparent sequelae. On May 23, after 24 hours of intractable vomiting, a 41-year-old man was found dead at home. He had attempted to remain hydrated by drinking an estimated 10 liters of water from water system 1 on May 21 and 22. His only known medical problem was peptic ulcer disease, for which he took cimetidine. The postmortem serum calcium concentration was 4.9 mg per deciliter (1.22 mmol per liter), and the urinary fluoride concentration (not corrected for creatinine content) was 55 mg per liter (2900 µmol per liter).

Epidemiologic Study

To determine the duration of the outbreak, we reviewed the health clinic records of all patients seen from May 1 to May 25 with nausea, vomiting, diarrhea, or abdominal pain. Of 31 patients with these

symptoms, 22 were seen on May 21, 22, or 23. We therefore defined a case patient with fluoride poisoning as a resident who had at least one of the following symptoms on May 21, 22, or 23: nausea, vomiting, diarrhea, abdominal pain, or numbness or tingling of the face or extremities.

On May 27 we went to the clinic and asked patients who were seen there on May 21, 22, or 23 to come to the clinic with any available family members. These persons, as well as patients being seen at the clinic for any reason on May 27, were interviewed as described below. On May 28 we identified case patients (and control subjects) through a household survey conducted by starting at one randomly selected household in each section of the village and walking door to door until residents of 15 households in section 1 and 17 households in section 2 had been interviewed. We collected information on all residents of each household, and adult residents were asked to provide information about anyone not at home. Since case patients identified at the clinic and through the household survey were similar with respect to age and onset, duration, and type of symptoms, we considered all case patients together.

All residents interviewed on May 27 or 28 responded to a questionnaire that included questions about symptoms and water consumption. Information about persons under 18 years of age was obtained from a parent or guardian. Some residents had difficulty recalling the details of their illness or their water consumption; results are given only for those able to recall the details.

To evaluate risk factors and calculate attack rates, we conducted a case-control study and a retrospective cohort study. The case-control study analyzed the case patients and control subjects who were interviewed at the clinic on May 27; it compared the characteristics of persons meeting the definition of a case patient with those of persons who did not. All persons interviewed on May 27 who did not meet the case-patient definition were considered controls for this analysis. The retrospective cohort study analyzed the case patients and control subjects identified during the household survey. Since almost all the acutely ill persons lived in section 1, we used the cohort study to estimate the total number with fluoride poisoning.

Biochemical Measurements

On May 27 and 28, we collected blood and spot urine specimens from 20 case patients and blood or urine alone from 7 case patients. Urine specimens were also collected from 15 control subjects, 3 of whom also provided a blood sample. Urine was analyzed for fluoride and creatinine. Serum was analyzed for fluoride and, in most cases, aspartate aminotransferase, calcium, creatine kinase, lactate dehydrogenase, magnesium, and phosphorus.

Follow-up urine specimens were collected from case patients with the highest initial urinary fluoride concentrations. Six case patients provided a specimen on June 5 or June 9, and three provided specimens on both dates. On June 9 follow-up blood specimens were collected from 11 case patients who had an abnormal result on serum-chemistry testing or an elevated serum fluoride concentration.

Urinary fluoride was measured by direct ion-specific electrode potentiometry and corrected for the creatinine content by dividing the measured fluoride concentration (in milligrams per liter) by the urinary creatinine concentration (in milligrams per deciliter) and then multiplying the value by 100 mg per deciliter. Serum fluoride was measured in the same manner after overnight diffusion by the hexamethyldisiloxane-facilitated technique[11].

Environmental Investigation

We reviewed the records of routine fluoride determinations for the Hooper Bay water systems and collected water samples from the two systems and from residents who still had water from system 1 in their homes. The Alaska Department of Environmental Conservation inspected both water systems and had water samples analyzed for fluoride using protocols approved by the Environmental Protection Agency.

Dose Estimates

To estimate the dose of fluoride ingested, we asked each case patient to recall how much water he or she had consumed that had been obtained from system 1 from May 21 to 23. Beverages made with water were included in the calculation, but water consumed in food was not. The estimated dose was calculated as the volume of water consumed (in liters) multiplied by the presumed fluoride concentration of the water (in milligrams per liter) divided by body weight (in kilograms). The presumed fluoride concentration was based on the results of the environmental investigation.

Statistical Analysis

Odds ratios, relative risks, and 95 percent confidence intervals were calculated with the Epi Info program[12]. Pearson's correlation coefficients were determined by the least-squares method[13]; we report corresponding two-tailed P values. The confidence interval for the estimated number of section 1 residents with fluoride poisoning was calculated according to the method of Levy and Lemeshow[14].

RESULTS

Epidemiologic Study

Overall we identified 91 case patients; all were Alaska Natives, ranging in age from 6 months to 73 years (median, 21 years), and 51 percent were female. The most common symptoms were nausea, vomiting, and abdominal pain (Table 1). All case patients had stopped drinking water from system 1 by May 23. Most case patients began to have symptoms on May 22 (Figure 1). The median interval between the consumption of water and the onset of symptoms was 7 minutes (range, <1 to 150), and the median duration of symptoms was 24 hours (range, 1 to 132).

TABLE 1

Symptoms of 91 Case Patients with Acute Fluoride Poisoning in Hooper Bay, Alaska, in May 1992.

There were 42 case patients and 54 control subjects in the case-control study. As compared with the controls, the case patients were 7 times more likely to live in section 1, 18.5 times more likely to consume water from water system 1, and 76 times more likely to have consumed water obtained from water system 1 on May 21, 22, or 23 (Table 2).

There were 175 persons in the retrospective cohort study. The attack rates among residents of sections 1 and 2 were 63 percent and 2 percent, respectively (Table 3). Among residents of section 1, the attack rate among those who usually drank water from system 1 was 71 percent, whereas among those who drank water obtained from system 1 from May 21 to 23, the attack rate was 91 percent (Table 3). Six residents of section 1 who were sick but who had not drunk water collected from system 1 on May 21, 22, or 23 recalled drinking water obtained on May 20. The attack rate among the residents of section 1 implies that 296 of the 470 residents (63 percent) had acute fluoride poisoning (95 percent confidence interval, 249 to 343).

FIGURE 1

Onset of Symptoms of Acute Fluoride Poisoning in 89 Case Patients and Onset of Gastrointestinal Symptoms in 15 Other Residents of Hooper Bay, Alaska, in May 1992.

Biochemical Measurements

For specimens collected on May 27 or 28, the median urinary fluoride concentrations in case patients and control subjects were 3.4 mg per liter (180 µmol per liter) and 1.7 mg per liter (89 µmol per liter), respectively. Ten case patients and no control subjects had elevated urinary fluoride concentrations (>5.0 mg per liter [>260 µmol per liter]). The median serum fluoride concentrations were 0.067 mg per liter (3.5 µmol per liter) in the case patients and 0.029 mg per liter (1.5 µmol per liter) in the control subjects. The serum fluoride concentrations in the case patients correlated strongly with the duration of illness ($r = 0.84$, $P<0.001$).

TABLE 2

Odds Ratios for Various Types of Exposure in a Case-Control Study of Acute Fluoride Poisoning.

The median urinary fluoride concentrations in case patients retested on June 5 and 9 were 6.4 mg per liter (340 µmol per liter) and 4.4 mg per liter (230 µmol per liter), respectively. The urinary fluoride concentrations decreased consistently from May 27 or 28 to June 9; for the persons retested on both June 5 and 9, the decrease was nonlinear.

TABLE 3

Attack Rates and Relative Risks for Various Types of Exposure in a Retrospective Cohort Study of Acute Fluoride Poisoning.

All 14 case patients tested on May 27 or 28 had an elevated serum lactate dehydrogenase concentration (median, 152 U per liter). Eleven case patients had elevated serum aspartate aminotransferase concentrations, eight had hypomagnesemia, three had hyperphosphatemia, and one had an elevated creatine kinase concentration. All serum calcium values were normal. Serum lactate dehydrogenase concentrations correlated with the urinary ($r = 0.74$, $P = 0.002$) and serum ($r = 0.57$, $P = 0.004$) fluoride concentrations obtained on May 27 or 28. Among 11 case patients retested on June 9, 6 had hypomagnesemia, 3 had hyperphosphatemia, and 1 had an elevated serum lactate dehydrogenase concentration; the median serum fluoride concentration was 0.086 mg per liter (4.5 µmol per liter).

Environmental Investigation

Water system 1 included a 6340-liter (1675 gal) holding tank, two 95-liter (25 gal) chemical vats (for chlorine and fluoride concentrates), a water pump and two chemical feed pumps, high and low floats connected to an electrical control system to regulate the volume of water in the holding tank, and a public watering point outside the well house (Figure 2). Regulations requiring that results of monthly fluoride measurements be submitted to the state had been unmet for almost two years. The operator lacked formal training and could not correctly perform fluoride tests. High fluoride concentrations were documented in January 1991 (7.3 mg per liter) and again six and three weeks before the outbreak (6.5 and 20 mg per liter, respectively). After the third report of an elevated fluoride concentration, local health officials asked the water-system operator to drain the holding tank and unplug the fluoride pump. On May 26, 1992, however, the fluoride pump was still operating. Water obtained from water system 1 by residents on May 20 and 21 had fluoride concentrations of 2

FIGURE 2

Diagram of Water System 1.

and 150 mg per liter, respectively; water obtained on May 27 had a fluoride concentration of 58 mg per liter. Water obtained from system 2 on May 27 had a fluoride concentration of 1.1 mg per liter.

Major electrical and mechanical defects of water system 1 were identified. The control system was unreliable and did not activate the water pump consistently. The fluoride pump performed four times faster than expected and, because of improper electrical wiring, could be activated independently of the water pump. Because of these defects, the fluoride concentration of a full holding tank could be increased from 0 to 150 mg per liter in 26 hours. Finally, under certain operating conditions, the fluoride concentrate (18,000 mg per liter) could be siphoned into the well if the hose was kept connected to the drop pipe (Figure 2) and its free end was placed in the fluoride vat. After the outbreak, tests demonstrated that siphonage could have led to the elevated fluoride concentration by emptying a full fluoride vat in several minutes.

Dose Estimates

Among 62 case patients able to remember how much water they had consumed from water system 1 from May 21 to 23, reports ranged from 2 to 140 ml per kilogram of body weight (median, 27). Assuming that the fluoride concentration of all water collected from May 21 to 23 was 150 mg per liter, the fluoride doses ranged from 0.3 to 21.0 mg per kilogram; 21 persons received an estimated dose of less than 2.0 mg per kilogram, and 10 received doses below 1.0 mg per kilogram. The man who died received an estimated dose of 17.9 mg per kilogram. The estimated fluoride dose strongly correlated with the urinary ($r = 0.78$, $P<0.001$) and serum ($r = 0.71$, $P<0.001$) fluoride concentrations obtained on May 27 or 28 and with the duration of illness ($r = 0.58$, $P<0.001$).

DISCUSSION

These results indicate that excess fluoride entered a community water system in a rural Alaska village, causing 1 death and almost 300 nonfatal cases of fluoride intoxication. The symptoms can be explained by well-described mechanisms[15]. Fluoride and hydrogen ions combine in the stomach to form hydrofluoric acid, which causes nausea, vomiting, diarrhea, and abdominal pain. Fluoride has a direct toxic effect on intracellular metabolism that includes the inhibition of glycolytic enzymes and cholinesterases. Profound hyperkalemia may result. Finally, fluoride forms a complex with calcium in extracellular fluid that causes hypocalcemia; the fate of the complex is not known. Our findings suggest that serum magnesium concentrations may also be reduced; the mechanism for this reduction is unknown. Death from fluoride poisoning is believed to occur from cardiac dysrhythmias due to hyperkalemia or hypocalcemia[16-18].

Although the interval between water consumption and the onset of symptoms was consistent with that in other reports,[7,19,20] the median duration of symptoms — 24 hours — was longer than the previously reported range of less than 1 hour to 5.5 hours[9,10,19]. The prolonged elevation of serum and urinary fluoride concentrations was also unexpected; more than two weeks after the outbreak, the median serum fluoride concentration among the case patients who were retested was two to three times normal. The half-life of fluoride in serum has been estimated to be 2.4 to 4.3 hours,[21] and after the ingestion of a low dose, the serum fluoride concentration usually returns to normal within 24 hours[22]. The prolonged elevation of serum and urinary fluoride concentrations may have been due to the continued ingestion of water with a high fluoride concentration. This explanation is unlikely, since water system 1 was shut off immediately after the outbreak was recognized, most residents discarded all water obtained from the system, and fluoridation of water system 2 was discontinued. Renal disease and exercise are associated with decreased fluoride excretion, thus lengthening the time that serum fluoride concentrations remain elevated[15]. However, only one person reported having renal disease, none used nephrotoxic drugs, and there is no reason to suspect that the case patients differed substantially from the control subjects with respect to other factors.

The lowest estimated dose of fluoride that caused symptoms was 0.3 mg per kilogram; 16 percent of the case patients received an estimated dose of less than 1.0 mg per kilogram. The lowest level at which an effect was observed -- a level of less than 1 mg of fluoride per kilogram — is similar to that reported in some studies,[19,23] but lower than that identified in one report[24]. Disordered mineral homeostasis and cellular damage, including abnormalities in serum magnesium, phosphorus, and lactate dehydrogenase concentrations, persisted for at least 19 days. These effects suggest that both follow-up of individual patients and studies of the long-term effects of acute fluoride poisoning may be indicated.

The correlations of the estimated dose of fluoride with the duration of symptoms and with urinary and serum fluoride concentrations imply that our dose estimates are valid. We made several assumptions, however, and the findings must therefore be cautiously interpreted. We relied on interviews conducted with residents four to five days after the outbreak and did not include water consumed in food in our estimate of the fluoride dose. Although the fluoride concentration in the water system was probably not constant, we assumed that the outbreak was caused by water with a fluoride concentration of 150 mg per liter. Finally, most case patients vomited within minutes after ingesting water with a high fluoride concentration, and the dose estimates did not account for the fluoride lost in this way.

We identified two possible causes of the outbreak. The fluoride pump could have been activated without activation of the water pump, or the fluoride concentrate could have been siphoned into the well. In addition, a series of human errors contributed to this incident. The water-system operator had no formal training and lacked a basic understanding of the operation of the fluoridation unit. Fluoride-test results had not been submitted to or monitored by state regulators. When elevated fluoride concentrations were discovered before the outbreak, the recommendation to disconnect the fluoride pump was not implemented.

The findings of our investigation should be of concern both to health care providers of patients with acute fluoride poisoning and to public health and other officials responsible for water fluoridation. The efficacy of fluoridation in preventing dental caries has been well documented, and the safety of this practice is supported by the extreme rarity of incidents of overfluoridation. We believe that the practice of fluoridation of public water systems should continue. However, public health officials must make certain that standard safety equipment is installed, that water-system operators are properly trained, and that routine, systematic monitoring and follow-up of fluoride concentrations in water systems and inspection of fluoridation units are undertaken.

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SOURCE INFORMATION

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Neurotoxicity of Sodium Fluoride in Rats

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MULLENIX, P. J., P. K. DENBESTEN, A. SCHUNIOR AND W. J. KERNAN. *Neurotoxicity of sodium fluoride in rats.* NEUROTOXICOL TERATOL 17(2) 169–177, 1995.—Fluoride (F) is known to affect mineralizing tissues, but effects upon the developing brain have not been previously considered. This study in Sprague–Dawley rats compares behavior, body weight, plasma and brain F levels after sodium fluoride (NaF) exposures during late gestation, at weaning or in adults. For prenatal exposures, dams received injections (SC) of 0.13 mg/kg NaF or saline on gestational days 14–18 or 17–19. Weanlings received drinking water containing 0, 75, 100, or 125 ppm F for 6 or 20 weeks, and 3 month-old adults received water containing 100 ppm F for 6 weeks. Behavior was tested in a computer pattern recognition system that classified acts in a novel environment and quantified act initiations, total times and time structures. Fluoride exposures caused sex- and dose-specific behavioral deficits with a common pattern. Males were most sensitive to prenatal day 17–19 exposure, whereas females were more sensitive to weanling and adult exposures. After fluoride ingestion, the severity of the effect on behavior increased directly with plasma F levels and F concentrations in specific brain regions. Such association is important considering that plasma levels in this rat model (0.059 to 0.640 ppm F) are similar to those reported in humans exposed to high levels of fluoride.

Fluoride Neurotoxicity Central nervous system

DENTAL fluorosis has been on the rise since the 1950s, indicating that our total fluoride exposure is increasing (9). Fluoride, including sodium fluoride (NaF), has been added to public water supplies for over 40 years in the United States as a preventative measure against dental caries. Other sources of fluoride exposure include processed beverages, toothpastes, mouth rinses, dietary supplements, and food. Although dental fluorosis causes discoloration of teeth, it is not considered a public health concern because it does not hinder tooth function or oral health. In addition, no clear link has been established between fluoride and cancer risk, bone fractures, birth defects, or problems of the gastrointestinal, genito-urinary, or respiratory systems (1). Therefore, the impetus to limit total fluoride exposure in the United States is currently based on cosmetic concerns and a general desire not to expose the public to any more fluoride than the amount necessary to prevent dental caries.

One concern that has not been fully investigated is the link between fluoride and effects on the central nervous system (CNS). In vitro studies have shown that intracellular fluoride can alter the kinetic properties of calcium currents in hippocampal neurons (22). Fluoride is a normal component of cerebrospinal fluid (21), but it has not been found to accumulate there during endemic fluorosis or nervous system disease (21,41). Yet, there have been reports from Chinese investigators that high levels of fluoride in drinking water (i.e., 3–11 ppm) affect the nervous system directly without first causing physical deformations from skeletal fluorosis (13,20,40). One study of adult humans found attention affected by sublingual drops containing 100 ppm of sodium fluoride (39), an exposure level potentially relevant to humans because toothpastes contain 1000 to 1500 ppm fluoride (8,48) and mouthrinses contain 230–900 ppm fluoride (48).

Many years of ubiquitous fluoride exposure have not resulted in obvious CNS problems such as seizures, lethargy, salivation, tremors, paralysis, or sensory deficits. Still unexplored, however, is the possibility that fluoride exposure is linked with subtle brain dysfunction. The present study evaluates the neurotoxic potential of sodium fluoride in an animal model. It uses behavioral methodology that focuses on behavioral repertoire, responses to novelty and the temporal or sequential organization of spontaneous behavior, all important

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TABLE 5

CONSISTENT BEHAVIORAL EFFECTS OF FLUORIDE EXPOSURE STARTED AT WEANING

Behavior	Females		Females		Males	
	Control	100 ppm F for 6 Weeks	Control	125 ppm F for 16 Weeks	Control	125 ppm F for 16 Weeks
Sit						
BI (±SE)	20.7 ± 3.0	16.0 ± 1.8	22.4 ± 2.2	15.2 ± 1.8*	57.7 ± 3.3	42.8 ± 3.5†
BTT (±SE)	76.6 ± 19.3	37.9 ± 5.1	66.8 ± 10.9	37.7 ± 5.5*	245.6 ± 21.8	174.4 ± 23.6*
Groom						
BI	13.8 ± 2.6	6.5 ± 1.1*	8.1 ± 2.0	5.2 ± 1.2	30.0 ± 5.5	14.3 ± 2.2*
BTT	29.8 ± 6.3	9.4 ± 1.8†	20.1 ± 6.2	11.4 ± 3.4	70.3 ± 16.1	35.8 ± 8.9
Turn						
BI	123.5 ± 5.5	123.1 ± 5.1	110.7 ± 3.8	105.9 ± 5.5	97.2 ± 5.0	81.7 ± 5.4*
Head turn						
BI	61.9 ± 3.5	57.4 ± 2.9	67.3 ± 2.2	58.6 ± 1.5†	68.1 ± 4.0	58.5 ± 5.0
BTT	75.8 ± 5.3	66.8 ± 3.7	78.8 ± 2.9	69.4 ± 1.9†	84.9 ± 5.5	72.7 ± 7.0
Groom/explore (cluster)						
BI	12.6 ± 3.1	8.5 ± 0.9	15.4 ± 1.9	8.6 ± 1.0†	37.5 ± 3.0	23.7 ± 2.9†
BTT	17.3 ± 4.8	10.7 ± 1.2	19.5 ± 2.4	11.5 ± 1.4†	49.5 ± 4.3	32.5 ± 4.6†
Groom/attention (cluster)						
BI	26.5 ± 4.4	16.1 ± 2.4*	20.4 ± 2.9	12.6 ± 1.8*	72.1 ± 5.2	46.1 ± 4.1‡
BTT	60.7 ± 11.8	43.2 ± 9.0	40.3 ± 8.6	21.5 ± 3.6*	184.9 ± 19.8	131.4 ± 18.7
Groom (cluster)						
BI	10.4 ± 2.5	4.6 ± 0.9*	6.9 ± 1.6	4.3 ± 1.1	22.6 ± 3.8	11.7 ± 2.0*
BTT	22.7 ± 6.2	7.0 ± 1.6*	13.9 ± 3.7	8.2 ± 2.4	42.4 ± 7.8	23.6 ± 4.5*
Stand						
BTT	576.0 ± 22.1	607.9 ± 12.0	608.1 ± 14.9	629.2 ± 17.7	532.5 ± 20.2	599.0 ± 22.0*
Attention (cluster)						
BTT	494.9 ± 19.9	529.5 ± 13.4	505.3 ± 14.8	528.7 ± 20.0	418.4 ± 21.4	499.3 ± 20.9*

*$p < 0.05$, t test; †$p < 0.01$, t test; ‡$p < 0.001$, t test.

distribution and time sequence of behavioral acts were calculated using equations for $K(t)$ as previously reported (27,28,33). The K function was calculated for specific behavioral acts (e.g., sit, rear) or sequences of specific behavioral acts (e.g., sit . . . rear) (33) and for combined acts (e.g., attention or attention/groom) or sequences of combined acts (e.g., attention . . . explore or attention/explore . . . groom/attention) (28). For each of these, a $\Delta K(t)$ [the difference between $K(t)$ for the fluoride animals and matched controls] was calculated for eight time points (2,5,10,20,30,45,100, and 200 s). At any one time point, when K values increase (compared to controls) for a behavior, it means that that particular behavior (or sequence) is "clustering" in time (as seen in hypoactivity), while a decrease means it is "dispersing" in time (it had increased regularity of timing between initiations as seen in hyperactivity). Whenever a behavioral act was initiated less than 10 times on average per animal, control or experimental, $K(t)$ values were not determined for that behavior and related sequences. The bootstrap technique was used for estimating SD at each time point of the K-function for a behavior, and the ad hoc criteria for significance of a difference between control and exposed groups have been described (23,25,27,28,33,34).

An RS statistic was determined for each fluoride treatment. The ad hoc RS statistic distinguishes low level behavioral ef-

TABLE 6

EFFECTS OF 100 ppm FLUORIDE FOR 6 WEEKS STARTING IN 3 MONTH-OLD-RATS

	Body Weight (g ± SD)		Plasma F (ppm ± SD)		Behavior (RS Statistic)
	Control	Exposed	Control	Exposed	
Females	331.8 ± 41.6	319.8 ± 36.1	0.010 ± 0.002	0.077 ± 0.040*	0.200‡
	$n = 21$	$n = 22$	$n = 5$	$n = 5$	$n = 20$ pairs
Males	620.3 ± 45.3	609.0 ± 72.1	0.012 ± 0.005	0.059 ± 0.027†	0.053
	$n = 24$	$n = 22$	$n = 6$	$n = 5$	$n = 18$ pairs

*$p < 0.05$, t test; †$p < 0.01$, t test; ‡$p < 0.001$.

fects from noise (24). This statistic encompasses all data produced in an experiment into one simple statistic. This is an advantage considering that the computer system generates over 100 behavioral measures of three distinctly different types (initiations, total times, and time structures) per experiment. The RS statistic indicates whether behavior is changed overall and the confidence level associated with that change. Statistical significance was set at the $p < 0.01$ level.

RESULTS

Prenatal Exposures

No maternal or offspring toxicity was indicated by reduced body weight in dams during treatment or in their pups soon after birth. Yet, prenatal exposure to sodium fluoride altered behavioral outcome in male offspring when exposure occurred on GDs 17–19 (Table 1). This effect consisted entirely of time structure changes in 11 behaviors and behavioral sequences, 10 of which were significantly dispersed compared to matching controls as illustrated in Fig. 1. These behavioral effects did not coincide with reduced body weight nor elevated plasma fluoride levels at 9 weeks of age (Table 1). At 3 weeks of age, plasma fluoride levels also were not elevated despite prenatal exposure on GD 17–19; plasma fluoride levels were no different in prenatal fluoride females (0.007 ppm ± 0.003 SD; $n = 7$) compared to matched controls (0.006 ppm ± 0.002 SD; $n = 7$) or in prenatal fluoride males (0.004 ppm ± 0.002 SD; $n = 8$) compared to controls (0.004 ppm ± 0.003 SD; $n = 8$).

Weanling Exposures

When fluoride exposures began at 21 days of age, effects on body weight depended on the fluoride concentration in the drinking water (Table 2). Concentrations below 125 ppm did not affect body weight gain at any time during a 5- to 6-week exposure. In contrast, at 125 ppm body weight was reduced throughout 20 weeks of exposure in both sexes. The 11 survivors of a 10-day exposure to 175 ppm F also had stunted growth compared to matched controls at 9 weeks of age. However, by 18 weeks of age, stunting among the 175 ppm female survivors was ameliorated (Table 2).

Plasma fluoride levels were significantly increased in all exposed animals, but again the increase depended upon the fluoride concentration given in the drinking water (Table 3). At 75 and 100 ppm fluoride in drinking water of females for 6 weeks, plasma fluoride levels increased respective of dose. When concentration in the drinking water was 125 ppm for 6 weeks, plasma fluoride levels increased compared to controls but not to levels expected considering results observed at lower drinking water concentrations (Table 3).

Fluoride in drinking water of weanlings altered behavior in both sexes (Table 4). The duration and concentration of exposure determined whether significant effects occurred. In females, a 6-week exposure to 100 or 125 ppm was sufficient to alter behavior, whereas in males an 11-week exposure to 125 ppm in drinking water significantly affected behavior. Too few 175 ppm fluoride females (11 in total) survived after a 10-day exposure to determine an RS statistic for that group. A relationship between behavioral effects and plasma fluoride levels was observed in females exposed for 6 weeks to 75, 100, or 125 ppm fluoride. Figure 2 illustrates that as plasma fluoride levels increased, the RS statistic increased, with significant behavioral impact estimated to occur at a plasma fluoride level of approximately 0.107 ppm. Significant behavioral impact in males, however, did not occur until plasma levels exceeded 0.126 and 0.170 ppm (Table 3 and Table 4).

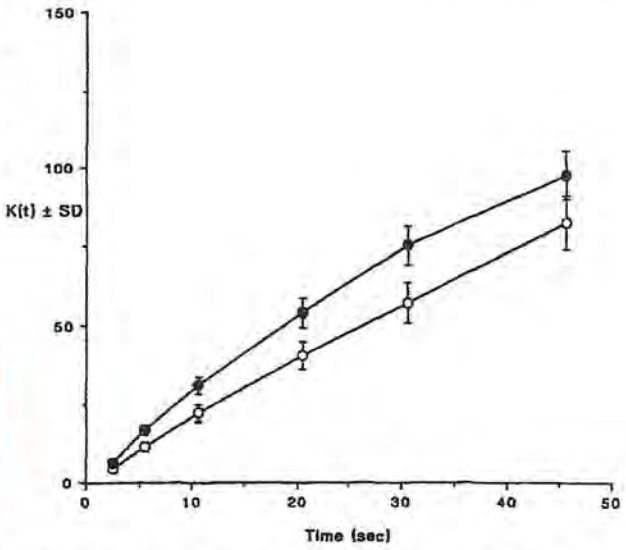

FIG. 4. Exposure to fluoride at the adult stage significantly altered (RS = 0.200; $p < 0.001$) behavior of female rats (●) compared to respective controls (○). This example K function illustrates time-structure changes typical of the adult F effect. Significant clustering (increased K values) is shown for the behavior groom/attention, which prenatal F, in contrast, significantly dispersed (Fig. 1A). Error bars indicate ± SD.

Regardless of sex, duration of exposure, or the fluoride concentration in the drinking water of weanlings, a common pattern among behavioral disturbances developed. Table 5 includes all behaviors that were significantly affected in BI and/or BTT by at least one fluoride exposure. Age and sex influenced the BI and BTT of these behaviors in controls, but still a general effect of fluoride emerged. Whether exposure lasted 6 or 16 weeks, at the 100 or 125 ppm level, in males or females, the same direction of change with respect to controls occurred for a certain array of behaviors and related behavioral clusters. Whereas the act of standing and the related attention cluster tended to increase in total time, the other acts consistently decreased in initiations and total times.

Adult Exposures

Male and female adult rats given 100 ppm fluoride for 6 weeks had significantly increased plasma fluoride levels with no effect on body weight, whereas behavior was affected only in females (Table 6). Compared to females exposed at weaning, females exposed as adults had a lower plasma fluoride level (0.077 ppm) associated with significant behavioral impact. However, the same pattern of BI and BTT changes seen with weanling exposures (Table 5) also developed in females exposed as adults. For example, initiations of sitting, the groom/attention cluster, and the groom/explore cluster in adult female controls (42.9 ± 3.0; 50.4 ± 5.4; 23.9 ± 2.4 SE, respectively) were more frequent than in adult exposed females (30.2 ± 3.0; 34.7 ± 3.7; 15.3 ± 1.8 SE, respectively; $p < 0.01$). Another similarity appeared among BTS effects when adult and weanling exposed rats approached 5 months of age (Fig. 3). Other BTS effects appeared to differ

development. Whether the hippocamus is indeed the brain region most susceptible to fluoride is a possibility deserving consideration in future studies.

Interruption of normal brain development often results in responses that are sex-dependent. The brain responds differently to drugs depending on which hormones are present at the time and whether the brain is male or female (30). In male primates the orbital cortex matures earlier than in females, and such developmental differences are thought responsible for the consequences of perinatal injuries appearing more frequently in males (18). This type of developmental difference might explain why transient peaks of fluoride on prenatal days 17–19 affected males and not females. The effects of chronic fluoride exposures at weanling and adult stages may have involved still other sexual dimorphisms. There are developmentally regulated sexual dimorphisms in hypothalamic somatostatin and growth-hormone-releasing factor signaling, growth hormone secretion and even hepatic metabolism (5,29,38). The sexually dimorphic control of growth would be especially important to fluoride distribution. The rate of fluoride uptake by bone depends on age or the stage of skeletal development; fluoride is deposited in mineralizing new bone more readily than in existing bone (49). As males experience greater and more prolonged growth spurts than females, their plasma fluoride might be directed more to bone than to brain, perhaps explaining why longer exposures and higher plasma fluoride levels were needed in males to affect behavior. Fluoride's tendency to seek developing bone may also explain why adult female rats had behavioral effects at a lower plasma fluoride concentration than did weanling female rats. Levels of fluoride in plasma and bone must be correlated with those in specific brain regions of both sexes to fully understand behavioral consequences.

Rats ingested 75–125 ppm fluoride for weeks to attain plasma fluoride levels of 0.059-0.640 ppm. Six weeks of consuming 75 and 100 ppm fluoride produced higher plasma fluoride levels than did 125 ppm. Perhaps a taste aversion limited water consumption at the 125 ppm level, prolonging the period needed to attain plasma levels that were achieved in 6 weeks by the two lower exposure levels. Regardless, it was fluoride levels in plasma, not fluoride levels of exposure, which best predicted effects on behavior. Similar plasma fluoride levels of 0.076–0.25 ppm have been found in humans ingesting 5–10 ppm fluoride in drinking water (19,37,42), and plasma levels as high as 0.28 to 0.43 ppm have been measured in children drinking water containing 16 ppm fluoride (44). This plasma fluoride range also occurs in certain therapies. Fasting serum fluoride levels of 0.2 to 0.3 ppm are used in the treatment of osteoporosis (31), and plasma fluoride levels as high as 1.44 ppm are found in children 1 h after receiving topical applications of an acidulated phosphate fluoride (1.23%) gel (14,15).

Because humans occasionally are exposed to high amounts of fluoride and plasma levels as high as those found in this rat study, neurotoxic risks deserve further evaluation. This is the first laboratory study to demonstrate that CNS functional output is vulnerable to fluoride, that the effects on behavior depend on the age at exposure and that fluoride accumulates in brain tissues. Experience with other developmental neurotoxicants prompts expectations that changes in behavioral function will be comparable across species, especially humans and rats (16,43). Of course behaviors per se do not extrapolate, but a generic behavioral pattern disruption as found in this rat study can be indicative of a potential for motor dysfunction, IQ deficits and/or learning disabilities in humans. Substances that accumulate in brain tissue potentiate concerns about neurotoxic risks, but the conditions leading to fluoride deposits in any species are still not clear such that quantitative extrapolations are not possible at this time. Thus, conclusions concerning the neurotoxic potential of fluoride require further rat and human studies, both focused on the relationship of plasma fluoride levels with the brain, behavior, and skeletal growth.

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Bottled Water, Fluoride Intake, and Risk of Decay in Young Children

Options

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October 23, 2007

Americans love their bottled water. In 2004, Americans consumed an estimated 26 billion liters of store-bought water, equaling one eight-ounce glass per American each day. Because bottled water tends to be low in fluoride and more Americans now drink less from fluoridated public sources, dentists have wondered whether some kids today might be at greater risk of tooth decay. In the Summer issue of the *Journal of Public Health Dentistry*, NIDCR grantees take a look at this issue using secondary data from the Iowa Fluoride Study, which evaluated fluoride intake dental fluorosis, and bone development in young children. The researchers determined that children who frequently drank bottled water did have significantly lower fluoride intakes than those who did not. However, they found that less than 10 percent of their cohort of 413 children frequently drank bottled water, and "no conclusive evidence of an association with increased caries" was found by age nine. The researchers encouraged further research, particularly because their study was not designed to look specifically at this issue. To read more about this article, please visit PubMed (The authors are Barbara Broffitt, MS; Steven M. Levy, DDS, MPH; John J. Warren, DDS, MS; Joseph E. Cavanaugh, PhD. The title of the article is, "An Investigation of Bottled Water Use and Caries in the Mixed Dentition.")

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* Children who drank bottled water instead of fluoridated tap water had significantly lower fluoride intakes but no associated increase in dental caries (cavities).

J Public Health Dent. 2007 Summer;67(3):151-8.

An investigation of bottled water use and caries in the mixed dentition.

Broffitt B, Levy SM, Warren JJ, Cavanaugh JE.

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2007 University of Iowa College of Dentistry Research Study

Abstract

OBJECTIVES: Bottled water consumption in the United States has greatly increased in the past decade. Because the majority of commercial **bottled water** is low in fluoride, there is the potential for an increase in **dental caries**. In these secondary data analyses, associations between **bottled water** use and **dental caries** were explored.

METHODS: Subjects (n = 413) are in the Iowa Fluoride Study, which included **dental** examinations of the primary (approximately aged 5) and early erupting permanent (approximately aged 9) dentitions by trained dentist examiners. Permanent tooth **caries** and primary second molar increments were related to **bottled water** use using logistic and negative binomial regression models. All models were adjusted for age and the frequency of toothbrushing.

RESULTS: Bottled water use in this cohort was fairly limited (approximately 10 percent). While **bottled water** users had significantly lower fluoride intakes, especially fluoride from **water**, there were no significant differences found in either permanent tooth **caries** (P = 0.20 and 0.91 for prevalence and D(2+)FS, respectively) or primary second molar **caries** (P = 0.94 and 0.74 for incidence and d(2+)fs increment, respectively). Results for smooth surfaces differed somewhat from those for pit and fissure surfaces, but neither showed significant differences related to **bottled water** use. *

CONCLUSION: While **bottled water** users had significantly lower fluoride intakes, this study found no conclusive evidence of an association with increased **caries**. Further study is warranted, preferably using studies designed specifically to address this research question. *

PMID: 17899900 [PubMed - indexed for MEDLINE]

FLUORIDE ACTION NETWORK HEALTH EFFECTS

HEALTH EFFECTS HOMEPAGE

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HEALTH EFFECTS: Dr. William Marcus' Internal Memo

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON. D.C. 20460

OFFICE OF WATER

MEMORANDUM

DATE: May 1, 1990
SUBJECT: Fluoride Conference to Review the NTP Draft Fluoride Report
FROM: Wm L. Marcus, Ph.D., Senior Science Advisor, Criteria & Standards Division, ODW (WH-550D)
TO: Alan B. Hais, Acting Director, Criteria & Standards Division, ODW (WH-550D)

The conference was held in RTP at the NIEHS headquarters on April 26, 1990. The subject of the conference was a peer review of the NTP draft report on the toxicology and carcinogenesis studies of Sodium Fluoride in F344/N Rats and B6C3F Mice (Drinking Water Studies) NTP Report Number 393. Dr. Robert Scala was to chair this meeting but was unable to attend because of ill health. Dr. Michael Gallo was appointed acting Chairperson. One of the attenders seated with the panel members was David Rall, Ph.D., M.D., Director of NIEHS. Dr. Rall took an extremely active interest in the proceedings and remained seated for the entire proceedings with only two minor interruptions.

The most disturbing part of the report was the continual reference to the historical controls as having the same or higher cancers as the test groups. On pages 89 - 90 of the report starting with the last paragraph the authors state the following:

An important consideration which limits the usefulness of the historical control data base in the current studies is that the diet used in all other NTP studies had not been closely monitored for fluoride content. Fluoride concentrations in typical batches of NHI-07 diet range between 28 and 47 ppm (.7 and 1.2 mg/kg/day)(Rao and Knapka (1), 1987). Assuming a minimum bioavailability of 60% (Tests show 66% absorption page I-18), the historical database animals actually constitute a group receiving sufficient fluoride to place them between the low- and mid-concentration group in the current (the studies reviewed at RTP at the conference). The fact that this fluoride is available for absorption from the standard diet is supported by the levels of fluoride found in the bones of animals maintained on this diet in the six months studies (Appendix I). (The levels in the bones of the rats on the standard NHI chow was ten [10] times the levels of those fed the semisynthetic diet and deionized water, 0.922 vs 0.0901). If the fluoride [is] in fact influencing the "spontaneous " or background incidence of osteosarcoma in male rats, comparisons with those in the historical database maybe misleading. This forces an even greater reliance on the within-study comparisons, ie., the incidences of the dosed groups compared with the concurrent control, in the interpretation of the results of the sodium fluoride studies.

When I plotted a bar graph of osteosarcoma in male rats and placed the historical controls on the graph 0.6% is just where expected. This helps demonstrate a relationship between osteosarcoma and fluoride. The purpose of such graphs is to predict occurrence. Since the historical controls comprise some 6,000 animals, this data point is extremely significant compared to the other three. Osteosarcoma is an extremely rare animal tumor and may be the result of the variable high fluoride content in the feed. In order to demonstrate this, all that need be done is require that the fluoride content of animal chow be lowered dramatically and that fluoride be removed from the water given to the animals under study.

The dose of fluoride to which the concurrent controls were exposed is 0.2 mg/kg/day. A 70 kg man who drinks 2 liters daily is exposed to 0.03 mg/kg/day. The "control" animals were exposed to an amount of fluoride six to seven (6-7 X) greater. Lois Gold, Ph.D. of the review panel concluded that, "this group of animals therefore, can hardly be termed a control group. It can best be described as a lowest dosed group." This is an important consideration because as the document reports on page 9, the levels of fluoride in bone are linearly dependent upon dose and length of exposure ("depends upon total intake") in people. The level of fluoride in

performed to determine the carcinogenicity of fluoride this should not have been addressed. There appear to be at least four different publications from the U.S., Canada, and New Zealand that have reported similar or lower tooth decay rates in nonfluoridated areas as compared to fluoridated areas (4,5,6,7). Therefore, the entire question of the efficacy of fluoridation based on extensive and multiple studies has been called into question. Our job is to set safe levels for fluoride in drinking water based on the scientific evidence.

The problem with this meeting was the inability of independent reviewers to get to see the slides prior to the meeting. We must perform our own scientific review of the slides and write our conclusions for use in the development of the revised fluoride regulation.

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NOTE: Due to his criticisms of the tumor downgradings, Dr. Marcus was **fired** by the EPA. The US Secretary of Labor, Robert Reich, later ruled that EPA fired Marcus out of "retaliation" for Marcus' stance on fluoride, and ordered EPA to reinstate Marcus with full back pay and compensation. To learn more about EPA's firing of Marcus, see:

- Reich Orders EPA to Reinstate Scientist - *National Whistleblower Center February 10, 1994*
- Scientist Who Spoke Out on Fluoride Ordered Reinstated to Job - *The Associated Press February 11, 1994*
- EPA Ordered to Reinstate Whistleblower - *The Associated Press December 18, 1992*

Fluoride Action Network | 802-338-5577 | health@fluoridealert.org

LOW-LEVEL FLUORIDATION AND LOW-LEVEL RADIATION
TWO CASE HISTORIES OF MISCONDUCT IN SCIENCE

by Albert Schatz, Ph.D, ©1996 , Philadelphia, Pennsylvania

Abstract

Poor, malnourished children, especially infants, are the most sensitive barometer of fluoride toxicity. Low- level fluoridation (fluoridation of drinking water) and low-level radiation are similar in many respects. Paradoxical effects of fluoride must be considered in determining harmful effects of both low-level fluoridation and low-level radiation. It is not surprising that low-level fluoridation is associated with paradoxical effects. It would be surprising if it were not. The occurrence of paradoxical effects with low- level fluoridation and low-level radiation shows that there is no threshold level below which fluoride and radiation are harmless. My article in Appendix C shows that fluoridation does not prevent dental caries. Iodination of drinking water was discontinued in the 1920s becaue it was harmful and did not prevent goiter. Fluoridation of drinking water should be discontinued now because it is harmful and does not prevent dental caries.

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Dedication

This report is dedicated to Salvador Allende, M.D., for the same reasons1 that my first report, *Increasing Death Rates in Chile Associated with Artificial Fluoridation of Drinking Water, with Implications for Other Countries*, was dedicated to him. Dr. Allende was elected President of Chile on September 4, 1970, and was assassinated by a military junta on September 11, 1973. As a result of health measures he instituted, the birthweight of babies increased for the first time in 50 years. Dr. Allende, as a doctor and as President, was concerned about the poor people of Chile. These are the people in all countries who, because they are malnourished, are most susceptible to the harmful effects of fluoridation.

My research on the harmful effects of fluoridation in Chile was the first study specifically concerned with poor, malnourished people. I chose Chile for my research because (a) the majority of the population of Chile is poor and malnourished, and (b) pro-fluoridationists have never adequately studied the effect of fluoridation on poor, malnourished people as a specific group of the total population.

In the United States and other developed countries, poor and malnourished people are a minority that is statistically drowned out in the total population which is researched for harmful effects of fluoridation. The increased death rates associated with fluoridation in Chile tell us that fluoridation is also killing poor, malnourished people in all countries that have fluoridation.

Prologue

Opponents of fluoridation have for decades pointed out numerous errors, omissions, and inaccuracies in what proponents of

fluoridation have published. This present report continues that criticism. However, it is qualitatively different because (a) it reports the widespread occurrence of paradoxical effects and emphasizes the importance of paradoxical effects in evaluating the safety of fluoridation, (b) it points out similarities between fluoridation, which I call low-level fluoridation, and low-level radiation, and (c) it highlights the well-earned position of fluoridation as a well-documented case history in the well-recognized category of scientific misconduct.

Fluoride is now recognized as a legally sanctioned environmental pollutant2 that is part of an increasing number of chemicals which are threatening our survival as a species. Fortunately, however, "more and more scientists insist that they are in a better position to understand the significance and implications" of science "for society than are the official decision-makers who may be paying their salaries or subsidizing their work. The point is that the real division is no longer between science and the humanities - the two cultures described by scientist-philosopher C. P. Snow - but between those who attach primary importance to human life and those who view their own discipline as sovereign."3

We ... see ... the difficulty that may arise in the attempt to persuade others to accept a new ... way of reasoning. We cannot convince others of it by formal argument for, so long as we argue within their framework, we can never induce them to abandon it. Demonstration must be supplemented, therefore, by forms of persuasion which can induce a conversion. The refusal to enter the opponent's way of arguing must be justified by making it appear altogether unreasonable.— Michael Polanyi, physical chemist

Mitchell, Thompson and Borman's research shows that fluoridation is harmful

Mitchell *et al* 's report *No Association between Fluoridation of Water Supplies and Sudden Infant Death Syndrome*,4 unjustifiably exonerated fluoride as a cause of sudden infant death syndrome (SIDS). The authors interpreted their data correctly, but then, by a verbal legerdemain shell game, drew the erroneous conclusion which their title reflects. The very fact that the straight line in their Figure 1 is not horizontal suggests an association between fluoride and SIDS.

What Mitchell et al's data clearly indicate is an inverse relationship between fluoride concentration in the drinking water and SIDS. Their interpretation of their results also clearly recognize this inverse relationship. "If anything" they wrote, "the higher the SIDS mortality rate the lower the fluoride concentration." However, in their very next sentence, they contradicted that interpretation by concluding that their "study clearly shows ... no indication of a relationship between fluoridation ... and SIDS".

According to Mitchell *et al*, "Analysis was carried out to find the correlation between variables and then simple linear regression was used".4 Their arbitrary selection of linear regression provided a straight line which is very different from the curve of a paradoxical effect (which I shall define and discuss). The typical paradoxical effect curve that I have inserted in their Figure 1, reproduced below, shows the increase in deaths as the fluoride concentration decreases in the very low concentration range. Dr. Albert W. Burgstahler, in the University of Kansas Department of Chemistry, kindly provided the paradoxical effect curve. This was derived by a computer-generated least squares best line fit.

Mitchell *et al* apparently did not know that the inverse relationship, which they acknowledged, suggests a paradoxical effect.5,6 This phenomenon is paradoxical because there is a critical dose below which one observes an increased adverse effect. This paradoxical effect is precisely what Mitchell *et al 's* data clearly reveal. Within that low concentration range, the infant death rate increased as the concentration of fluoride decreased. Consequently, there is a relationship between fluoride concentration and infant deaths, but it is an inverse relationship.

At concentrations above the low concentration range, dose-responses show the direct, linear relationship with which we are familiar. Increasing doses are increasingly toxic until a particular dose kills most or all the test subjects. This is what one expects and observes. Consequently, there are two different dose- responses to fluoride — a paradoxical effect at very low levels and a linear relationship at higher levels. These two dose-responses are not mutually exclusive; one does not preclude the other.

The paradoxical effect curve explains what the data of Mitchell *et al* clearly reveal and what they clearly acknowledge; namely that there are more deaths at the low doses of fluoride than at the higher doses. The straight line which they derived from their linear regression analysis does not explain the increased deaths at the lower doses. Mitchell *et al* and all other researchers who are interested in knowing whether fluoridation produces harmful effects should familiarize themselves with paradoxical effects5,6 and understand that they "cannot exclude" an "explanation" they "have not considered".7

Paradoxical effects show that fluoridation is not safe at any level

There many reasons why fluoridation has been highly controversial since its conception half a century ago. We can now add another reason — ignorance of paradoxical effects. If Mitchell *et al* had been familiar with paradoxical effects, Occam's razor would have directed them to a paradoxical effect. The cluster of infant deaths at very low fluoride levels suggests that the relationship between fluoride concentration and SIDS is paradoxical, not linear.

Most research which purports to demonstrate the safety of fluoridation has not been concerned with very low concentrations of fluoride, at which paradoxical effects occur, for three reasons. (1) Individuals vary significantly in fluoride uptake. (2) There is considerable individual variation. (3) It has been unjustifiably assumed that there is a threshold level; namely, the sacrosanct one

part per million in drinking water, below which fluoridation is safe. The occurrence of paradoxical effects at very low levels of fluoride means that there is no threshold level below which low-level fluoridation is safe. There is also no threshold level below which low-level radiation is safe. We call fluoridation low-level fluoridation because that term associates it with the well-known low-level radiation. Both are harmful and similar in other ways.

The paradoxical effect in the report by Mitchell *et al* is similar to what has been observed in a variety of fluoride systems where paradoxical effects occur.5 Paradoxical effects may be involved in SIDS in some fluoridated cities in Australia."8 The data in the Australian report, which claimed that there was no linear relationship between fluoridation and SIDS, should be reexamined to see if there are inverse (paradoxical) relationships.

Paradoxical effects occur in complex systems where they are influenced by conditions that vary from time to time within the same system.6 For these reasons, paradoxical effects may not always appear in a very low concentration range. But a linear dose-response always appears at higher concentrations which, in the case of fluoride, begin at the arbitrarily defined safe level of one part per million. Therefore, the relationship between fluoride concentration and SIDS may be linear and/or paradoxical, depending on the concentration range of fluoride and other variables. This variability and, at times, even irreproducibility of results are characteristic of paradoxical effects.6

Statistics can conceal paradoxical effects

Because Mitchell *et al* used linear regression, the only conclusion they could draw from their straight line is that there is no linear relationship between fluoride concentration and SIDS. But they unjustifiably concluded that there is no relationship with fluoride at all. The fact that their statistics reveal no significant linear correlation does not mean that there is no other significant correlation. Their linear regression analysis may be mathematically correct, but it is inappropriate to ascertain whether there is another kind of relationship; namely, a paradoxical effect. Like the Sufi Mulla Nasrudin, they looked for the key in the wrong place.

There is "a famous Sufi story" about "Mulla Nasrudin, an enlightened fabled teacher. While on his hands and knees, peering on the street for a lost key, he was approached by a friend. 'You lost your key here, Mulla?' his friend inquired. 'No,' said Nasrudin, 'I lost it in my house.' 'Then why are you looking here?' asked his friend. 'Because,' said Nasrudin, 'the light is better here.'" — Larry Dossey

It is paradoxical that statistics, employed to assure the validity of conclusions drawn from data, can be responsible for concealing paradoxical effects." 6 "The fact that statistical analysis of experimental data does not reveal paradoxical effects does not mean that such phenomena do not exist. On the contrary, statistical methods of analysis can effectively prevent recognition of paradoxical effects if the methods do not consider these phenomena. With scattered points, statistical methods are too frequently used to determine where a straight line should be drawn. To often, the statistical approach assumes that straight lines are the correct lines. Deviations or irregularities caused by paradoxical effects have too often been attributed to experimental variation or errors.6 More information about paradoxical effects and risk assessment is in Appendix B.

The pig mentality

"If artificial fluoridation causes deaths among individuals who are for one reason or another more sensitive to fluoride toxicity than in the total population taken as a whole, then the controversy over whether fluoridation does or does not reduce caries becomes purely academic because it is criminal to implement a so-called public health measure which kills certain people even if it does reduce tooth decay in some of the survivors."1

We have been told that fluoridation is economical because it lowers the cost of dental care. But, even if that were true, "let us get our priorities right. If it is economic to poison people, then there must be something wrong with economics." 9

With respect to economics, let us look at fluoride and what I call the *pig mentality*. In 1952, seven years after fluoridation was started, some U.S. government agencies were concerned about possible harmful effects of fluoride. But they were more concerned with safeguarding pregnant pigs than pregnant women and their unborn children. Their reason for giving priority to the welfare of pregnant pigs was that pregnant pigs were economically more important than pregnant women.

The following testimony, which reports this shocking revelation, is taken from a U.S. Congressional investigation held in 1952.10

Dr. Miller: The United States Department of Agriculture made some examination as to what happened in brood sows. They recommended to the farmers that fluoride not be added to the water or feed of brood sows because it did something to pigs that were unborn.

Dr. Porterfield: Yes.

Dr. Miller: Do you think it might be wise for the Public Health Service or some group of people to inquire what might happen to pregnant women and the unborn child when they are given fluoride? Do you think it is necessary to complete the examinations that have already been started on that subject?

Dr. Porterfield: I do not think there is enough money, sir, from the Federal Government or any other source, to pursue all of the possible hypotheses that may be proposed for most of our programs. We have to screen them from the point of view of greatest probability, and since we can find no cause from the physicians or the dentists or the investigating scientist pointing to this, it would seem to us not of a high priority to devote money for something that has shown no suggestive indications.

Dr. Miller: Would you say that the agricultural Department went off on a tangent then when they investigated what might happen to pigs or brood sows?

Dr. Porterfield: No, sir, I think there is a difference.

Dr. Miller: It is alright to do it with pigs, but you do not want to do it with women. Is that the attitude you take?

Dr. Porterfield: They have different objectives in mind, sir. There is more money available for matters that have economic value than there is for health.

Have the fluoridationists, like Dr. Faustus, no understanding of "the morality of knowledge" (Erich Heller) and no "insight into ultimate meanings"? (Victor Lange) Did they, like Faustus, "outwit the Devil by creating a Hell of" their "own"? (Victor Lange11) No wonder fluoridation calls to mind Albert Einstein's lament: "Strange that science, which in the old days seemed harmless, should have evolved into a nightmare that causes everyone to tremble." And Linus Pauling's comment: "Most problems in the modern world are the result of the contributions of science."

More criticism of Mitchell, Thompson and Borman's research

There are additional reasons why the research of Mitchell *et al* has "been weighed and found wanting". The variables they considered were SIDS, mean daily temperature and median fluoridation. The validity of their research is questionable because they did not consider other important variables. Walker pointed out that Mitchell *et al* did not consider a significant intake of fluoride from sources other than fluoridated water.8 The National Health Council and the Medical Research Council of Australia reported that babies were being overdosed with fluoride from many sources, especially formula foods prepared with fluoridated water.8 It would be surprising if that overdosing did not also occur in New Zealand where Mitchell *et al* did their research. Mitchell *et al* provided no information at all about "background fluoride"; that is, fluoride from other sources; e.g., air, food, tea (dry tea leaves are high in fluoride) and baby formulas prepared with fluoridated water. The terms background fluoridation and background radiation apply to fluoride and radiation from sources that are not always taken into consideration.

Mitchell *et al* (like Nelson and Taylor whom they cite) found a correlation between mean daily temperature and SIDS. People generally drink more liquids in warmer weather. One would therefore like to know, for example, whether the dead infants had been nursed and how much water and tea their mothers consumed. Tea is notoriously high in fluoride. But Mitchell *et al* did not estimate each infant's total daily fluoride intake. The correlation of infant deaths with mean daily temperature suggests that more infants may die in hotter weather when mothers and infants consume more tea and other fluids and therefore more fluoride. Finally, Mitchell *et al* and all proponents of fluoridation apparently also do not understand why it is inappropriate to apply the results of random testing to an individual.12

Another variable overlooked by Mitchell *et al* is nutrition, especially calcium intake. The consumption of milk, which is the major source of calcium for infants, is especially important. Our research on fluoridation in Chile (like many other studies) points out that malnourished infants comprise the human population that is most susceptible to fluoride toxicity. It is also well known that calcium protects against fluoride. When Salvador Allende, M.D. became president of Chile in 1970, he initiated a government program under which "pints of ... free ... milk were delivered ... daily ... to pregnant mothers, nursing mothers and every child under the age of 15." 1 At that time, "half the children in Chile under 15 years were undernourished, and 600,000 were mentally retarded thorough lack of protein, especially during the first months of life." 1 This was the health status of half the children who were being fluoridated in Chile.

I am not implying that malnutrition and/or calcium deficiency were significant factors in SIDS researched by Mitchell *et al*. No one knows because Mitchell *et al* did not provide that and other information. To properly evaluate the role of fluoride in SIDS, it is necessary to consider nutrition, especially calcium intake, and total fluoride intake.

Promoters of fluoridation are in Plato's cave

"Plato's famous parable of the cave describes a group of people who are chained inside a cave in such a way that they can see only the shadows on the wall of the cave. These shadows are the only world that these people know. One day one of them escapes into the world outside the cave. At first he is blinded by the sunlight, but when he recovers, he realizes that this is the real world, and what he previously considered to be the real world was, in fact, only the projection of the real world onto the wall of the cave. Unfortunately, when he returned to the people who were still chained inside the cave, they thought he was mad." — G. Zukav

Those who are for and against fluoridation have little in common other than the issues they disagree on. They cannot dialogue objectively because they have different realities. They see things differently, and have different criteria for determining validity. Our lives are enriched by artists for whom "beauty is in the eyes of the beholder." But our lives are often endangered when scientific

truth is in the eyes of the beholder.

"There's something rotten in Denmark"

Mitchell, Thompson and Borman's publication4 is a classic, textbook case history of the pathology of (i.e., what is wrong with) epidemiological surveys which have concluded that fluoridation is safe. Such studies, are invalid for the following reasons.

1. Researchers assumed that there is only one dose-response to fluoride and that dose-response is linear. They do not know that there are two distinctly different dose-responses which occur within two different concentration ranges of fluoride. The dose-response which they completely overlooked is the paradoxical, non-linear dose-response. This occurs only with low-level fluoridation, just as it also occurs only with low- level radiation,13,14 where it is described as supra-linear, quadratic and non-linear. (I shall shortly discuss low-level radiation and what it has in common with low-level fluoridation.) The reasons why paradoxical effects occur at low levels of fluoride and other chemicals,5 and why supra-linear dose-responses occur at low levels of radiation are beyond the scope of this report, but have been explained elsewhere.5,6,13,14,15

2. Researchers did not selectively study minority populations, such as infants, that are most susceptible to fluoride toxicity. But it is not enough to study infants. One must select infants who are malnourished and whose mothers received little or no prenatal care. Otherwise, as I discussed in detail in my report on deaths association with fluoridation in Chile, "the relatively well-nourished majority overwhelms ... the undernourished minority which is most susceptible to fluoride toxicity." 1

The importance of selecting a minority population of infants that are malnourished and disadvantaged in other ways is clearly revealed by the shockingly high variation of infant deaths in different sections of Philadelphia. This variation is directly related to poor nutrition, inadequate prenatal care and other factors.16 No one knows how many poor, malnourished children in Philadelphia, which is fluoridated, are being killed or otherwise harmed by fluoridation?

Spittle recognized the importance of working with a malnourished minority,17 which I had pointed out in my Chile report. Both Brown18 and the same Mitchell,19 who criticized my Chile report, did not recognize (1) the above-mentioned inadequacies in the report of Mitchell *et al*, and (2) the importance of focusing research a malnourished minority.

The 1994 report by the New Zealand Public Health Commission,20 which endorsed fluoridation, accepted without question the erroneous conclusion of the report by Mitchell *et al,*4 and Brown18 and Mitchell's19 unjustified criticism of my report on fluoridation in Chile. The New Zealand report referred to my article in its text, but did not cite it in its bibliography. The results of Mitchell *et al* which, despite their inadequacies, show that fluoridation is harmful as very low levels, are all the more significant because the infants they researched were not specifically selected as a malnourished population. Despite that limitation, their results support my report of increased deaths associated with fluoridation in Chile.

This New Zealand Public Health Commission report has as little scientific validity as a British report on the effectiveness of fluoridation drafted by a special Committee on Research into Fluoridation. This is Report No. 122 published by the British Department of Public Health and Social Security in London. I challenge Brown18 and Mitchell,19 who criticized my report on fluoridation in Chile, to publish a criticism of my analysis21 (See Appendix C) of that British report. This British report has to be seen to be believed. This and many other reports that support fluoridation reveal incompetence, bias, and arrogance, and are examples of pseudoscience. One does not know whether to accuse the authors of misconduct in science or conclude that they just don't know any better.

Those who do research in Plato's cave do not realize that: (1) All they see are only shadows on the wall. (2) They "can fool some of the people all of the time, and all of the people some of the time, but" they "cannot fool all of the people all of the time." (3) "No man was ever so much deceived by another as by himself. (Greville) and (4) "The learned fool writes his nonsense in better language than the unlearned, but still 'tis nonsense." (Benjamin Franklin)

Low-level fluoridation and low-level radiation are both harmful

"New scientific truth does not triumph by convincing its opponents and making them see the light, but rather because its opponents eventually die, and a generation grows up that is familiar with it." Max Planck, the father of quantum physics

The history of progress is the history of controversy. The antifluoridation movement has made both history and progress. The report you are now reading contributes to both. Its objective is to provide new information about the nature of the sand upon which the house of fluoridation has been built. It points out the importance of paradoxical effects which have not received adequate attention with respect to fluoridation. These effects occur at low levels of fluoride and radiation. We call fluoridation low-level fluoridation because that term associates it with the well-known low-level radiation. Both are harmful and similar in other ways.

For low-level radiation, we now know that "linearity underestimates the true cancer risk per rad when one derives values from studies based on higher doses of radiation than the" low "doses at which we wish to apply those values." 13 We now know that linearity also underestimates the risk of low-level fluoridation. This is why we criticize those who have disregarded paradoxical effects associated with low-level fluoridation.

Low-level fluoridation, like low-level radiation, may produce adverse effects that appear after prolonged periods of time. For this reason, a cause and effect relationship is not always clear. The adverse effects of large doses of fluoride and radiation, with which this report is not concerned, occur sooner after exposure and are therefore clearly associated with the cause.

Low-level fluoridation and low-level radiation have both been controversial for decades. Both have used human beings as guinea pigs. They have killed and continue to kill untold numbers of people. There is no threshold level below which fluoride and radiation are harmless. Background radiation, such as radon gas, is harmful; and background fluoride (from sources other than drinking water; i.e., air, food and tea) is harmful. Topical application of fluoride and topical application of radiation (e.g., for skin cancer) are harmful. The dose-response to both low-level fluoridation and low-level radiation is not linear but paradoxical.

Low-level fluoridation, like low-level radiation, is a major pollutant which environmentalists should recognize as such. Like other pollutants, the harmful effects of both low-level fluoridation and low-level radiation have a history of denial which is associated with scientific, professional and academic misconduct. Tolerance levels for low-level fluoridation and low-level radiation are not based on scientific findings. Instead they are determined by special interest groups which have enough money to buy enough political clout to influence government and other high-level agencies.2 "The wages of sin are death." But those who die from low-level fluoridation and low-level radiation are not the sinners, but their victims.

Supra-linear (paradoxical) effects of low-level radiation

Paradoxical effects may not make sense to those who are unfamiliar with these peculiar dose-responses. Nevertheless, these phenomena, disquieting as they may be, are irrefutable. A wide variety of paradoxical effects has been reported with fluoride5 and many other chemical substances.6 In radiation, the paradoxical effect is known as a non-linear, quadratic and supra-linear dose-response.13,14 It is also called the Petkau effect.15 All these peculiar phenomena, associated with both chemicals and radiation, are characterized by a low concentration range within which the adverse effect increases as the dose decreases.

Gould and Goldman, in their book *Deadly Deceit. Low-Level Radiation, High-Level Cover-Up*,15 report the results of Charles Walden *et al* who observed a supra-linear effect of ionizing radiation on human chromosomes. "Their findings contradict the conventional scientific dogma that the dose- response is linear, and that a straight line can be used to estimate low-dose effects from studies of high doses." Gould and Goldman also discuss the "Petkau effect." In 1971, Abram Petkau, a physician and biochemist, observed an unusual and entirely unexpected effect of radiation. He found that low levels of radiation produced more damage to fresh beef brain cellular membranes than higher doses did.

Gofman pointed out, in his book *Radiation and Human Health,*13 that "Enthusiasts of nuclear power and of medical irradiation are forever hoping, quite understandably, that there will be found some threshold — a dose of radiation below which no harm would occur." But "It turns out that nuclear-power and medical-irradiation enthusiasts have all been going in exactly the wrong direction, They have consistently suggested that linearity may overestimate the true cancer risk per rad. The real problem is that linearity underestimates the true cancer risk per rad when one derives values from studies based on higher doses of radiation than the doses at which we wish to apply those values."

According to Gofman and O'Connor (in their book *X-Rays: Health Effects of Common Exams*), "It is natural for everyone, ourselves included, to wish that radiation would be less harmful per rad at low dose-ranges than at high dose-ranges... Those who cling to this wish, in spite of all the evidence, claim that the linear 'hypothesis' exaggerates the risk of getting cancer from irradiation at low doses. But wishful thinking is gradually yielding to evidence." 14

The books by Gofman13 and Gofman and O'Connor14 are replete with reports which prove that low doses of radiation are in many cases more harmful than higher doses. These data fit what is called a supra-linear dose response curve, which is significantly different from a linear curve. Gofman and O'Connor14 conclude that the "linear model may actually underestimate the risk of getting cancer and leukemia. There is, unfortunately, evidence which is accumulating and growing ever stronger that the cancer risk per rad of dose is worse in the low-dose range than in the high dose... Moreover, during the nearly four years of extraordinary scrutiny and widespread peer review of the book13 in professional journals, scientific symposia and in trials concerning radiation injury, no one has made a single scientifically valid refutation of any of its data, methods, or conclusions. Probably no work in this field has received more review by peers." 14

Who is John W. Gofman?

Because it is so difficult for some to accept the unusual supra-linear effects, let us familiarize ourselves with the credentials of the individual who has done decades of research on these phenomena. John W. Gofman is a physician with a doctorate in nuclear/physical chemistry, who is recognized as one of the world's leading medical experts on low-level radiation. He has been Professor of Medical Physics at the University of California in Berkeley, and a member of the Clinical Faculty at the University of California School of Medicine in San Francisco.

As a graduate student at Berkeley, he was one of those who discovered uranium-233, and demonstrated that it was fissionable. In 1941-1943, he developed a method for isolating plutonium and provided the plutonium first used at the Manhattan Project. From 1962-1969, he was Associate Director of the Lawrence Livermore Laboratory, and set up the Laboratory's Biomedical Division. From 1963-1972, he directed research in that Division. This research, requested by the Atomic Energy Commission, evaluated

ionizing radiation and chromosome injury as causes of human cancer. Gofman has received many awards and honors, including a citation from the American College of Cardiology. He has written six books.

Fluoridation is iatrogenic and pathological science

Fluoridation is iatrogenic science because it creates more serious problems than the problem it was originally designed to resolve, but did not. Fluoridation also conforms to what Irving Langmuir, the 1932 Nobel laureate in chemistry, called the dynamics of "pathological science — the science of things that aren't so. The basic progression is that sloppy work by sloppy scientists gets picked up by even worse scientists, who do worse and worse work... But the kicker is that this kind of bad science may not completely die for a very long time, but just take on more and more peculiar forms." 22

One of these forms was pointed out by Sutton in his cogent article *Are Most Fluoridation Promoters Neurotics?*23 Sutton quoted Kurt Thoma, Professor of Oral Pathology at Harvard University: "The neurotic depends on opinions other than his own and is swayed by remarks of others without analyzing the facts. He feels that his opinions must be enforced, and even if proven in error he will not 'give in' because this hurts his ego ideal."

This is why the pathology of fluoridation is so easily diagnosed. Its distinctive syndrome consists of hype, brouhaha, hullabaloo, echolalia, pleonasm, tautology, propaganda, weasel words, propaganda, recycled nonsense, double speak,24 the invention of reality, 25 denial, the graying of reality and the manufacture of consent,26 Fluoridation is part of "the new age mythology"27 in which, as Werner Erhard proclaimed, "Reality is make-believe." Wishful thinking is presented as scientifically established fact to achieve self-serving ends. An egregious example of the pathology of fluoridation is Report No 122, compiled by a special Committee on Research into Fluoridation and published by the British Department of Public Health and Social Security in London. See Appendix C for my analysis21 of this British report, which erroneiously concludes that fluoridation is effective in preventing tooth decay.

The denial syndrome

Unfortunately, those who question fluoridation are ignored as if they did not exist, or are attacked and derogated by foxes who are guarding the fluoridation chicken coop. A recent example of this is the hit- and-run attempt to discredit John Yiamouyiannis. This is discussed in the section "Truth will come to light. Murder cannot be hid."

A clinical psychologist whom I consulted about fluoridation referred me to the psychiatric definition of *denial* which includes "refusal to admit ... reality... Known also as negation, denial is a primitive defense ... consisting of an attempt to disavow the existence of unpleasant reality.28

Denial is not isolated and sporadic, but may be pandemic in science, medicine, academia, and elsewhere. It occurs in the controversial fluoridation of drinking water which, contrary to what authorities claim, does not prevent dental caries and is not safe.1,23 In quantum physics, physicists have for decades refused to acknowledge an inconsistency in Einstein's special theory of relativity.29 They also have been slow to recognize Bohm's alternative.30 Quantum theory is itself paradoxical because it "demands conflicting or mutually incompatible descriptions — an example of this is the description of an electron as both a wave and a particle." A well-known scientist wrote an unfavorable review of one of Velikovsky's books without (she admitted) having read it. Other well-known scientists who edited prestigious scientific journals rejected papers, which Velikovsky submitted for publication, without (they admitted) having read them.31

Hendershot, editor of the *Journal of the American Dental Association,* provided another example of denial. I wrote Hendershot to ask if he would be interested in seeing my report on harmful effects of fluoridation in Chile.1 When he did not reply, I sent him copies of my report on three separate occasions, one month after another. He rejected my report three times without ever having even seen it. To publicize his censorship, my report, which then appeared in the *Journal of Arts, Science and Humanities*, included a photograph of three envelopes, each of which contained a copy of my manuscript and was addressed to Hendershot. These letters were certified so that the recipient had to sign a receipt of delivery. All three envelopes were stamped **REFUSED - RETURN TO SENDER.**

Misconduct in science

Misconduct and denial are two sides of the same coin because denial is a form of misconduct and misconduct often involves denial. Fluoridation is an example par excellence of denial and misconduct in science but it is in good company, whatever that means. The 1992 report on *Responsible Science. Ensuring the Integrity of the Research Project*, Volume I (by the National Academy of Sciences, the National Academy of Engineering and the Institute of Medicine)32 received a "cool response" and "can be credited with adding silt to muddy waters".33

Some areas of higher education are also hotbeds of misconduct by academic robber barons in administration, research, and teaching.34,35,36 The May/June, 1994, Special Issue of *The Journal of Higher Education* was devoted to "Perspectives on Research Misconduct".35 In some colleges and universities, plagiarism, falsification of data, and other kinds of individual and institutional misconduct appear to be pandemic.30 Much of this pollution of the ethical environment of research and graduate education is covered up by structured silence.36 Structured silence denies misconduct by ignoring it. Unanswered questions are ignored, as if they did not exist. Denial preserves the illusion that all is well, and perpetuates false public images of academia and science. Brown18 and Mitchell19 used structured silence when they selectively criticized my data but not Briner and Carmona's data,

included in my report.1

The most recent and most comprehensive report36 of misconduct in colleges, universities and science was rejected for publication by Koshland, the editor of *Science*. This is a prestigious journal published by the American Association for the Advancement of Science. Koshland had previously claimed that 99.9999% of scientific reports are accurate and truthful, without having any justification for using that figure! He then did not publish a report36 which proved him wrong.

Thirty years earlier, Abelson, editor of the same journal, *Science*, did not publish one of Velikovsky's papers, without having read it.31 In 1992, the same Abelson was a member of the panel which produced the *National Academy of Sciences report on Responsible Science, Ensuring the Integrity of the Research Process*.32

Several years before Abelson rejected Velikovsky's paper, without having read it, there was a high-level effort to censor scientific publications which presented new ideas that were at variance with what so-called authorities defined as truth. The American Association for the Advancement of Science seriously considered, but fortunately did not approve, a resolution "that henceforth any publication that presents new scientific hypotheses should not be allowed to be printed without the imprimatur of a proper professional body." 31 If this resolution had been accepted and implemented, it would avoid the problem of having to deny something because that something would never have appeared in print. There would thus be nothing to deny and therefore no controversy.

Those who are opposed to fluoridation usually find it impossible to publish in so-called reputable dental and medical journals. So they publish in whatever ways they can. Pro-fluoridationists then criticize them and derogate their research because they did not publish in reputable journals. That is precisely what Brown18 and Mitchell19 did to me and my report on fluoridation in Chile. They not only criticized my research, but also derogated the journal in which it appeared and the university which published that journal.

"Those who ignore history are destined to repeat it"

People have been and are still being harmed by fluoride because those who are responsible for fluoridation have, among other things, ignored history. They have ignored what happened when the drinking water of several cities was iodinated in the 1920s. Iodination of Rochester, NY, water began on April 24, 1925.37 It was discontinued officially because it "did not seem practical owing to the great waste." 38 However, complaints of adverse results engendered widespread opposition. "Iodination was practiced for a short time in Sault Ste. Marie, MI, and Virginia, MN, but was speedily abandoned because of numerous objections from residents." In Duluth, MN, "objections ... prevented the inauguration of" iodination of the drinking water.37 "Iodination of the Anaconda, Montana, water supply began in April, 1925, and was continued in October, 1925, April, 1926, and October, 1926. Children in the Anaconda schools [were] also receiving 10 milligrams of chocolate-iodine tablets once a week for 30 weeks during the school year."37

"Some physicians [were] apprehensive lest the 'promiscuous distribution of iodine,' as they put it, to those not in need of the element, cause a marked increase in hyperthyroidism."37 This was actually observed in Derbyshire, England, where it was reported that "An increase in the prevalence of goiter among children following the use of iodine [including 'iodized water'] is most unusual."37 This increase in the prevalence of goiter in Derbyshire may well be due to a paradoxical effect of iodine. Iodine is a close relative of fluorine and both are in the same chemical family know as halogens. Data suggestive of a paradoxical effect of fluorides and iodides on the clotting of milk by pepsin were published in 1928.39 "Thyroid disorders associated with iodine deficiency and excess" also suggests that iodine produces paradoxical results.40

The results of iodination of drinking water in the above-mentioned cities led Robert Olesen, Surgeon in the United States Public Health Service, to conclude in 1927 that "So far, there is considerable doubt as to the ability of iodinized water to reduce the incidence of endemic goiter." And, "The iodination of public water supplies, in its present state of development, can not be recommended for widespread use."37 Olesen's report was published in *Public Health Reports* issued weekly by the United States Public Health Service.37 One year before Olesen's report appeared, Hartsock pointed out, in the *Journal of the American Medical Association*, that "The continuous use of iodine over a long period of time should never be prescribed for adults, and when its periodic use is prescribed, frequent observations of the pulse and weight should be made."38 In 1931, Weston concluded, in the *American Journal of Public Health and the Nation's Health*, that "The addition of iodine in drinking water [to prevet goiter] has also proved disappointing."41

I should not have implied, without qualification, that proponents of fluoridation have ignored history. What they have learned from history is that they should not permit opponents of fluoridation to publish in professional medical and dental journals. As I already pointed out, that is precisely what Hendershot, editor of the *Journal of the American Dental Association*, did to me.

The fluoridators have also ignored the history of what Schubert and Lapp called "radioactive poisons" that were used therapeutically for over half a century with disastrous results." 42 Following the therapeutic use of radon drinking water in 1903 and 1904, first radium salts and then Thorotrast (a commercial product which contained a radioactive isotope and became popular in 1929) continued to be used well into the 1960s. In 1913, the *Journal of the American Medical Association* reported that "the value of [radon] is unquestionably established." Over 80% of 1038 patients with a variety of ailments, recalcitrant to other treatments, "were considered ... by 20 foreign doctors ... to have been improved by the use of radium emanation." Radium was also injected into

mental patients to treat psychoses and other mental problems.

In 1916, an article in the journal *Radium* declared that "Radium had absolutely no toxic effects, it being accepted as harmoniously by the human system as is sunlight by the plant." Radium therapy was listed in the New and Non-official Remedies of the American Medical Association until 1932.42 In 1936, Percy Brown, M.D., who died from overexposure to X-rays, published his book *American Martyrs to Science through the Roentgen Rays*.43 This book presented the biographies of professionals who died from the effects of X-rays.

In the 1950s, articles in medical journals recommended Thorotrast treatment for children. In 1953, a Denver company was marketing a contraceptive jelly containing radium.42 In the 1940s, 1950s and 1960s, hundreds if not thousands of military personnel and civilians were given radium treatments to prevent and cure colds, hearing loss and other ear ailments, and adenoid problems. The victims attributed head and neck cancers, miscarriages, and thyroid and other problems to the radium treatment.44

Fluoridation déja vu

For half a century, promoters of low-level fluoridation and low-level radiation have denied repeated and continuous warnings about the dangers they both pose.

In 1957, Schubert and Lapp pointed out that "One of the strangest aspects of the attitude toward radiation poisoning is that as late as 1924 — nearly twenty-five years after the discovery of radium — no one seemed to understand that when radioactive substances were taken into the body they emitted radiations just as damaging as those produced by an X-ray machine. This seems incomprehensible in view of the fact that it was well known by then that all kinds of radiation — whether X-rays, alpha rays, beta rays, or gamma rays — damage tissues." 42

Actually, many people not only understood but warned about the dangers of radiation. Schubert and Lapp themselves comment on numerous reports of injury and death, caused by radiation, which continuously appeared in newspapers and in medical and scientific journals. But the so-called experts ignored these reports while people died, in some cases agonizing deaths.

In the case of fluoridation, the so-called experts also have also ignored repeated warnings about the toxicity of fluoridation while people have been harmed and in some cases killed. For information about the political, economic and social syndrome of low-level fluoridation pathology, read Joel Griffiths' *Fluoride: Commie Plot or Capitalist Ploy*.2

Sartor Resartus

I shall now respond to criticism of my report on fluoridation in Chile1 by Brown18 and Mitchell.19

I did not select the three communities in Chile which I researched. They were the ones selected by the National Health Service of Chile. The demographic data I used are taken from official government reports. In my report, I also analyzed Briner and Carmona's data and took issue with their conclusions. Briner and Carmona were high-ranking officials in the National Health Service of Chile. Brown and Mitchell criticized my conclusion that fluoridation is harmful because the three communities are not comparable in certain respects. But they did not criticize Briner and Carmona's conclusion (based on data from the same communities) that fluoridation is safe. Brown and Mitchell's selective criticism is a form of structured silence, as I have already pointed out.

Brown and Mitchell also ignored Briner and Carmona's erroneous claim that fluoridation had no effect on the death rates of individuals with congenital malformations. In fact, Briner and Carmona's data revealed that fluoridated Curico had 244% more such deaths than non-fluoridated San Fernando, and 288% more such deaths than the entire country of Chile taken as a whole.

Finally, neither Brown nor Mitchell commented on any of the other serious errors and inadequacies in Briner and Carmona's publication, about which my report presented detailed information. Their silence about the deficiencies in Briner and Carmona's work raises questions about their objectivity.

Briner and Carmona were the two highest ranking officials in the National Health Service of Chile, Section of Odontology, when they presented their report in 1965 at the Fifth International Odontological Congress of Chile. The Commission of Dental Health of that Congress endorsed the safety of fluoridation in Chile on the basis of their report.

The *Journal of Arts, Science and Humanities* and Anthony University exist

Brown18 and Mitchell19 attempted to cast doubt about the validity of my Chile report by pointing out that it was published in an obscure journal. They thus obscured the real issue which is why responsible scientists and other professional researchers have been blackballed and denied the opportunity to publish their criticism of fluoridation in professional dental and medical journals in the United States.

Despite the fact that my report on fluoridation in Chile was published in an obscure journal, that report was directly responsible for terminating fluoridation in Chile. As soon as it was printed, I sent copies to every dental and medical officer in the Pan-American Health Organization and the National Health Service of Chile. I also sent copies to professors in the faculties of medicine, dentistry and pharmacy in the University of Chile. Shortly thereafter, fluoridation was discontinued in Chile. However, the fight may not be

over. Present-day bureaucrats may not know that some 20 years ago fluoridation was discontinued in Chile because it was killing people in the experimental city of Curaco..

The existence of the *Journal of Arts, Science, and Humanities* and Anthony University which published that journal has also been questionsed (The name Anthony University has since then been changed to Susan B. Anthony University.) The university was establihsed as a not-for-profit corporation in the State of Missouri on June 13, 1973. This can be verified by directing an inquiry to the Secretary of State, Department of State, Jefferson City, Missouri.

Doubts about the existence of the *Journal of Arts, Science and Humanities*, which contains my report, can also be easily laid to rest. This journal was copyrighted in 1976, which is its legally documented year of birth. The copyright and date of publication can be verified by directing an inquiry to the Library of Congress, Copyright Office, Washington, D.C. Copies of this publication can be ordered from the Copyright Office, Library of Congress.

Additional evidence that the *Journal* exists is the fact that copies of that journal, containing my report on fluoridation in Chile, have been distributed by the National Institute of Dental Research. This is part of the National Institutes of Health in the U.S. Public Health Service (Bethesda, Maryland). On October 28, 1986, John S. Small, Information Specialist at the National Institute of Dental Research, wrote me as follows on official, government letterhead

Dear Dr. Schatz:

We would appreciate having a few more reprints (3-5) or your permission to reproduce several copies of your January 1976 article on fluoridation in Chile (Anthony University *Journal of Arts, Science, and Humanities*. v. 2, no. 1)

The needed copies would be for distribution to interested health professionals or writers specializing in health sciences as requests arise.

A postage-free envelope is enclosed for your use in sending copies or your note or permission to make copies. Thank you.

"Truth will come to light. Murder cannot be hid"

The *hit-and-run tactic* is an appropriate way to describe how proponents of fluoridation attempt to discredit those who oppose them. They publish a critique of one sort or another in a professional journal, but the individual they attack cannot publish his rebuttle in the same journal. An example of this *hit- and-run* is the recent attempt to discredit John Yiamouyiannis by John Hunt, chief executive of the British Dental Society, and four of his colleagues with supposedly impressive credentials.45 Unfortunately, few if any readers of the British Dental Society, in which Hunt *et al* published their critique, will see Yiamouyiannis' reply 46 They will therefore not be able to decide for themselves "where ... misleading statements are coming from, who is using 'deception by omission,' and whose references do not support their claims."46

In his reply, Yiamouyiannis poses an important question, "If one doesn't 'have a complete knowledge of the detailed and voluminous scientific literature on the relationship of water fluoridation to dental caries,' or close to [that], how [is he] going to respond to someone with an opposing viewpoint who does? [Will it be] by name-calling, by character assassination, by distortions, by misrepresentations, by undocumented fabrication?"46

But there are other important questions that should be asked about low-level fluoridation because of what we know about low-level radiation. Does low-level fluoridation, like low-level radiation, involve something far more serious than the usual misconduct of science? John Gofman's realization of the profound responsibility that scientists have to warn people about the danger of low-level radiation are best described in his own words.

"I was stupid in those days. In 1955, '56, people like Linus Pauling were saying that the bomb fallout would cause all this trouble. I thought, 'We're not sure. If you're not sure, don't stand in the way of progress.' I could not have thought anything more stupid in my life.

"The big moment in my life happened while I was giving a health lecture to nuclear engineers. In the middle of my talk it hit me! What the hell am I saying? If you don't know whether low doses are safe or not, going ahead is exactly wrong. At that moment, I changed my position entirely."47

In 1979, Gofman expressed his feelings as follows. "There is no way I can justify my failure to help sound an alarm over these activities many years sooner than I did. I feel that at least several hundred scientists trained in the biomedical aspect of atomic energy - myself definitely included - are candidates for Nuremburg-type trials for crimes against humanity for our gross negligence and irresponsibility. Now that we know the hazard of low-dose radiation, the crime is not experimentation - it's murder." 48

Now, back to questions about fluoridation which is still highly controversial after half a century. Are the harmful effects of low-level fluoridation due to "gross negligence and irresponsibility"? If so, is low-level fluoridation a "crime against humanity? If it is,

should those responsible for the harmful effects of low- level fluoridation be considered "candidates for Nuremburg-type trials for crimes against humanity."?

Acknowledgement

I salute John Yiamouyiannis and others for what they has been doing to inform people about the dangers of low-level fluoridation, just as I salute John Gofman and others who have been informing people about the dangers of low-level radiation. Many of these people, however, pay a high price for what they do because they have publicly questioned the integrity of the scientific establishment.

Gofman has recounted some of that happened to him.47Robert O, Becker, who refused to keep silent about electropollution - the dangers of manipulating our electomagnetic environment - has told the sad story of what happened to him. His account is in the last chapter in his book *The Body Electric*, that he co-authored with Gart Selden.49 Becker explained as follows why he revealed the retribution he experienced.

"I've taken the trouble to recount my experience in detail for two reasons. Obviously, I want to tell people about it because it makes me furious. More important, I want the general public to know that science isn't run the way they read about it in the newspapers and magazines. I want lay people to understand that they cannot automatically accept scientists' pronouncements at face value, for too often they're self-serving and misleading. I want our citizens, nonscientists as well as investigators, to work to change the way research is administered. The way it's currently funded and evaluated, we're learning more and more about less and less, and science is becoming our enemy instead of our friend."49

Both sides of the coin are important

"The importance of scientists' writing their own personal accounts of their dicoveries is now recognized. "For the historian of science, few documents are as valuable as the description of a discovery by the scientists involved in the action. Unfortunately few scientists take the time to record for posterity the course of events which led to the discoveries which were the fruit of their labor." (Lechevalier)50

"I have often thought how much more interesting science would be if those who created it told how it really happened, rather than reported it logically and impersonally, as they often do in scientific papers." (Beadle)50

"Over the years, the story of streptomycin's discovery has been terribly garbled. I think … it would be a great service if … Dr. Schatz told his own accurate and interesting account of his finding. Streptomycin turned out to be a milestone in the history of drugs to treat tuberculosis and other infections. Dr. Schatz's role has been largely ignored. The record about this discovery should be set straight." (Doris Jones Ralson, a fellow graduate student of Schatz when he did the streptomycin research)50

Our survival as a species is now threatened

I agree that personal accounts of discoveries by the scientists who made those discoveries are important. That is why I wrote *The True Story of the Discovery of Streptomycin.*, which my friend and colleague Doris Jones Ralston suggested I do.50 However, it may be even more important for whistleblowers to publish detailed accounts of how the scientific establishment has attempted to silence and punish them.

We are now at a critical time in history because our survival as a species is threatened as a result of our global devastation of nature. Science is the main force that has been used for the manipulation, exploitation, and devastation of nature. It is therefore important for the history of science, for the welfare of life on this planet, and for our survival as a species that those who have been pilloried for questioning the integrity of science and exposing misconduct by scientists tell, in their own words, the stories of what happened to them.

Fluoridation, is a major environmental pollutant,2 which along with many other chemicals is now a major part of the threat to our survival. Those scientists and other professionals who have opposed fluoridation have a unique oportunity to make a major contribution to history, to science, and to the survival of our species by telling the stories of how they were persecuted for their continued determination to inform their fellow men and women about the dangers of fluoridation.

Epilogue

"Until about a hundred years ago, rational men lived like spies in an enemy country. They never walked abroad unless disguised in irony or allegory. To have revealed their true selves would have been fatal.

"Today their status is more that of guerillas. They snipe from cover, ambush stragglers, harass retreating rear guards, cut communications, and now and then execute swift forays against detached units of the enemy. But they dare not yet risk an open engagement with the main force; they would be massacred. Their life is dangerous but exciting and is warmed by a sense of camaraderie not often known among the dull conscripts of orthodoxy.

"This" report "is intended as a sort of handbook for ... recruits in the ... cause of common sense. It indicates where the main armies of ignorance are now encamped and tells in a secret code what garrisons are undermanned or mutinous. It tries to show the use of

cover and camouflage and the techniques of infiltration and retreat. It maps road blocks and mine fields and shows how to rig a booby trap. It warns of counterespionage and gives — again in code — the ... infallible signs to know a fool.

*"When the recruit has finished with it, he can toss it over the walls into the enemy's barracks. It may encourage desertion."*51

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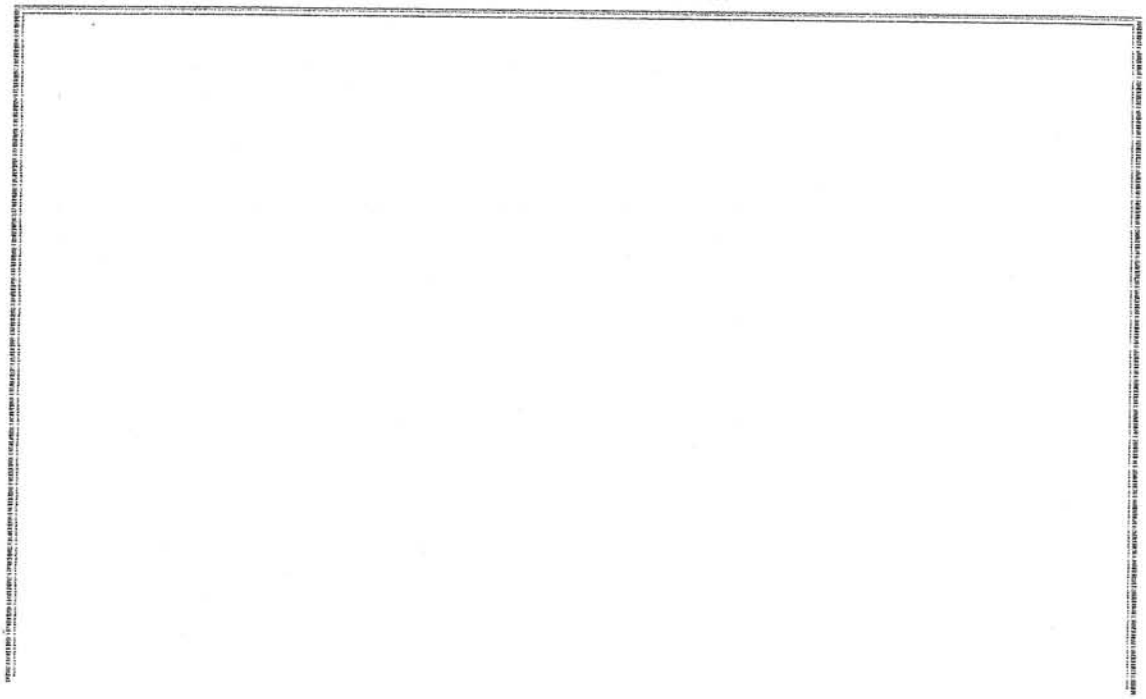
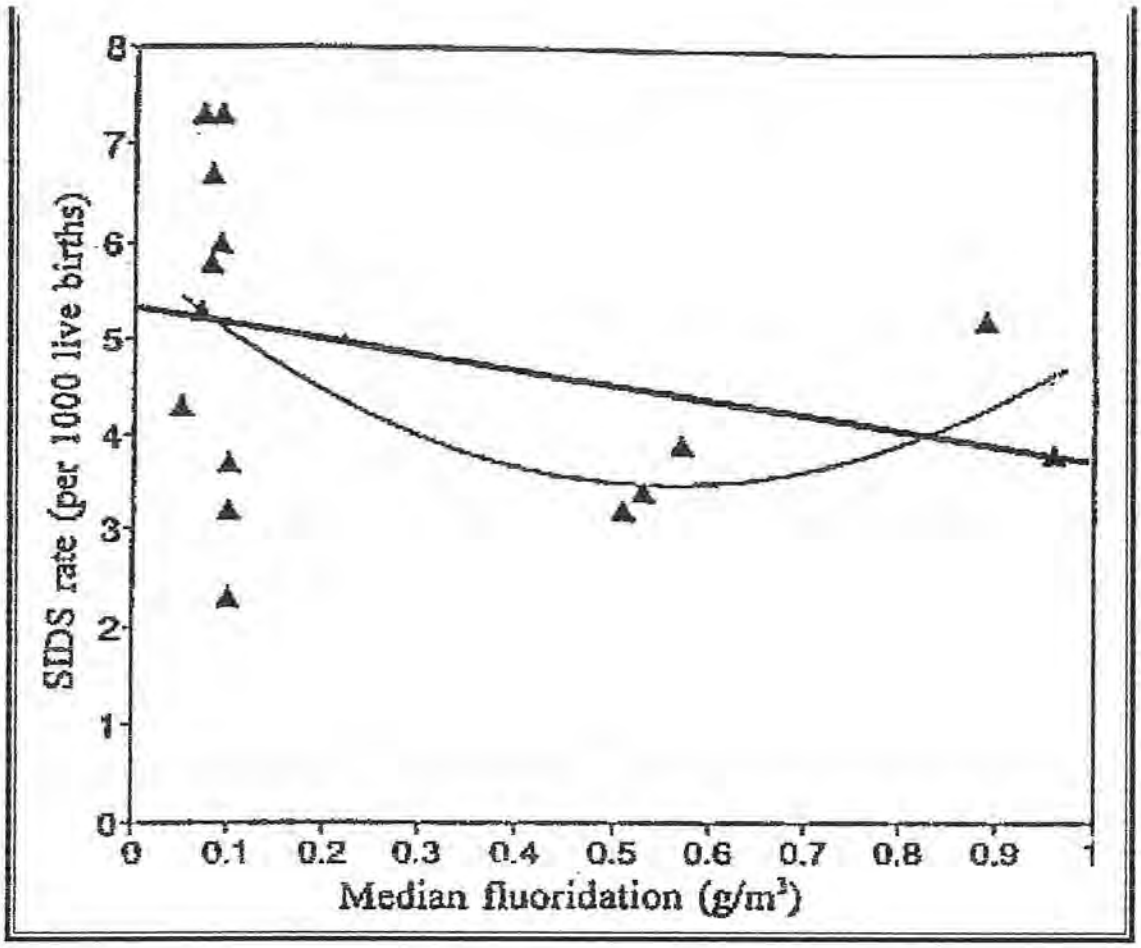
Appendix A:

Who is Albert Schatz, the author of this report?

FIGURE 1,
Figure 1. Mean SIDS mortality rates (1980-1984) and median fluoridation in reticulated water supplies for New Zealand health districts.





Appendix B: Risk assessment and paradoxical effects

This is not the place to review the literature on the limitations of risk assessment. Suffice it to say that we need adequate and reliable risk assessments for many pharmacological products, food additives, chemical environmental pollutants such as pesticides, radioactive contamination, hazardous wastes, and other harmful and potentially harmful substances that pollute our air, water, food, homes, work places, schools, recreational areas, etc. Unfortunately, risk assessment is too often little more than guesswork or a trade-off. Also unfortunately, political considerations not infrequently influence decisions as to how much exposure is permitted. What is even more deplorable is that there is no risk assessment at all for many toxic and potentially toxic substances to which we are frequently exposed.

It is not surprising that the risk assessments of low-level fluoridation and low-level radiation are highly controversial issues because there is disagreement on methods of determining risks and interpretation of research results. At the same time, it is inconceivable that meaningful risk assessment can be done without ascertaining whether paradoxical effects are involved. If they are, then risk assessment becomes considerably more complex, time-consuming, and costly. Nonetheless, the Food and Drug Administration (FDA), the Environmental Protection Agency (EPA), and other state and federal agencies should seriously consider paradoxical effects.

In 1964, Schatz , Schalscha, and Schatz published the first review of paradoxical effects6 in which they pointed out that "Paradoxical effects occur more frequently and are more widely distributed than one would ordinarily assume from scattered reports.... Paradoxical effects have been produced by radiation, temperature, mutagenic and carcinogenic chemicals, steroid hormones, dextran, detergents, trace metals, herbicides, fungicides, insecticides, germicides, antibiotics, drugs, and a host of other agents...

"Since numerous chemical and physical agents cause paradoxical effects by different mechanisms in many biological systems, these reactions will no doubt become increasingly important in pharmacology, toxicology, chemotherapy drug idiosyncrasies, air pollution, chemical carcinogenesis, fluoridation, fallout, radiation effects, nutrition. biogeochemistry, the weathering of rocks and minerals, soil formation and soil fertility, and many other areas"6 including behavioral toxicology.

Also in 1964, Schatz and Martin published the first review on *The Importance of Paradoxical Effects of Fluoride with Respect to Fluoridation and the Toxicology of Fluoridation.*5

Thirty years later, in 1994, Milton Wainwright (in the Department of Molecular Biology and Biotechnology, at the University of Sheffield, England) published a review of our two reports which he called *Strange bumps in the data - mycological implications of the paradoxical concentration effect.*52 In his review, Wainwright commented as follows, "Schatz himself experienced difficulty in publishing his work on paradoxical effects in 'front-ranking' journals. His work on the subject, including two reviews, which are well written and thought-provoking by any standards, were eventually accepted for publication in *Compost Science* 6 and the *Pakistan Dental Review*5 and so have probably been read by only a few microbiologists.

"I hope this article has highlighted the fact that unusual bumps in data are not always the result of an experimenter having a bad day. Perhaps under the umbrella of Schatz's 'paradoxical effect', this potentially important phenomenon will gain respectability and receive the attention it deserves."52

It is interesting that Wainwright was the first to publish information on (a) the importance of my work on paradoxical effectsx and (b) my role in the discovery of streptomycin. 53

Unfortunately, the FDA, the EPA, other agencies, and individuals concerned with toxicology and other areas in which paradoxical effects undoubtedly occur, have yet to pay attention to this important phenomena which may literally influence not only health but also determine whether people live or die.

Aside from paradoxical effects, there is another reason to doubt the claim that low-level fluoridation is safe. When radiation and medication are administered, the dose is quantitatively adjusted daily for each individual. But fluoridation disregards "the uniqueness of each individual and the degree of variability of response among individuals.12 Individuals differ in how much water they consume daily. Furthermore, the fluoride content of drinking water varies from day to day. It is not invariably one part per million, which it is supposed to be. Therefore, the risk assessment of fluoridation is based on an average daily intake of fluoride from a source whose fluoride content varies daily. Consequently, no one knows how much fluoride each individual ingests daily from drinking water, and from other background sources. This is "playing with fire" because fluoride is a highly toxic substance. For these reasons, independently of paradoxical effects, it is not surprising that low-level fluoridation is harmful to some people. It would be surprising if there were no harmful effects.

"The fluoridation bottom line floats to the top when realizing governments and responsible scientific documentation on fluorides and fluoridation confirm that after 50 years of forced fluoridation, no government has felt it necessary or morally obligated to study the effect of fluoridation on human fertility and fluoride damage to the foetus, and the subsequent birth of babies.

"No drug except fluoride is allowed such evil medical exception from government, pharmaceutical and poison laws, and that evil is compounded when the same responsible people force fluoridation against the wish of the people.

"As it cannot be judged democratic, one may consider it the enemy of freedom, honesty, science, and morality, all based on their [the promoters' of fluoridation] fear of the dreadful truth about fluoridation and fluoride chemicals…

"Hidden on page 91 of the U.S. Department of Health and Human Services [in the section *Review of Fluoride, Benefits and Risks,* 1991] the authors make this recommendation: 'Conduct studies on the reproductive toxicity of fluoride using various dose levels including minimally toxic maternal dose.'

"Question - Why not?"54

Appendix C: The failure of fluoridation in England[21]

The title of this Appendix is the title of an article which I published in 1972.[21] It is reproduced on pages 17,18,19, and 20 of this report because (a) it is not otherwise readily available and (b) as I have already said, "It has to be seen to be believed." My 1972 publication shows that fluoridation does not prevent dental caries.

The information in the following two paragraphs was inadvertently left out of the original manuscript of the 1972 article when it was submitted for publication.

"There is a flaw in this British study which the authors were unaware of. The flaw invalidates epidemiological surveys that show less decay in fluoridated children than in non-fluoridated children of the same age. The flaw involves the difference in the age of the children versus the post-eruption age of their teeth; that is, the age of the teeth after they appear above the gum line. Although the two groups of children are the same age, the teeth of the children in the two groups are not the same age because fluoride delays tooth eruption.

"The teeth of fluoridated children, which erupt later, are younger than the teeth of non-fluoridated children, which erupt earlier.

Because the teeth of the fluoridated children are younger, they have been exposed to cariogenic conditions in the mouth for less time than the teeth of non-fluoridated children. Because of this shorter exposure to cariogenic conditions, the teeth of fluoridated children understandably have less decay. Therefore, the less tooth decay that occurs in fluoridated children cannot be attributed to any cariostatic action of fluoride. This conclusion is supported by the fact that the rate at which tooth decay occurs is the same in both groups, as shown in Figure 1."

FIGURE 1, Curves showing that dental caries develops at the same rate in the permanent teeth of fluoridated and non-fluoridated children

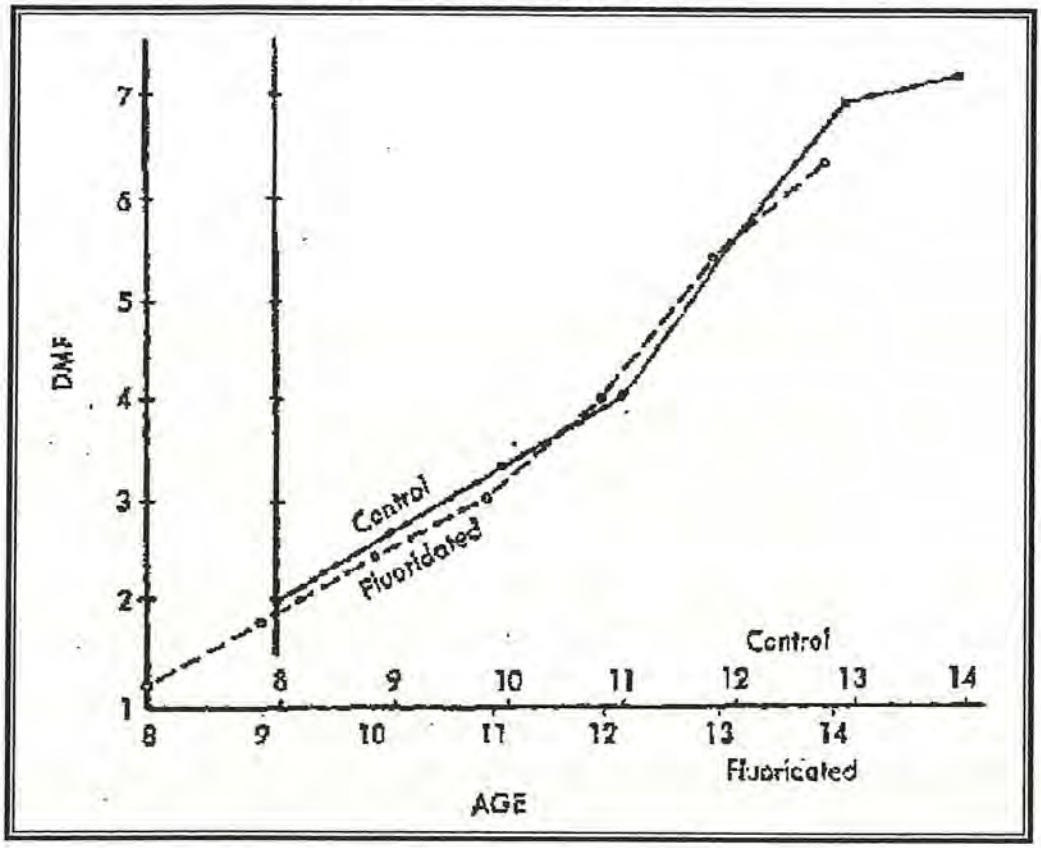


TABLE I

DMF for permanent teeth of fluoridated and non-fluoridated children.

Age	Average DMF per child: Fluoridated Areas	Control Areas	% Difference in DMF
8	1.2	2.0	67*
9	1.8	2.7	50
10	2.4	3.3	37
11	3.0	4.0	33
12	4.0	5.6	40
13	5.4	6.9	28
14	6.3	7.2	14

*67% = (2.0 - 1.2)/1.2 x 100.

Home

Fluoride: Protected Pollutant or Panacea?
Are the claimed benefits of ingesting fluoride over-rated
and the risks to our health and eco-system under-reported?

Abstracts

Bones | Calgary | Cavities | Fertility | Cancer | Health risks | Neurological | Dental Fluorosis and Pictures
ISFR | Ethics | Tributes | Fraud | Authors | Deaths | Quotes | Environment | Skeletal Fluorosis | Definitions

Newsweek, Feb. 5, 1990

SCIENCE

Don't Drink the Water?

Brush your teeth, but the fluoride from your tap may not do much good—and may cause cancer

Remember the great fluoride debate? Back in the 1950s, every voice of authority, from the U.S. Public Health Service to the PTA, supported adding fluoride to the water supply as an effective and totally safe way to promote healthy teeth. The only opponents seemed to be John Birchers and other extremists who regarded the scheme as a diabolical communist plot. In the years since, most of the nation's major cities fluoridated their water, and the issue appeared closed. No less an objective voice than Consumer Reports declared in 1978, "The survival of this fake controversy . . . represents one of the major triumphs of quackery over science in our generation."

In fact, the debate never ended. Now it may explode as never before, posing new challenges to medical dogma and giving parents one more thing to worry about. Government researchers have new evidence that casts doubt on the benefits of fluoridation and suggests that it is not without risk. The most incendiary results come from the National Toxicology Program (NTP), which in 1977 was ordered by Congress to determine whether fluoride causes cancer. This week NTP plans to release data showing that lab rats given fluoridated water had a higher rate of a rare bone cancer called osteosarcoma. According to a memo by the Environmental Protection Agency, "very preliminary data from recent health studies . . . indicate that fluoride may be a carcinogen."

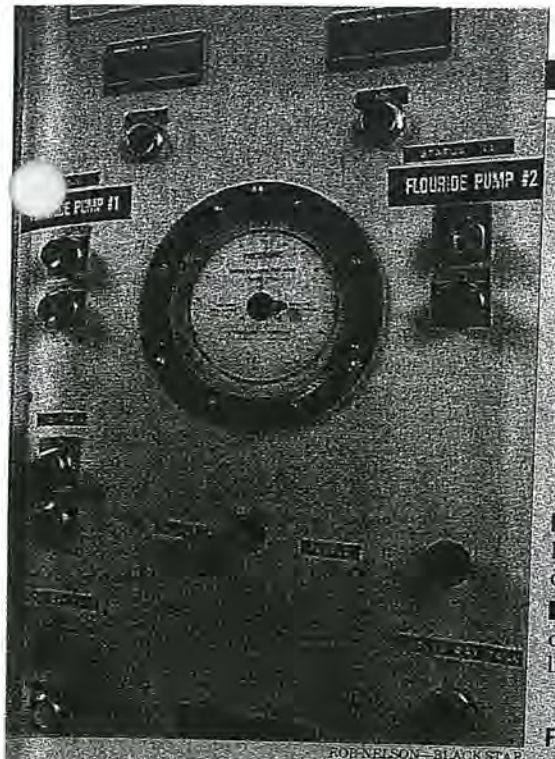
Fluoridation proponents are already criticizing the NTP study, but it will be harder to discredit or ignore than the hundreds of earlier experiments, of varying quality and from around the world, that have linked fluoride to mottled teeth, skeletal damage, genetic defects and other ills. During the two-year experiment, rats and mice drank water with different levels of sodium fluoride. None of the animals drinking fluoride-free water developed cancer, nor did any of those drinking water with the lowest fluoride concentration, 11 parts per million (ppm). But of the 50 male rats consuming 45-ppm water, one developed osteosarcoma. Four of 80 male rats drinking 79-ppm fluoride developed osteosarcoma. No mice or female rats showed signs of bone cancer. Although the anima drank higher concentrations of fluori than people do (the legal standard is fo ppm), such megadosing is standard toxic logical practice. It's the only way to dete an effect without using an impossibl- larg number of test animals to stand th humans exposed to the substance.

Although the final NTP report will nc be released for months, several indepen ent toxicologists find the results signif cant. Most important, the rats who did nc drink fluoride did not get cancer, indica ing that the malignancies are "not a fluke, says EPA scientist William Marcus. Ther is also a convincing relationship betwee dose and response: the more fluoride, th more cancers. Pathologist David Kaufma of the University of North Carolina warn that the rat data must be examined to see i the cancers appeared in the long bones o the arms and legs, as osteosarcomas do ir humans, or in other places, which migh make the results less relevant to people Still, Kaufman says the NTP data "make fluoride look like a weak carcinogen. It's obviously something to worry about"—but not panic over. There are about 750 cases of osteosarcoma in the United States annually; even if fluoride caused all of them—an impossibility—the lifetime risk to any individual from drinking fluoridated tap water would still be only about one in 5,000.

Too crude: If fluoride causes bone cancer in lab rats, then why, after 45 years of fluoridation, haven't researchers seen a rash of osteosarcomas in fluoridated cities? Because epidemiology is too crude to de even if the cancers are there. In the 19 0s, the National Cancer Institute found no sign of higher cancer rates in fluoridated

From the beginning, controversy: *In 1965, the protests reached the reservoir's edge*

UPI-BETTMANN NEWSPHOTOS



ROB NELSON—BLACK STAR

Fluoridation: *Atlanta's waterworks*

Fluoride Facts

- Fluoride—in water or toothpaste—helps teeth resist decay. It seems to work by redepositing calcium and other ions in tooth enamel, repairing and strengthening it.
- 53% of the U.S. population drinks water containing fluoride. 121 million people have artificially fluoridated water, 9 million drink from naturally fluoridated supplies.
- 41 of the 50 largest U.S. cities have fluoride in the water; those that don't include L.A. and San Diego.
- The legal standard for fluoride in drinking water is four parts per million; for toothpastes, 1,100 ppm.

cities. But that reassuring finding may be misleading. According to Donald Taves, a fluoride expert, if the difference were anything less than 7 percent it would not be detectable. Another obstacle to definitive epidemiology is mobility: just because someone got osteosarcoma in a fluoridated city does not mean he had been living there all his life.

The NTP results assume an added importance when combined with recent data on the shrinking benefits of fluoridation. According to the American Dental Association (ADA), tooth decay is anywhere from 50 to 70 percent less in fluoridated areas. But figures from the National Institute of Dental Research (NIDR), part of the National Institutes of Health, suggest otherwise. A 1987 survey of almost 40,000 schoolchildren found that tooth decay had declined sharply everywhere. Children who had always lived in fluoridated areas had 18 percent less decay, compared with their peers who had lived in nonfluoridated areas. This 18 percent translates into a difference of fewer than one cavity per child. Similarly, in a 1986 paper in the British journal Nature, Australian researcher Mark Diesendorf assessed 24 studies from eight countries and found that cavity rates had declined equally in fluoridated and nonfluoridated areas, suggesting fluoridated water isn't that important.

How can that be? "A good case can be made that it has to do with fluoride in toothpaste and rinses," says dental-health expert Brian Burt of the University of Michigan. And even if drinking fluoridated water is slightly risky, there is no hint that fluoridated toothpaste—as long as you don't swallow any—is dangerous. Tooth decay may also be declining because of better diet and hygiene. Also, foods and beverages processed with fluoridated water are ubiquitous. (Many bottled waters, though, do not have fluoride.) As a result, argues Alan Gray, a leading pro-fluoridation dentist in Canada, "it is becoming difficult to provide accurate, ethical advice" about fluoridation.

Among environmental controversies, fluoridation is unique in that one side has consistently denied that questions of risk or benefit even exist. The ADA states, "Anti-fluoridation groups attempt to create the illusion of a scientific controversy [which is] merely a ploy to create doubt about a well-researched, well-demonstrated preventive measure." But even well-researched articles raise hackles. When, in 1988, Chemical & Engineering News presented a balanced report on fluoridation, it attracted the wrath of the medical establishment. Says Taves, "Too many scientists lost their objectivity. This has become a religion on both sides."

Safe water: And that undercut the scientific process. The NIDR kept files on people perceived as threats to fluoridation. Political decisions were at odds with expert advice: a panel convened by the surgeon general in 1983 expressed concern, in closed sessions, about skeletal and dental damage from fluoride. At one point, a member said, "You would have to have rocks in your head, in my opinion, to allow your child much more than two parts per million [fluoride]." Said another, "I think we all agree on that." Even so, in 1986 EPA *raised* the fluoride standard from about two ppm to four.

This month EPA opened a review of the standard. Once EPA receives the official NTP report, it will establish a target "safe" fluoride level. The Safe Drinking Water Act requires that the level be zero for carcinogens, but the standard may be based on what is technically feasible. Fluoridation can be stopped immediately, but many communities with naturally fluoridated water—up to 12 ppm—would have to remove it. As EPA wrestles with the standard, fears John Sullivan of the American Water Works Association, "confusion will reign": local laws will still require fluoridation, a practice that may cause cancer.

As they await EPA's decision, pro-fluoridationists are invoking arguments of social justice. Dental researcher Ernest Newbrun of the University of California, San Francisco, contends that fluoridation promotes the health of children of "all races and all socioeconomic classes," not only those with enough money or discipline or access to the health system to take a fluoride supplement every day. He and others say it is morally wrong not to provide the benefits of fluoride. Although the NIDR's and other surveys suggest that fluoride in toothpastes and dental rinses also ensures healthy teeth for those who use the products, those who do not might suffer.

No one can foresee how the fluoride debate will play out this time. But since the 1950s, the country's environmental consciousness has been heightened. In the end, deciding whether or not to fluoridate turns less on science than on values. The sheer weight of good research may finally, after four decades, begin to inform those judgments and even overwhelm the unscientific rhetoric that has characterized both sides of the debate for far too long.

SHARON BEGLEY

PHOTOS BY JACQUES CHENET—NEWSWEEK

After every meal: *Toothpastes to fight cavities*

NoFluoride.com

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Don't Drink the Water?

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Excerpts from the Feb 5, 1990 Newsweek article:

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the more fluoride, the more cancers

There is also a convincing relationship between dose and response: the more fluoride, the more cancers. Pathologist David Kaufman of the University of North Carolina warns that the rat data must be examined to see if the cancers appeared in the long bones of the arms and legs, as osteosarcomas do in humans, or in other places, which might make the results less relevant to people. Still, Kaufman says the NTP data "make fluoride look like a weak carcinogen...

If fluoride causes bone cancer in lab rats, then why, after 45 years of fluoridation, haven't researchers seen a rash of osteosarcomas in fluoridated cities? Because epidemiology is too crude to detect it even if the cancers are there. In the 1970s, the National Cancer Institute found no sign of higher cancer rates in fluoridated cities. But that reassuring finding may be misleading. According to Donald Taves, a fluoride expert, if the difference were anything less than 7 percent it would not be detectable. Another obstacle to definitive epidemiology is mobility: just because someone got osteosarcoma in a fluoridated city does not mean he had been living there all his life.

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Sharon Begley

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Anti-Fluoridation Portland Dentist Hails New Harvard Study

Dr. Kyung L. Boen is among a sprinkling of dentists in the Portland Metropolitan area that are against adding fluoride to the water supply known as fluoridation. Dr. Boen believes that anti-fluoridation advocates in the United States have been partially vindicated by a new Harvard study and a recent National Academy of Sciences report. Dr. Boen is confident that a complete vindication of her anti-fluoridation stance and an ADA fluoridation support reversal will come within the next decade.

Portland, OR, April 09, 2006 --(PR.com)-- Dr. Kyung L. Boen is among a sprinkling of dentists in the Portland Metropolitan area that are against adding fluoride to the water supply known as fluoridation. While she advocates limited fluoride dental applications that are applied directly to the teeth, she generally promotes using a fluoride substitute such as Xylitol, which is safe for young children and even infants. Dr. Boen believes that anti-fluoridation advocates in the have been partially vindicated by a new Harvard study and a recent National Academy of Sciences report. Dr. Boen is confident that a complete vindication of her anti-fluoridation stance and an American Dental Association (ADA) fluoridation support reversal will come within the next decade.

It was reported this week that young boys who drink fluoridated water, considered safe by federal guidelines, are at an increased risk of developing bone cancer than boys who drink unfluoridated water, according to a new study published in the May issue of the Harvard journal, Cancer Causes and Control. A team of Harvard University scientists, led by Dr. Elise Bassin, found a 5-fold increased risk of developing Osteosarcoma in teenage boys who drank fluoridated water at ages 6, 7, and 8. The research, funded by the National Institute of Environmental Health Sciences, reinforces previous findings in both humans and animals.

This is only two weeks after the prestigious National Academy of Sciences' National Research Council recommended the immediate reduction of fluoride in drinking water. The committee reported that children exposed to the current maximum allowable concentration of fluoride in drinking water risk developing severe tooth enamal fluorosis, which is a condition characterized by teeth discoloration, enamel loss, and pitting of the teeth. The majority of the committee stated that the damage to teeth caused by severe enamel fluorosis is a toxic effect that is consistent with prevailing risk assessment definitions of adverse health effects. Additionally, the majority concluded that people who consume water containing that much fluoride, over a lifetime, are likely at increased risk for bone fractures.

Most interestingly for many to learn may be that relative to their body weight, infants and young children are exposed to 3 to 4 times as much fluoride as adults. Moreover, on average, approximately 10 percent of children in communities with water fluoride concentrations near or at 4 mg/L develop severe tooth enamel fluorosis. That means thousands and thousands of American children are being slowly poisoned and doomed to suffer enamel loss and pitted teeth due to this high-concentration of fluoride in their drinking water.

Some of the countries that do not fluoridate their water supply include Austria, Belgium, Bulgaria, Denmark, Finland, France, Germany, Greece, Hungary, Iceland, Italy, Japan, Luxembourg, Netherlands, Norway, Philippines, Romania, and Sweden. Yet despite the fact that these countries have decided strongly against water-supply fluoridation, they have experienced the same significant declines in dental cavities as the United States. Approximately 68 percent of Americans currently have access to optimally fluoridated water.

The reported hazards to human health from ingesting fluoride include acute toxic hazard, such as to people with impaired kidney function, as well as chronic toxic hazards of gene mutations, cancer, reproductive effects, neurotoxicity, bone pathology and dental fluorosis. Most of the beverages we drink, such as beer, soda and juice, are made with fluoridated water. Fish and other foods contain fluoride. The fruits and vegetables we eat often are grown with fertilizers

that contain fluoride, thus they can have high concentrations of fluoride such as grapes and watermelon. Most Americans cook their foods in fluoridated water.

Dr. Boen graduated from the renowned Oregon Health & Sciences University School of Dentistry in 1994. Five years later, she received her Fellowship from the Academy of General Dentistry. Dr. Boen is a solo-practitioner in her high-tech dental clinique and perhaps only one of a handful of dentists on the West Coast that personally offers invisalign, full orthodontics, i.v. sedation, CEREC 3D porcelain restorations, and Waterlase MD laser dentistry. The doctor holds dental licenses in Oregon, Washington and California.

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Dental fluorosis is the 1st visible sign of fluoride poisoning. (a discoloring of the teeth).

COMMUNITY AND HEALTH & HUMAN SERVICES STUDIES

Town of Wilmington

Board of Health

121 Glen Road
Wilmington, Massachusetts 01887

Fluoridation

A Discussion on Whether the Board of Health
Should order the Fluoridation of the Municipal Water Supply

By Gregory Erickson, Director of Public Health

February 15, 2000

Near the beginning of 1999, the Wilmington Board of Health began to investigate the proposition that the town's water supply should once again be fluoridated. In an effort to learn the latest information about fluoride, I began my own research on the subject. In doing so, I have looked at the background, history, science and research presented to date, and, have encountered many areas which have raised a good deal of concern. In pursuing suitable explanations to these concerns, I have only encountered even more concerns.

As is the case with many public programs, it is common to find disagreement, even after serious investigation and debate on the issues, and even among friends and long standing associates. Such is the case with the issue of fluoridating the water supply for the Town of Wilmington. As Director of Public Health, I have made my own observations and study and I have done so independently with the intent of making a responsible recommendation to the Board of Health and to the public.

Board of Health has divided the fluoridation issue into three main categories:
(1) Is it safe?
(2) Is it effective?
(3) Is it right for the Town of Wilmington?

I have divided my observations into the same categories for the sake of consistency. My concerns are based on government documents, research papers, personal communications, personal interviews, a related seminar, and two Board of Health hearings. I have formulated a recommendation which I feel focuses on the major concerns that should be considered by the Board of Health and the public at large.

The First Issue: Is Fluoridation safe?

I have discussed this one issue with both of the authors of the paper **Applying the NAEP Code of Ethics to the Environmental Protection Agency and the Fluoride in Drinking Water Standard,** by Robert J Carton, Ph.D. and J. William Hirzy, Ph.D. Both are presently scientists, employed by the Environmental Protection Agency (EPA) and they have confirmed to me the authorship and authenticity of their paper which describes in detail the procedure that the EPA used to raise the Maximum Contaminant Level (MCL) of fluoride in drinking water from 1.0 ml/L to 4.0 mg/L and how it was done inappropriately, with a succession of irregularities, and in violation of the EPA's own Code of Ethics. As a result of this, in 1997, the National Federation of Federal Employees Union (local 2050)

which is comprised of the scientists, researchers, and attorneys who work for the EPA has taken the unanimous position to oppose the fluoridation of public water supplies.

This point, taken by itself, causes a great deal of concern to me. I question how the EPA as a governmental agency charged with the responsibility of insuring safe drinking water standards for the nation can recommend the fluoridation of water supplies when its own scientists working there are unanimously opposed to it. These EPA employees have taken other actions to openly oppose the fluoridation of water supplies, such as the writing of several papers on the issue, making videos, and actively lobbying the Governor of California, for example, to not fluoridate the water supplies of that state.

Considering that these are the professionals who comprise the scientific community that sets the standards for toxicity and enforcement of the drinking water standards for the nation, this point alone, without adequate explanation, is sufficient for anyone to reject the notion that fluoridation is safe. On this point, I remain open to any explanation that can be offered up to adequately counter the logical conclusion.

Another concern is that the EPA has reclassified fluorosis from that of a "health effect" to a "cosmetic effect", and has done so without the benefit of hearings or scientific input. This includes all levels of fluorosis, including severe fluorosis. It is the position of the EPA that there is no "health effect" until there is a "loss of tooth function". This means, in layman's terms, that one must loose a tooth or teeth, or loose the function of a tooth or teeth in order for a "health effect" to exist. Up to that point, one can have permanently stained, cracked, pitted and mottled tooth enamel, and a "health effect" is not considered to be present, according to the EPA. This is a completely absurd and unacceptable position. I don't think that any parent would accept this standard for their own child. As a public health administrator, I cannot accept it for the Town of Wilmington.

Point in fact: Fluoride causes fluorosis of the teeth. This is not a debatable issue. In fact, the discovery that fluoride causes less cavities to occur was a result of the correlation found between the occurrence of fluorosis and the occurrence of less cavities, where in Texas, naturally occurring fluoride existed at higher levels. It has been presumed that by adding fluoride to the water supply, it would result in better dental health. However, history has shown that by adjusting the fluoride at a lower level (1.0 mg/L) which causes less fluorosis negates the effect of the fluoride with regard to dental health. Those who support fluoride have said that this is mild fluorosis fox the most part. And this is true, for the most part. What of the lesser part?

The **Executive Summary, Review of Fluoride Benefits and Risks** by the U: S. Public Health Service, Department of Health and Human Services, February 1991, states that:,

"Moderate and severe forms of dental fluorosis, considered by some investigators as presenting a cosmetic problem, do not appear to produce adverse dental health effects, such as the loss of tooth function, and represents less than six percent of the cases of fluorosis nationally."

So according to the Public Health Service, it's acceptable that 6% of the 300 children born to residents of the Town of Wilmington in 1999 (actual statistic) should be expected to have moderate to severe [not mild] dental fluorosis. That's 18 children in a "one year" age group. If the Town of Wilmington continues to have 300 children added to the rolls in each coming year, the school system should be expected to have [18 x 12] = 216 children in the school system with moderate to severe fluorosis. Of those, how many will have severe fluorosis? One? Two? This is a totally unacceptable tradeoff. This is

taken from a governmental document which purports to support fluoridation. [Note: total fluorosis (mild, moderate and severe) is expected to be 22% of our 300, or 66 of our children per year]

Point in fact: Fluoride also causes crippling skeletal fluorosis. This is not debatable. Just as the EPA ignores fluorosis as a "health effect", the EPA ignores all of the precursor signs of crippling skeletal fluorosis as a "health effect" (such as arthritic pain, rigidity of the spine, and the mal shaping of bones) and recognizes skeletal fluorosis as a "health effect" only at the onset of actual crippling. This means that a person can be experiencing the preliminary signs of crippling skeletal fluorosis, including real arthritic pain and malformation of the skeleton, but the EPA says it's not a "health effect" because you're not crippled yet? How ever absurd this seems to the reader, this is the rational that the EPA has used to raise the MCL from 1.0 mg/L to 4.0 mg/L, allowing the so called "optimum" level of 1.0 mg/L to neatly fit into it's standards.

Why? It would seem that there must be some reason that would cause the U.S. EPA to go against its own scientists and encourage states and municipalities to fluoridate their water supplies in the face of the information given above.

For your consideration, a letter from the EPA, signed by Rebecca Hanmer, Deputy Assistant Administrator for Water, which states:

"Water treatment chemicals, including fluosilicic acid have been evaluated for their potential for contributing to the contamination of drinking water. The Water Treatment Chemicals Codex, published by the National Academy of Sciences, prescribes the purity requirements for fluosilicic acid and other fluoridation chemicals.

In regard to the use of fluosilicic acid as a source of fluoride for fluoridation, this Agency regards such use as an ideal environmental solution to along-standing problem. By recovering by-product fluosilicic acid from fertilizer manufacturing, water and air pollution are minimized, and water utilities have a low-cost source of fluoride available to them."

As the letter states, one motivation for the EPA allowing the disposal of "fluosilicic acid and other fluoridation chemicals" into our water supplies is because it is "an ideal environmental solution to a long-standing problem". Note that "other fluoridation chemicals" would include sodium fluoride which is recommended by the Massachusetts Department of Public Health for the Town of Wilmington. Sodium fluoride is a hazardous waste. produced by the aluminum industry, and hydrofluorosilic acid is a hazardous waste produced by the fertilizer industry.

Sodium fluoride is a very toxic and very reactive chemical. It's toxicity is very well documented. As a by product of industry, it is a hazardous waste, which if properly disposed of would need to be taken to a class 1 landfill at the cost of approximately $7000 per truckload. How convenient for industry, that the EPA was so willing to make these adjustments to the MCL to facilitate the disposal of their hazardous wastes into the drinking water supplies.

It is true that nearly all of the water in any municipal water system never gets consumed. Most goes down the drains of America, washes the cars, waters the lawns, laundry, and so on. What little percentage is consumed, goes through the body and only 50% of the fluoride is absorbed. So what's the problem. The fluoride that is absorbed goes to the teeth and bones. It can cause fluorosis of the teeth and crippling skeletal fluorosis as described above. There are many other mechanisms that have only recently come to light which also go to the issue of whether fluoride is safe.

The very well known research by Dr. Phyllis Mullinex has shown that fluoride causes Central Nervous System disorder in rats. The levels at which the rats were exposed were appropriate for the comparative study of fluoride's effect in humans. Dr. Mullinex was dismissed from her position as chairman of the toxicology department at Forsythe immediately after publishing her work. A subsequent law suit ended in a settlement with sealed results. Forsythe was endowed with a grant from Colgate.

Fluoride has been shown to be an equivocal cause of cancer in rats. There are several other associated disorders that have been positively linked to fluoridated water supplies. Among them are increased occurrence of hip fracture, Down's Syndrome, earlier onset of menses, delayed eruption of teeth, a reduction in IQ of approximately 10 points, and the occasional person who simply happens to be hypersensitive to fluoride because of some other medical reason resulting in an allergic type reaction, and the occasional death or multiple deaths caused by accidental over fluoridation.

And finally on the question of "safety", many have claimed that the AMA endorses or supports fluoridation. I wish to include a quote from a letter from Dr. Flanagan, Assistant Director of the American Medical Association which states in part: .

"this Association endorses the principle of fluoridation of public water supplies to reduce the incidence of dental caries; it does not become involved in endorsement of the fluoridation of water supplies of specific cities.

The American Medical Association is not prepared to state that "no harm will be done to any person by water fluoridation."

The American Medical Association has not carried out any research work, either long-term or short-term, regarding the possibility of any side effects."

The fact is, I have found no study ever being done, not by any governmental agency, or any professional organization, such as the AMA, the ADA, the FDA, or the EPA, not the USPHS or the CDC. No agency has determined that fluoride is safe.

The Second Issue: Is it Effective?

Higher levels of fluoride have been correlated to fewer caries but when fluoride is reduced to a level at which there is a lower risk of fluorosis, the correlation no longer exists. No credible study has produced factual proof that fluoridation of a water supply at the so called "optimum" level is beneficial to dental health.

Virtually all studies comparing fluoridated and non fluoridated communities, or other fluoridated and non-fluoridated populations in subsets not necessarily limited to communities, have shown that there is no difference between the resultant dental condition. In fact, some studies have shown that the non-fluoridated populations have slightly better dental condition than do the fluoridated communities.

Some early studies which are used to support the claim that fluoridated communities have better results are not dependable. Many of the early studies were manipulated to give the false appearance of effectiveness because industry and a willing government was determined to find a solution to their "long-standing problem". This has been documented by sworn court testimony in subsequent civil proceedings. Additionally, many credible studies have been done which show clearly that at the so called "optimum" level, there is no difference in dental condition.

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Finally, there was a very recent paper published in Community Dentistry and Oral Epidemiology by Kumar and Swango, 1999, which demonstrates that the many current sources of fluoride in food products, fruit juices, and other sources today results in excessive fluorosis and concludes with the recommendation of lowering the intake of fluoride, not increasing it.

The Third Issue: Is it Right for the Town of Wilmington?

After reading hundreds of documents, I have found that much of the most condemning information has come from the EPA itself. These are by far the most troubling. No other drug, or medicine has such a wide spread application, and yet has had so little scrutiny as to its safety. To purchase sodium fluoride tables, one is required to first obtain a prescription. Yet to fluoridate an entire community no prescription is necessary.

Sodium fluoride tablets can be purchased by prescription at a local drug store at a cost of $6.99 per hundred (1 tablet per day for 100 days which equals $26/year) and a dentist can prescribe tablets at a regular visit at no additional cost to the parents. If individual parents wish to supply fluoridated water to their children, let them have that freedom of choice. The common complaint is that they forget to give the tablets to their children.

Recommendation:

Fluoride is not a nutrient as many have claimed, only being adjusted to it's "optimum" level. Fluoride is a toxin, like arsenic and lead, occurring naturally in the environment. We have come to discover that many of the elements that we commonly used were dangerous to health. We have removed lead from paint, once a primary ingredient, and from pipes and solder, as we have learned that it caused lead poisoning. We have removed asbestos from our schools and public buildings because of the remote possibility that the smallest exposure could cause asbestosis. We have done this by passing laws prohibiting the use of these elements. We should look at the many countries that have prohibited the use of fluoride in water supplies. We should look at the long list of cities in the U.S. that have changed their position and reversed their previous action to fluoridate, and have rejected its use.

As a society, we have done many things in the name of science and good health, like sulfur drugs and thalidomide, Laetrile and Fen-phen, only to find out that they were wrong and we rejected their use. There is no compelling reason to fluoridate an entire community, adults and children, with a toxic chemical at any dose, in the face of such compelling evidence against it, especially when there are alternatives available.

Therefore my recommendation to the Board of Health and the Town of Wilmington is to not go forward with the fluoridation of the municipal water supply.

Respectfully submitted,

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Comments on
Proposed HHS Recommendation for
Fluoride Concentration in Drinking Water
for Prevention of Dental Caries

Prepared for the
Department of Health and Human Services

February 14, 2011

Submitted at the request of the
International Academy of Oral Medicine and Toxicology (IAOMT)
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These comments on the proposed HHS recommendation for fluoride concentration in drinking water are submitted to the Department of Health and Human Services (HHS) in response to their January 13, 2011, request for public comments (Federal Register 2011). These comments are not to be considered a comprehensive review of fluoride exposure or toxicity.

The author of these comments is a professional in the field of risk analysis, including exposure assessment, toxicity evaluation, and risk assessment. She has recently served on two subcommittees of the National Research Council's Committee on Toxicology that dealt with fluoride exposure and toxicity, including the NRC's Committee on Fluoride in Drinking Water. She has also authored an Environmental Protection Agency report on fluoride toxicity.

These comments are submitted at the request of the International Academy of Oral Medicine and Toxicology (IAOMT), and their preparation was supported in part by the IAOMT. Opinions and conclusions expressed herein are those of the author.

Summary. The Department of Health and Human Services (HHS) has proposed a new recommendation regarding fluoride concentrations in drinking water (Federal Register 2011), the primary change being from a recommended range of 0.7-1.2 mg/L fluoride in drinking water (0.7-1.2 ppm) based on ambient local temperatures, to a single value of 0.7 mg/L (0.7 ppm), regardless of temperature. The main concern is the prevention of dental fluorosis, a condition ranging from mild spotting of the teeth to severe pitting and staining. Dental fluorosis is caused by excessive fluoride ingestion during the early years of childhood, before the permanent teeth erupt. The HHS recommendation is intended to limit the risk of dental fluorosis while maintaining caries protection (Federal Register 2011), something that was suggested earlier by Heller et al. (1997), based on an analysis of a national survey of children conducted in 1986-1987.

The HHS is to be commended for recommending a reduced concentration of fluoride in drinking water. However, several important concerns remain:

(1) The proposed reduction in fluoride concentration in drinking water is insufficient to achieve the HHS goal of a significant reduction in the nationwide prevalence of dental fluorosis.

(2) The HHS recommendation does not address a number of other health concerns for the American population with respect to fluoride exposures.

(3) Available data do not support a role of community water fluoridation in improving dental health.

(4) HHS remains in the precarious position of recommending indiscriminate administration of a drug to the American population, without individual evaluation of need, appropriate dose, efficacy, or side effects.

These concerns are discussed in more detail below. If HHS is serious about limiting the risk of dental fluorosis and protecting the health of the American population, HHS should revise its proposed recommendation to call for an immediate end to community water fluoridation.

(1) The proposed reduction in fluoride concentration in drinking water is insufficient to achieve the HHS goal of a significant reduction in the nationwide prevalence of dental fluorosis.

The previous range of recommended fluoride concentrations was based on ambient temperature and the assumption that people's water consumption varies with outdoor temperature. Thus the recommended fluoride levels vary from 0.7 mg/L in Florida and parts of Arizona and Texas to 1.2 mg/L in Alaska, Maine, Michigan, Minnesota, and North Dakota. The new HHS recommendation will therefore have a larger effect on the northern states than on the southern states, with some areas seeing no change in the recommended fluoride concentration. The proposed change will also put the U.S. in agreement with Canada, which in 2009 recommended a fluoride concentration of 0.7 mg/L for all parts of the country (Health Canada 2009). However, the HHS has offered no data by state or region to show that the northern states have the highest prevalence of dental fluorosis or that reducing the fluoride concentration in northern states will bring the national prevalence of dental fluorosis down to an "acceptable" level. In addition, the Centers for Disease Control and Prevention (CDC) has reported that the black population in the U.S. has higher rates of dental fluorosis, including higher rates of moderate and severe dental fluorosis (CDC 2005). Since the black population is greatest in the southern states, reducing the fluoride concentration in the northern states but not the southern states cannot be expected to produce a significant reduction in dental fluorosis in the black population as a whole.

The 1986-1987 survey data mentioned by HHS (Federal Register 2011) show a clear dose response for fluorosis prevalence and severity with fluoride concentration (Heller et al. 1997; Table 1; Fig. 1). Unfortunately, the more recent 1999-2004 survey did not include water fluoride concentrations. The increase in total fluorosis prevalence (40.7% in 1999-2004 vs. 22.6% in 1986-1987 for children ages 12-15; Beltrán-Aguilar et al. 2010) probably represents both a higher fraction of the population receiving fluoridated water and higher total fluoride exposures for a given water fluoride concentration. Although increased fluoride intake from other sources such as toothpaste could be contributing to higher total fluoride exposures, a simple decrease in milk consumption with higher consequent intake of water and water-based beverages would also contribute to higher fluoride intake for a given water fluoride concentration.

Reducing fluoride concentrations in fluoridated community water will, at best, bring down fluorosis prevalence for that group only to the level experienced by the part of the group with water fluoride already at 0.7 mg/L. For example, based on the 1986-1987 data as reported by Heller et al. (1997), reducing fluoride concentrations in fluoridated community water would bring the fluorosis prevalence for that group down from about 30% (for the 0.7-1.2 mg/L group; Fig. 1) to about 27% (for 0.7 mg/L; see Fig. 2 of the Heller et al. paper), a relatively small decrease. However, elimination of fluoridation entirely, for the whole population, would be expected to bring the fluorosis prevalence down to that of the low-fluoride population (e.g., for the 1986-1987 cohort, from 30% to around 13%; Fig. 1), a much more substantial decrease.

HHS bases its recommendation partly on observations that water consumption among children does not depend greatly on ambient temperature (Federal Register 2011). The National Research Council (NRC 2006) also indicated that for much of the U.S. population, water consumption does not depend on outdoor temperature. However, the NRC also identified a number of population subgroups that have high water consumption, including people with high activity levels (more of a concern for older children and adults) and people with medical conditions such

as diabetes insipidus and diabetes mellitus (applicable to all ages). People in hot areas who are unable to afford air conditioning would be another subgroup of concern (applicable to all ages). Perhaps the most important population subgroup is composed of infants fed formula prepared with tap water. A substantial number of infants have water consumption rates in excess of 0.1 L/kg/day (100 mL per kg body weight per day; NRC 2006; EPA 2004a). At the proposed fluoride concentration of 0.7 mg/L in drinking water, these infants would still have fluoride intakes at and above 0.07 mg/kg/day, and some will exceed 0.15 mg/kg/day (NRC 2006). The only U.S. study to have looked at dental fluorosis and individual fluoride intake at various ages (the Iowa study) reported that for children with fluoride intakes above 0.06 mg/kg/day during the first 3 years of life, fluorosis rates were as high as 50% (Hong et al. 2006b). Eight individuals in the cohort were considered to have severe fluorosis (Hong et al. 2006b); their individual intakes were not reported, so one must assume that they did not necessarily have the highest intakes of the cohort. Thus a large fraction of infants and young children fed formula made with fluoridated tap water can be expected to develop dental fluorosis even at a water fluoride concentration of 0.7 mg/L.

The National Research Council considers severe dental fluorosis to be an adverse health effect and reports the general consensus in the literature that both severe and moderate dental fluorosis should be prevented (NRC 2006). Health Canada (2009) considers moderate dental fluorosis to be an adverse effect. The Iowa study indicates that high fluoride intake during the first 2 years of life is most important with respect to development of dental fluorosis of the permanent maxillary central incisors (the "top front teeth")—the teeth that most affect a person's appearance—although fluoride intake up to at least 4 years old was also important (Hong et al. 2006a). The American Dental Association has issued a brief statement to the effect that parents should not prepare infant formula with fluoridated water if they are concerned about the possibility of their child developing dental fluorosis (ADA 2007). This is an admission that dental fluorosis is undesirable, and that fluoridated tap water is not "safe" for all individuals. The CDC (2005) reports a higher likelihood of moderate and severe fluorosis for minority and low-income children. HHS should remember that while encouraging breastfeeding of infants is certainly appropriate, in many family situations breastfeeding is not possible (e.g., in cases of adoption or of ill-health or death of the mother). Especially since most assistance programs for low-income families do not cover the cost of bottled water or ready-to-feed formula, it is essential that tap water be safe for use in infant formula, without putting infants at increased risk of dental fluorosis.

In summary, the change in recommended water fluoride concentration proposed by HHS, while definitely a step in the right direction, is unlikely to bring about a substantial change in the prevalence of dental fluorosis in the U.S. In particular, infants and young children fed formula made with fluoridated tap water can still be expected to have high rates of dental fluorosis in their permanent teeth. Children with diabetes insipidus or diabetes mellitus and children in hot areas whose homes are not air conditioned can also be expected to have a substantial risk of developing dental fluorosis. To bring about a significant decrease in the prevalence and severity of dental fluorosis, HHS should recommend elimination of community water fluoridation throughout the entire U.S.

(2) The HHS recommendation does not address a number of other health concerns for the American population with respect to fluoride exposures.

The HHS recommendation addresses only dental fluorosis, while ignoring a long list of other health concerns for the U.S. population. Dental fluorosis itself has been associated with increased risks of various adverse health effects, including thyroid disease, lowered IQ, and bone fracture (Alarcón-Herrera et al. 2001; Zhao et al. 1996; Li et al. 1995; Lin et al. 1991; Desai et al. 1993; Yang et al. 1994; Jooste et al. 1999; Susheela et al. 2005). To the best of my knowledge, no studies in the U.S. or Canada have looked for associations between dental fluorosis and risk of other adverse effects. However, the failure to look for adverse health effects does not demonstrate the absence of adverse health effects.

The National Research Council (2006) indicated that the Environmental Protection Agency's (EPA's) present drinking water standards for fluoride (maximum contaminant level goal [MCLG] and maximum contaminant level [MCL], both at 4 mg/L) are not protective of human health, based on preventing severe dental fluorosis, stage II skeletal fluorosis, and increased risk of bone fractures. Given the wide range of water intake within the American population and the presence of other sources of fluoride intake, one can reasonably expect that a "safe" level of fluoride in drinking water would be at least a factor of 10 below the "unsafe" level of 4 mg/L. EPA's MCLG is defined as a "non-enforceable health goal which is set at a level at which no known or anticipated adverse effect on the health of persons occurs and which allows an adequate margin of safety" (EPA 2006). Dental fluorosis, skeletal fluorosis, and increased risk of bone fracture are all reasonably well known and acknowledged adverse health effects from fluoride exposure. However, EPA is also required to consider the "anticipated" adverse effects (which may occur at lower levels of fluoride exposure than the "known" effects) and allow for an adequate margin of safety. HHS should insist that EPA properly consider all of the anticipated adverse health effects, with an adequate margin of safety. The proposed HHS recommendation for water fluoridation at 0.7 mg/L is not adequate to protect against known or anticipated adverse effects and does not allow an adequate margin of safety to protect young children, people with high water consumption, people with kidney disease (resulting in reduced excretion of fluoride), and other potentially sensitive population subgroups.

In addition to the "known" adverse health effects of dental fluorosis, skeletal fluorosis, and increased risk of bone fracture, "anticipated" adverse health effects from fluoride exposure or community water fluoridation include (but are not limited to) carcinogenicity, genotoxicity, endocrine effects, increased blood lead levels, and hypersensitivity (reduced tolerance) to fluoride. These effects (described in more detail below) are not as well studied as the dental and skeletal effects, which should indicate that a greater margin of safety is necessary to ensure protection of the population—"in the face of uncertain evidence it is important to act in a manner that protects public health" (Tickner and Coffin 2006). In addition, it should be noted that some of these effects may occur at lower fluoride exposures than those typically associated with dental or skeletal effects, such that protection against the dental or skeletal effects does not necessarily ensure protection against other anticipated adverse health effects. Again, elimination of community water fluoridation is the best way to reduce fluoride exposures to a level at which adverse health effects are unlikely.

A few comments regarding the interpretation of the available fluoride studies may be helpful. As Cheng et al. (2007) have described, a "negative" study may simply mean that the study was not

sufficiently sensitive to demonstrate a moderate (as opposed to large) effect. This is often due to use of too small a sample size. In addition, study populations are often grouped by community, water source, or fluoride concentration in the water, rather than by individual intake. Due to the wide variation in drinking water intake, this approach results in study groups with overlapping intakes and makes it difficult to detect dose response relationships that do in fact exist.

The few studies that have looked at age-dependent exposure to fluoride have found increased risks of adverse effects (e.g., Bassin et al. 2006 for osteosarcoma; Danielson et al. 1992 for hip fracture risk); studies that have not looked at age-dependent exposure cannot be assumed to provide evidence of no effect. Similarly, studies that have used a measure of current exposure where a cumulative measure would be more appropriate, or vice versa, cannot be assumed to demonstrate lack of an effect.

Studies of fluoride toxicity in laboratory animals are sometimes dismissed as irrelevant because the exposures or fluoride concentrations used were higher than those expected for humans drinking fluoridated tap water. It is important to know that animals require much higher exposures (5-20 times higher, or more; see NRC 2006; 2009) than humans to achieve the same effects or similar fluoride concentrations in bone or serum. In other words, humans are considerably more sensitive to fluoride than are most animal species that have been studied.

A number of adverse health effects can be expected to occur in at least some individuals when estimated average intakes of fluoride are around 0.05 mg/kg/day or higher (NRC 2006; 2009). For persons with iodine deficiency, average intakes as low as 0.01-0.03 mg/kg/day could produce effects (NRC 2006). The next few sections briefly summarize some (not all) of the adverse health effects, known and anticipated, that should be considered in any reevaluation of the drinking water standards for fluoride. Most of these effects have been reviewed in detail by the NRC (2006), although the NRC did not specifically evaluate health risks over the whole range of fluoride intakes or attempt to identify a "safe" level of fluoride exposure.

Skeletal fluorosis

Bone fluoride concentrations in the ranges reported for stage II and III skeletal fluorosis will be reached by long-term fluoride exposures of 0.05 mg/kg/day or higher (estimated from NRC 2006). Bone fluoride concentrations, radiologic changes, and symptoms are not clearly correlated (Franke et al. 1975), and most U.S. studies do not categorize cases by stage. Recent case reports include fluorosis attributed to excessive ingestion of tea or toothpaste (Whyte et al. 2005; Hallanger Johnson et al. 2007; Kurland et al. 2007). Most of the literature addresses high fluoride exposures over a few years; there has been essentially no investigation of effects of low exposures over many years and no effort to identify fluorosis of any stage in the U.S. "Arthritis" (defined as painful inflammation and stiffness of the joints) is the leading cause of disability in the U.S., currently affects at least 46 million adults in the U.S. (including 50% of the population > 65 years old), and is expected to affect 67 million adults in the U.S. by 2030 (CDC 2006). The possibility that a sizeable fraction of "bone and joint pain" or "arthritis" in U.S. adults is attributable to fluoride exposure has not been addressed, although it is plausible, given what is known about fluoride intakes.

Increased risk of bone fractures

The NRC (2006) concluded that lifetime exposure to fluoride at an estimated average daily intake of 0.08 mg/kg/day (average adult fluoride intake with water at 4 mg/L) is likely to result in higher bone fracture rates, and the available information suggests an increased likelihood of bone fracture for daily fluoride intakes of 0.05 mg/kg/day (average adult fluoride intake at 2 mg/L). The Agency for Toxic Substances and Disease Registry (ATSDR) has identified a chronic-duration Minimal Risk Level (MRL) for oral exposure to fluoride of 0.05 mg/kg/day, based on an increased risk of bone fracture (ATSDR 2003). The NRC's findings (NRC 2006) indicate that the ATSDR's MRL is not protective enough. The available studies consider fluoride intake only in terms of the concentration in the local drinking water, and most use fluoridated water (1 mg/L, corresponding to an average daily intake of 0.03 mg/kg/day for adults) as a control. Thus there is probably considerable overlap in exposures between groups, making effects more difficult to distinguish, and the entire dose response range of interest has not been well studied. The findings in humans are consistent with animal studies that have found increased brittleness of bones with increased fluoride exposure (Clark and Mann 1938; Turner et al. 1997; 2001).

Danielson et al. (1992) reported an increased relative risk for hip fracture in a fluoridated area of 1.27 (95% CI 1.08-1.46) for women and 1.41 (95% CI 1.00-1.81) for men. These authors reported a difference between women exposed to fluoride prior to menopause and those exposed afterwards. For women exposed prior to menopause, the fracture risk was considerably higher than for those not exposed to fluoride. Many studies of fracture risk have not looked at age-specific exposure, or have involved women exposed only after menopause, when fluoride uptake into bone is probably substantially lower.

The Iowa study reported effects on bone mineral concentration and bone mineral density with average childhood fluoride intakes of 0.02-0.05 mg/kg/day (Levy et al. 2009). Linear correlation between dental fluorosis and risk of bone fracture has been reported for children and adults (Alarcón-Herrera et al. 2001). Bone fracture rates in children in the U.S. may be increasing (e.g., Khosla et al. 2003), but fluoride exposure has not been examined as a possible cause or contributor.

Carcinogenicity

Three U.S. courts have found water fluoridation to be injurious to human health, specifically that it may cause or contribute to the cause of cancer and genetic damage (described in detail by Graham and Morin 1999). The NRC's committee on fluoride toxicology unanimously concluded that "Fluoride appears to have the potential to initiate or promote cancers," even though the overall evidence is "mixed" (NRC 2006). Referring to the animal studies, the committee also said that "the nature of uncertainties in the existing data could also be viewed as supporting a greater precaution regarding the potential risk to humans." The committee discussed the limitations of epidemiologic studies, especially ecologic studies (those in which group, rather than individual, measures of exposure and outcome are used), in detecting small increases in risk—in other words, the studies are not sensitive enough to identify small increases in cancer risk; therefore a "negative" study does not necessarily mean that there is no risk (see also Cheng et al. 2007).

While the NRC did not assign fluoride to a specific category of carcinogenicity (i.e., known, probable, or possible), the committee did not consider either "insufficient information" or "clearly not carcinogenic" to be applicable. The committee report (NRC 2006) includes a discussion of how EPA establishes drinking water standards for known, probable, or possible carcinogens; such a discussion would not have been relevant had the committee not considered fluoride to be carcinogenic. The question becomes one of how strongly carcinogenic fluoride is, and under what circumstances.

The case-control study by Bassin et al. (2006) is the only published study thus far to have looked at age-dependent exposure to fluoride. This study reported a significantly elevated risk of osteosarcoma in boys as a function of estimated age-specific fluoride intake. Osteosarcoma is a bone cancer that commonly results in amputation of an affected limb and may result in death. At the very least, this study indicates that similar studies of pediatric osteosarcoma that have not looked at age-dependent intake cannot be considered to show "no effect."

While a few other studies (e.g., Gelberg et al. 1995) have looked at individual fluoride exposure (as opposed to group or ecologic measures of exposure), these have looked at total fluoride exposure until time of diagnosis or treatment. Given that there is a "lag time" of a few years between onset of a cancer and its diagnosis, use of cumulative fluoride exposure until time of diagnosis is potentially misleading, as fluoride exposure during the last several years (during the "lag time") cannot have contributed to the initiation of a cancer but could have a significant effect on the estimate of cumulative fluoride exposure.

The 1990 National Toxicology Program (NTP) study on sodium fluoride officially concluded that "there was *equivocal evidence of carcinogenic activity* of sodium fluoride in male F344/N rats, based on the occurrence of a small number of osteosarcomas in dosed animals" (NTP 1990; italics in the original). According to the published report, a "small number of osteosarcomas occurred in mid- and high-dose male rats. These neoplasms occurred with a significant dose response trend, but at a rate within the upper range of incidences previously seen in control male rats in NTP studies" (NTP 1990). It is important to realize that the historic controls from previous studies had not had the special low-fluoride diet used for this study, and therefore more properly constitute a low- to mid-range exposed group rather than a control group. This and other concerns were described in a memo within the Environmental Protection Agency (Marcus 1990) and reported in the press (Hileman 1990). These concerns and the testimony before the U.S. Senate of the union representing EPA scientists (Hirzy 2000) should be taken seriously by the HHS.

In humans, osteosarcomas tend to occur most commonly in young people (pediatric cases) or the very old (adult or geriatric cases), with a higher incidence in males than in females (Bassin et al. 2006). Sergi and Zwerschke (2008) indicate that 60-75% of cases are in patients between 15 and 25 years old. In the NTP 2-year study, fluoride exposure was begun when the animals were 6 weeks old, as is typical for NTP and similar studies (Hattis et al. 2004). Puberty in the rat typically occurs at about 32 days of age in females and 42 days in males (e.g., Gray et al., 2004; Evans 1986). Thus, the age of 6 weeks in the NTP study probably corresponds to pubertal or post-pubertal animals. The cases of osteosarcoma in the rats were reported in the late stages of the test, and probably corresponded to geriatric osteosarcomas in humans. In Bassin's study, the age range for which the fluoride-osteosarcoma association was most apparent was for exposures at ages 4-12 years, with a peak for exposures at age 6-8 years (Bassin et al. 2006). Very likely,

the fluoride exposures in most of the animal studies have started after the age corresponding to the apparent most susceptible age in humans, and thus these animal studies may have completely missed the most important exposure period with respect to initiation of the majority of human osteosarcomas. Therefore, this animal study cannot be interpreted as showing no evidence of causation for pediatric osteosarcoma, although, properly interpreted, it does show evidence for causation of geriatric osteosarcoma.

Genotoxicity

Genotoxicity, or the ability to damage the genetic material (genes and chromosomes) of cells, is considered indicative of potential carcinogenicity. A number of mammalian *in vitro* systems have shown dose-dependent cytogenetic or cell transformational effects from fluoride exposure (reviewed by NRC 2009). Several reports suggest an indirect or promotional mechanism, e.g., inhibition of DNA synthesis or repair enzymes, rather than a direct mutagenic effect (Lasne et al. 1988; Aardema et al. 1989; Aardema and Tsutsui 1995; Meng and Zhang 1997). Human cells seem to be much more susceptible to chromosome damage from fluoride than are rodent cells (Kishi and Ishida 1993).

A recent paper by Zhang et al. (2009) describes a new testing system for potential carcinogens, based on induction of a DNA-damage response gene in a human cell line. Sodium fluoride tests positive in this system, as do a number of other known carcinogens, representing a variety of genotoxic and nongenotoxic carcinogenic mechanisms. Known noncarcinogens—chemicals not associated with carcinogenicity—did not test positive. The system described by Zhang et al. (2009) is considerably more sensitive than the older systems for most chemicals examined; a positive effect was seen at a fluoride concentration of about 0.5 mg/L, or a factor of 10 lower than in other systems.

A fluoride concentration of 0.5 mg/L in urine will routinely be exceeded by many people consuming fluoridated water (NRC 2006); for people with substantial fluoride intake, serum fluoride concentrations may also reach or exceed 0.5 mg/L. Acute fluoride exposures (e.g., accidental poisoning, fluoride overfeeds in drinking water systems) have resulted in fluoride concentrations in urine well in excess of 5 mg/L in a number of cases (e.g., Penman et al. 1997; Björnhagen et al. 2003; Vohra et al. 2008). Urine fluoride concentrations can also exceed 5 mg/L if chronic fluoride intake is above about 5-6 mg/day (0.07-0.09 mg/kg/day for an adult; based on NRC 2006). Thus, kidney and bladder cells are probably exposed to fluoride concentrations in the ranges at which genotoxic effects have been reported *in vitro*, especially when the more sensitive system of Zhang et al. (2009) is considered. Based on the results of Zhang et al. (2009), most tissues of the body are potentially at risk if serum fluoride concentrations reach or exceed 0.5 mg/L. In addition, cells in the vicinity of resorption sites in fluoride-containing bone are potentially exposed to very high fluoride concentrations in extracellular fluid (NRC 2006) and thus are also at risk for genotoxic effects.

Endocrine effects

The NRC (2006) concluded that fluoride is an endocrine disruptor. Endocrine effects include altered thyroid function or increased goiter prevalence (at fluoride intakes of 0.05-0.1 mg/kg/day,

or 0.01-0.03 mg/kg/day with iodine deficiency), impaired glucose tolerance (at fluoride intakes above 0.07 mg/kg/day), a decrease in age at menarche in girls in fluoridated towns, and disruptions in calcium metabolism (calcitonin and parathyroid function, at fluoride intakes of 0.06-0.15 mg/kg/day or higher). ATSDR's toxicological profile for fluoride (ATSDR 2003) refers to an animal study of thyroid function that would give a lower MRL (value not given) than the MRL derived for bone fracture risk (0.05 mg/kg/day).

Thyroid dysfunction and Type II diabetes presently pose substantial health concerns in the U.S. (NRC 2006). Of particular concern is an inverse correlation between subclinical maternal hypothyroidism and the IQ of the offspring. In addition, maternal subclinical hypothyroidism has been proposed as a cause of or contributor to development of autism in the child (Román 2007; Sullivan 2009). Steingraber (2007) has described the decrease in age at puberty of U.S. girls and the associated increased risk of breast cancer. Calcium deficiency induced or exacerbated by fluoride exposure may contribute to other health effects (NRC 2006).

Increased blood lead levels

An increased likelihood of elevated blood lead levels is associated with use of silicofluorides (usually H_2SiF_6 or Na_2SiF_6) as the fluoridating agent (NRC 2006; Coplan et al. 2007). Approximately 90% of people on fluoridated water are on systems using silicofluorides (NRC 2006). The chemistry and toxicology of these agents, especially at low pH (e.g., use of fluoridated water in beverages such as tea, soft drinks, or reconstituted fruit juices), have not been adequately studied (NRC 2006). Associations between silicofluoride use and biological effects in humans have been reported, in particular, elevated levels of blood lead in children and inhibition of acetylcholinesterase activity (reviewed by Coplan et al. 2007). A recent study in rats found significantly higher concentrations of lead in both blood and calcified tissues of animals exposed to both silicofluorides and lead (Sawan et al. 2010).

In addition to biological effects of silicofluorides, the interaction of silicofluorides (as the fluoridating agent) and disinfection agents (specifically, chloramines) also increases the leaching of lead from plumbing fixtures into drinking water (Maas et al. 2005; 2007). A recent Congressional investigation discussed the failure of the CDC to publicize information about high lead levels in drinking water and children's blood in Washington, D.C. (Leonnig 2010). The interaction of silicofluorides and chloramines is the probable explanation for the high lead levels (Maas et al. 2005; 2007). EPA considers lead to be a probable human carcinogen and to have no practical threshold with respect to neurotoxicity (EPA 2004b)—in other words, there is considered to be no safe level of lead exposure, and the MCLG for lead is zero (EPA 2006a).

Additional adverse health effects

Fluoride intake is likely to affect the male reproductive-hormone environment, beginning at intakes of around 0.05 mg/kg/day (reviewed by NRC 2009). A "safe" intake with respect to male reproductive effects is probably somewhere below 0.03 mg/kg/day.

The NRC has reviewed the possible association between exposure to fluoridated water (approximately 0.02 mg/kg/day for adults) and increased risk of Down syndrome (trisomy 21) in children of young mothers, discussed a possible mechanism, and recommended further study

(NRC 2006). Fetuses with Down syndrome are less likely to survive to birth, due both to higher natural fetal loss and to a high rate of pregnancy termination (Buckley and Buckley 2008; Forrester and Merz 1999; Siffel et al. 2004; Biggio et al. 2004).

Hypersensitivity or reduced tolerance to fluoride has been reported for exposure to fluoridated water (approximately 0.02 mg/kg/day for adults) or use of fluoride tablets (approximately 1 mg/day). Symptoms include skin irritation, gastrointestinal pain and symptoms (nausea, vomiting, diarrhea, constipation), urticaria, pruritus, stomatitis, chronic fatigue, joint pains, polydipsia, headaches, and other complaints (Waldbott 1956; 1958; Feltman and Kosel 1961; Grimbergen 1974; Petraborg 1977; Spittle 2008; reviewed by NRC 2006). Patients were often unaware that their drinking water contained fluoride. Symptoms improved with avoidance of fluoridated water and recurred with consumption of fluoridated water or with experimental challenge with sodium fluoride. Double-blind tests of patients have confirmed hypersensitivity to fluoride (Grimbergen 1974; Waldbott 1956; 1958). Many of the observed symptoms represent true allergic phenomena, while others (e.g., gastrointestinal symptoms) could be due to a lower level of tolerance for fluoride (intoxication at lower exposure; Waldbott 1956; 1958).

(3) Available data do not support a role of community water fluoridation in improving dental health.

HHS continues to consider community water fluoridation to be important in the prevention of dental caries (Federal Register 2011). However, the question of whether water fluoridation actually produces a benefit requires further attention.

The University of York has carried out perhaps the most thorough review to date of human studies on effects of fluoridation. Their work (McDonagh et al. 2000) is cited by HHS and others as showing the safety and efficacy of water fluoridation, but it actually does neither (Wilson and Sheldon 2006; Cheng et al. 2007). The report mentions a surprising lack of high quality studies demonstrating benefits, and also finds little evidence that water fluoridation reduces socioeconomic disparities:

> Given the level of interest surrounding the issue of public water fluoridation, it is surprising to find that little high quality research has been undertaken. (McDonagh et al. 2000)
>
> Water fluoridation aims to reduce social inequalities in dental health, but few relevant studies exist. The quality of research was even lower than that assessing overall effects of fluoridation. (Cheng et al. 2007)
>
> Evidence relating to reducing inequalities in dental health was both scanty and unreliable. (Wilson and Sheldon 2006)

The apparent benefit is modest, about a 15% difference in the proportion of caries-free children (McDonagh et al. 2000). The American Dental Association (2005) states that "water fluoridation continues to be effective in reducing dental decay by 20-40%," which would translate to less than 1 decayed, missing, or filled permanent tooth (DMFT) in older children and adolescents (based on U.S. data from CDC 2005).

Neither McDonagh et al. (2000) nor the ADA (2005) mentions that fluoride exposure appears to delay the eruption of permanent teeth, although this has been known since the 1940s (Short 1944; NRC 2006). A delay in tooth eruption alters the curve of caries rates with respect to age and complicates the analysis of age-specific caries rates (Psoter et al. 2005; Alvarez 1995; Alvarez and Navia 1989). Komárek et al. (2005) have calculated that the delay in tooth eruption due to fluoride intake may explain the apparent reduction in caries rates observed when comparisons are made at a given age, as is usually done.

Most studies of benefits of fluoride intake or fluoridation have failed to account for a number of important variables, including individual fluoride intakes (as opposed to fluoride concentrations in the local water supplies), sugar intake, socioeconomic variables, and the general decline in caries rates over the last several decades, independent of water fluoridation status. When World Health Organization data on oral health of children in various countries are compared, similar declines in caries over time are seen in all developed countries, regardless of fluoridation status (Cheng et al. 2007; Neurath 2005).

The only peer-reviewed paper to be published from California's major oral health survey in the 1990s reported no association between fluoridation status and risk of early childhood caries (Shiboski et al. 2003). The paper did not address other types of caries.

A number of sources (reviewed by NRC 2006), including the CDC (2001), indicate that any beneficial effect of fluoride on teeth is topical (e.g., from toothpaste), not from ingestion. Featherstone (2000) describes mechanisms by which topical fluoride has an anti-caries effect and states that "[f]luoride incorporated during tooth development [i.e., from ingested fluoride] is insufficient to play a significant role in caries protection." Also:

> The fluoride incorporated developmentally—that is, systemically into the normal tooth mineral—is insufficient to have a measureable effect on acid solubility. (Featherstone 2000)
>
> The prevalence of dental caries in a population is not inversely related to the concentration of fluoride in enamel, and a higher concentration of enamel fluoride is not necessarily more efficacious in preventing dental caries. (CDC 2001)

Fluoride concentrations in drinking water or saliva are too low to be contributing significantly to a topical anti-caries effect, especially since most drinking water is not "swished" around the teeth before being swallowed. CDC (2001) states that "The concentration of fluoride in ductal saliva, as it is secreted from salivary glands, is low—approximately 0.016 parts per million (ppm) in areas where drinking water is fluoridated and 0.006 ppm in nonfluoridated areas. This concentration of fluoride is not likely to affect cariogenic activity."

The single study that has examined caries experience in relation to individual fluoride intakes at various ages during childhood (the Iowa study) has found no association between fluoride intake and caries experience; caries rates (% of children with or without caries) at ages 5 and 9 were similar for all levels of fluoride intake (Warren et al. 2009). The authors state that "the benefits of fluoride are mostly topical" and that their "findings suggest that achieving a caries-free status may have relatively little to do with fluoride *intake*" (emphasis in the original). Most of the children with caries had "relatively few decayed or filled surfaces" (Warren et al. 2009). The authors' main conclusion:

> Given the overlap among caries/fluorosis groups in mean fluoride intake and extreme variability in individual fluoride intakes, firmly recommending an "optimal" fluoride intake is problematic. (Warren et al. 2009).

The national data set collected in the U.S. in 1986-1987 (more than 16,000 children, ages 7-17, with a history of a single continuous residence) shows essentially no difference in caries rates in the permanent teeth of children with different water fluoride levels (Table 1; Fig. 2; data obtained from Heller et al. 1997; similar data can be obtained from Iida and Kumar 2009). Analysis in terms of mean DMFS (decayed, missing, or filled tooth surfaces) for the group (Fig. 3), as opposed to caries prevalence, shows an apparent 18% decrease between the low-fluoride (< 0.3 mg/L) and fluoridated (0.7-1.2 mg/L) groups. In absolute terms, this is a decrease of about 1/2 (0.55) of one tooth surface per child. One possible explanation is delayed tooth eruption, which was not considered in the study. Note that the mean DMFS for the highest fluoride group is higher than for either of the two intermediate groups, also indicating that DMFS scores are not solely a function of water fluoride concentration. When the data are examined by the distribution of DMFS scores (Fig. 4), no real difference in caries experience with respect to water fluoride concentration is observed.

The available data, responsibly interpreted, indicate little or no beneficial effect of water fluoridation on oral health. The HHS should not assume or suppose beneficial effects of community water fluoridation and should not continue to support or encourage water fluoridation, even at the proposed level of 0.7 mg/L.

(4) HHS remains in the precarious position of recommending indiscriminate administration of a drug to the American population, without individual evaluation of need, appropriate dose, efficacy, or side effects.

The U.S. Food and Drug Administration (FDA) considers fluoride in toothpaste to be a non-prescription drug (e.g., FDA undated-a; undated-b) and fluoride "supplements" (usually tablets or lozenges) to be prescription drugs (e.g., Medline Plus 2008). The goal of community water fluoridation is to provide a dental health benefit to individuals and to the population generally (Federal Register 2010), and EPA's recent reference (Federal Register 2010) to a "treated population" acknowledges this use of drinking water systems to deliver a drug to entire populations. This in effect puts local governments and water treatment personnel in charge of administering a chemical (i.e., a drug) to the population in an effort to improve individual and population health (Cross and Carton 2003; Cheng et al. 2007). In this context, HHS should be aware that many people consume more fluoride from tap water than from either non-prescription (toothpaste) or prescription (tablets or lozenges) fluoride sources, without any monitoring for either efficacy or side effects, without the "drug information" or warning labels generally provided for drugs, and without any semblance of informed consent.

In addition, most fluoridation operations use fluorosilicates (usually H_2SiF_6 or Na_2SiF_6) rather than sodium fluoride (NaF). As described above, the chemistry and toxicology of these compounds have not been adequately studied, although important differences in biological effects between silicofluorides and simple fluorides (e.g., NaF) have been reported (Coplan et al. 2007; NRC 2006; Masters et al. 2000; Masters and Coplan 1999). HHS no doubt is aware of the variety of effects (both targeted effects and side effects) associated with fluoride-containing,

FDA-approved, pharmaceutical agents (e.g., Prozac, Sevoflurane, Fenfluramine). The NRC (2006) discussed the increased toxicity of aluminofluorides and beryllofluorides vs. fluoride alone, as well as the different mechanisms of action of the different chemical combinations. Thus it is irresponsible for HHS to recommend addition of fluoride, or a particular concentration of fluoride to be added, without a comprehensive review of the substances (H_2SiF_6 or Na_2SiF_6,) that are actually added. HHS should also be aware that fluoridation chemicals often contain impurities such as lead and arsenic, for which EPA has set MCLGs of zero (EPA 2006), such that a water supplier is actually adding contaminants for which the ideal maximum amount in drinking water is zero.

HHS refers to a plan to "enhance surveillance of dental caries, dental fluorosis, and fluoride intake" (Federal Register 2011). CDC conducts periodic biomonitoring studies (e.g., CDC 2009), in which a number of chemicals in blood or urine of members of the U.S. population are measured. The HHS has made no mention of whether fluoride will be added to the list of chemicals, although this was recommended by the National Research Council (NRC) in 2006 and would be an obvious action to take if CDC or HHS is truly interested in knowing how much fluoride exposure is received by the American public.

In summary, HHS should not continue to promote or encourage uncontrolled exposure of the U.S. population to a drug that, at best, is not appropriate for many individuals (e.g., those who do not want it, those whose water consumption is high, formula-fed infants) and for which the risks are inadequately characterized and inadequately disclosed to the public. HHS should act in the interest of public health by eliminating community water fluoridation in the U.S. at the earliest possible date.

Table 1. Caries prevalence and fluorosis prevalence with water fluoride concentration.[a]

Water fluoride concentration mg/L	Children with no caries %	Mean DMFS score[b]	Children with fluorosis[c] %	Mean severity of fluorosis[d]
< 0.3	53.2	3.08	13.5	0.30
0.3 - < 0.7	57.1	2.71	21.7	0.43
0.7 - 1.2	55.2	2.53	29.9	0.58
> 1.2	52.5	2.80	41.4	0.80

[a] Data for permanent teeth of children ages 5-17 (caries experience and DMFS score) or 7-17 (dental fluorosis), with a history of a single residence, from Tables 2 and 5 of Heller et al. (1997).
[b] Decayed, missing, or filled tooth surfaces (permanent teeth).
[c] Includes very mild, mild, moderate, and severe fluorosis, but not "questionable."
[d] Dean's Community Fluorosis Index.

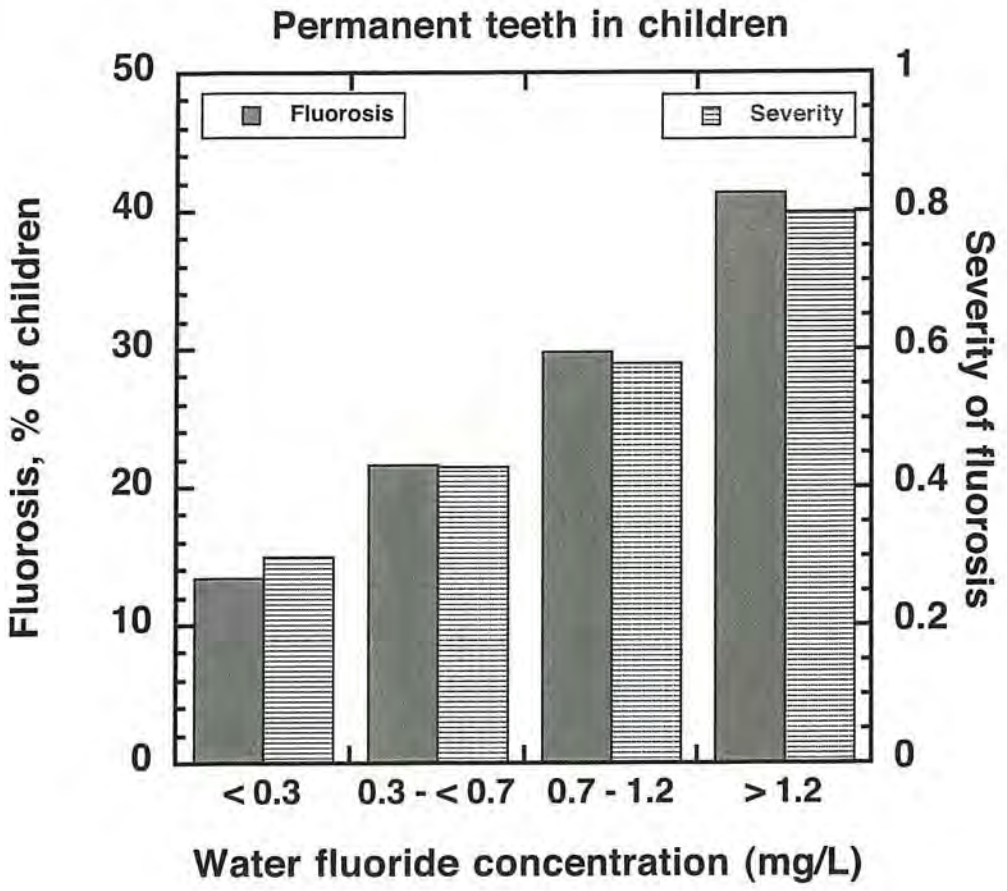

Fig. 1. Fluorosis prevalence and severity with water fluoride concentration for children ages 7-17 with a history of a single continuous residence. Data are shown as (left) % of total children having fluorosis (very mild, mild, moderate, or severe, but not questionable) or (right) severity of fluorosis by Dean's Community Fluorosis Index. Numerical values are provided in Table 1 of these comments and were obtained from Table 5 of Heller et al. (1997).

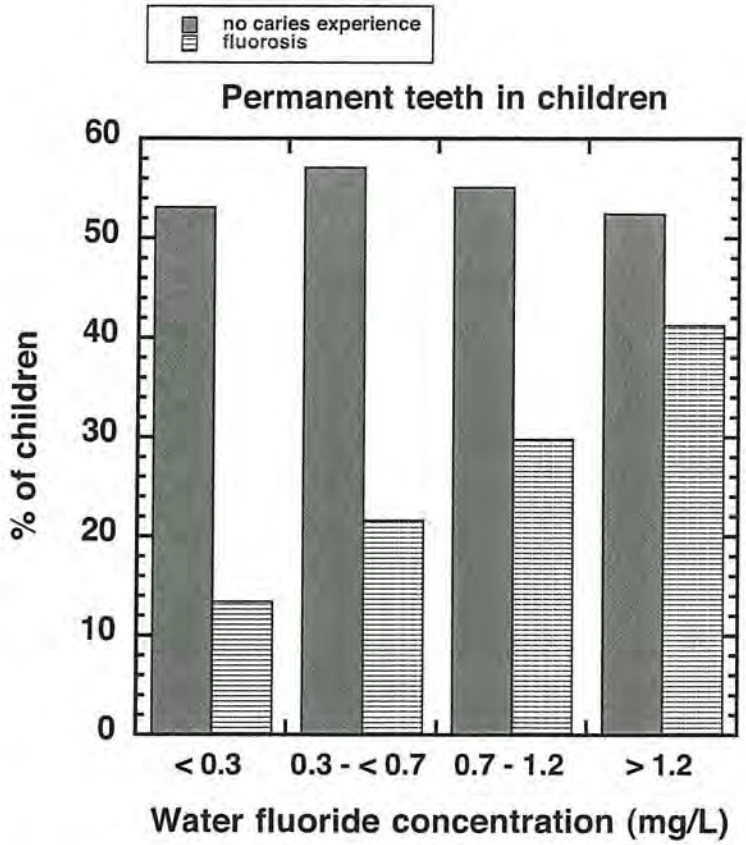

Fig. 2. Percent of children with no caries experience in the permanent teeth (DMFS = 0) and with fluorosis, with respect to water fluoride concentration. Data are shown as % of total children having no caries experience (blue) or having fluorosis (very mild, mild, moderate, or severe, but not questionable; red). Numerical values are provided in Table 1 of these comments and were obtained from Tables 2 and 5 of Heller et al. (1997).

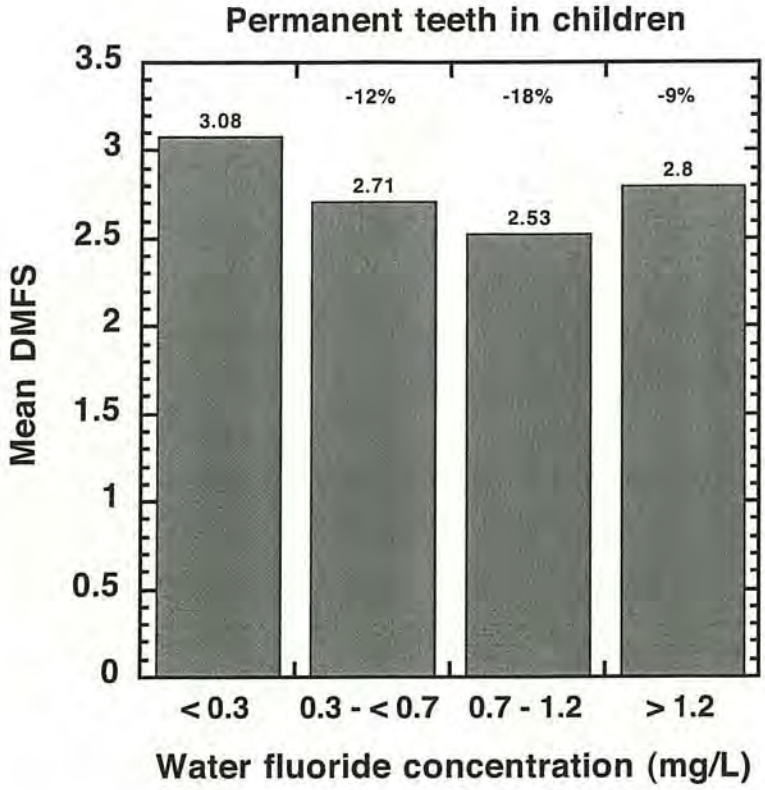

Fig. 3. Mean DMFS score (decayed, missing, or filled permanent tooth surfaces in permanent teeth), with respect to water fluoride concentration. Numerical values are provided in Table 1 of these comments and were obtained from Table 2 of Heller et al. (1997). The percent difference with respect to the lowest fluoride group is also provided.

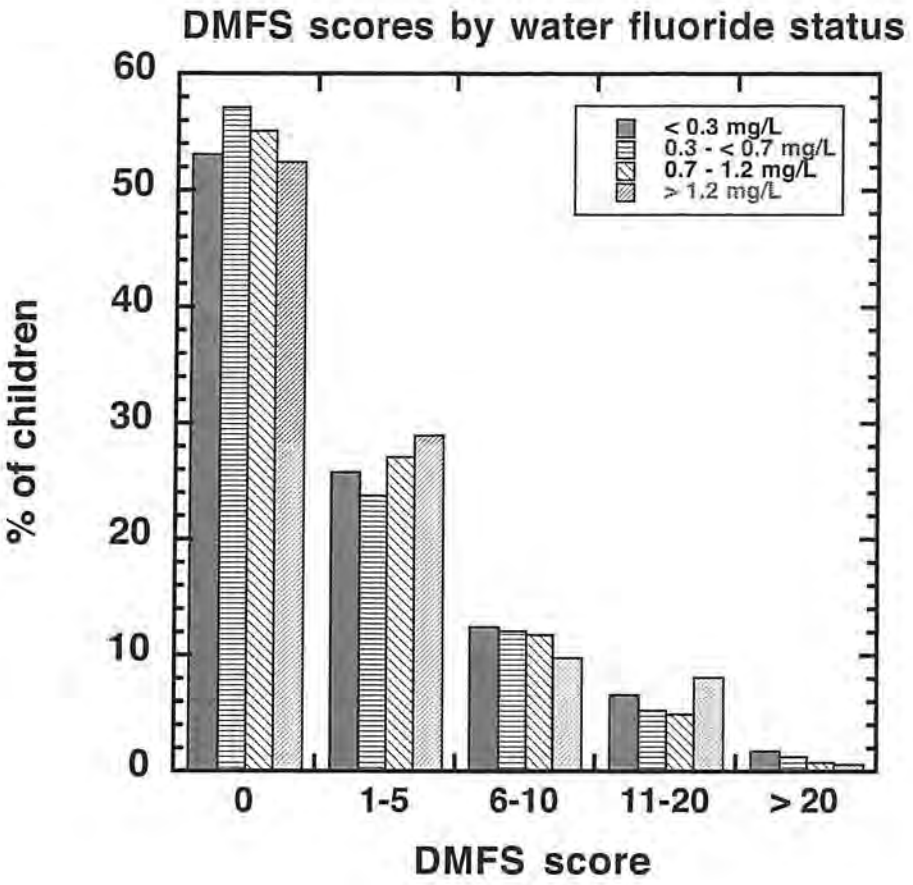

Fig. 4. Percent of children by DMFS score, with respect to water fluoride concentration. Data are shown as % of total children in a given group according to the number of decayed, missing, or filled tooth surfaces in the permanent teeth (DMFS). Data were obtained from Table 2 of Heller et al. (1997).

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Should Natick Fluoridate?

A Report to the Town and the Board of Selectmen
Prepared by the Natick Fluoridation Study Committee
13 E. Central Street, Town of Natick, MA October 23, 1997

Cover Letter to the Selectmen

Fluoridation Study Committee
Town of Natick, Town Hall
13 E. Central St.
Natick MA 01760

Benedict J.Gallo, Ph. D.
Jason Kupperschmidt, B.
Norman R. Mancuso, Ph.D.
Alfred J. Murray, M. S. T.
Harlee S. Strauss, Ph. D.
Chairman

27 September, 1997
Jay H. Ball, Clerk
Office of the Board of Selectmen
Town Hall
13 East Central Street
Natick, MA 01760

Dear Selectman Ball:

This letter accompanies transmission of the report ***"Should Natick Fluoridate?"*** prepared jointly by the Fluoridation Study Committee of the Town of Natick. A summary version of this report is being prepared and copies will be provided as soon as it has been completed. Please advise if we can be of further service to the Board of Selectmen.

Sincerely,

☒

Norman R. Mancuso, Ph. D.
Chairman, Natick Fluoridation Study Committee

Findings, Conclusion, and Recommendations of the Natick Fluoridation Study Committee

Introduction

This statement of Findings, Conclusion and Recommendation specifically addresses the following question of the Board of Selectmen:

On the basis of the documentation provided to you by the proponents and opponents, do you

believe that the potential side effects associated with fluoridating Natick's public water supply outweigh the potential benefits?

Findings The Natick Fluoridation Study Committee conducted a thorough review of the scientific literature and made the following findings regarding the benefits and risks of water fluoridation.

- **Recent studies of the incidence of cavities in children show little to no difference between fluoridated and non-fluoridated communities.**
- **Ten to thirty percent (10-30%) of Natick's children will have very mild to mild dental fluorosis if Natick fluoridates its water (up from probably 6% now). Approximately 1% of Natick's children will have moderate or severe dental fluorosis. Dental fluorosis can cause great concern for the affected family and may result in additional dental bills. It should not be dismissed as a "cosmetic" effect.**
- **Fluoride adversely effects the central nervous system, causing behavioral changes and cognitive deficits. These effects are observed at fluoride doses that some people in the US actually receive.**
- **There is good evidence that fluoride is a developmental neurotoxicant, meaning that fluoride effects the nervous system of the developing fetus at doses that are not toxic to the mother. The developmental neurotoxicity would be manifest as lower IQ and behavioral changes.**
- **Water fluoridation shows a positive correlation with increased hip fracture rates in persons 65 years of age and older, based on two recent epidemiology studies.**
- **Some adults are hypersensitive to even small quantities of fluoride, including that contained in fluoridated water. At least one such person is a Natick resident.**
- **The impact of fluoride on human reproduction at the levels received from environmental exposures is a serious concern. A recent epidemiology study shows a correlation between decreasing annual fertility rate in humans and increasing levels of fluoride in drinking water.**
- **Animal bioassays suggest that fluoride is a carcinogen, especially for tissues such as bone (osteosarcoma) and liver. The potential for carcinogenicity is supported by fluoride's genotoxicity and pharmacokinetic properties. Human epidemiology studies to date are inconclusive, but no appropriate major study has been conducted.**
- **Fluoride inhibits or otherwise alters the actions of a long list of enzymes important to metabolism, growth, and cell regulation.**
- **Sodium fluorosilicate and fluorosilicic acid, the two chemicals Natick intends to use to fluoridate the water supply, have been associated with increased concentrations of lead in tap water and increased blood lead levels in children, based on case reports and a new, as-yet-unpublished study.**
- **If Natick fluoridates its water supply at the proposed level, most children under**

the age of three will daily receive more fluoride than is recommended for them.

The scientific literature supporting these findings is summarized in the full report which also discusses a variety of non-health related concerns that have been raised about water fluoridation

Conclusion

The Committee reached the firm conclusion that the risks of overexposure to fluoride far outweigh any current benefit of water fluoridation.

Recommendations

1. The Natick Fluoridation Study Committee unanimously and emphatically recommends that the town of Natick NOT fluoridate the town water supply.

2. The Natick Fluoridation Study Committee unanimously and emphatically recommends that the Board of Selectmen take appropriate action to ensure that fluoridation of the town water supply does not take place.

GO TO FULL REPORT -- natick.htm (239K)

Home

Fluoride: Protected Pollutant or Panacea?
Are the claimed benefits of ingesting fluoride over-rated
and the risks to our health and eco-system under-reported?

Abstracts

Bones | Calgary | Cavities | Fertility | Cancer | Health risks | Neurological | Dental Fluorosis and Pictures
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Should Natick Fluoridate?

A Report to the Town

and the

Board of Selectmen

Prepared by the

Natick Fluoridation
Study Committee

13 E. Central Street
Town of Natick, MA
October 14, 1997

Cover Letter to the Selectmen

Benedict J.Gallo, Ph. D.
Jason Kupperschmidt, B.
Norman R. Mancuso, Ph.D.
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Fluoridation Study Committee
Town of Natick
Town Hall 13 E. Central St. Natick MA 01760

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Findings

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Table of Contents

Background and History

History of the Fluoridation Issue in Natick

In May of 1988, the Natick Board of Health ordered the upward adjustment of fluoride in the town water supply. A petition was filed by more than ten (10%) per cent of the town's registered voters to place the question on the ballot. A public referendum was held on Nov. 8, 1988 on the question:

"Shall the public water supply for domestic use in this Town be fluoridated?"

The voters approved the issue by a vote of 7453 (51.4%) yes to 7044 (48.6%) no. A subsequent referendum was held on Mar. 25, 1997 on the identical question, except that in this case, the status of the question was non-binding. In this latter case, the voters did not approve this question by a vote of 2635 (54.3 %) no to 2220 (45.7 %) yes, a reversal of opinion of almost six (6%) percent.

Subsequent Town Meeting Articles 35 and 36 sought to put aside the 1988 vote and to pass special legislation which would place the issue on the ballot at the next annual town election or biennial state election, whichever was held first. Town Meeting voted to indefinitely postpone both articles.

The Chairmen of the Board of Health and the Selectmen requested a legal opinion from the Town Counsel, John P. Flynn, Esq., which was provided on May 8, 1997. (1)

Appointment of the Natick Fluoridation Study Committee

Recognizing the existence of an incredibly large database of conflicting opinion and that both proponents and opponents on the fluoridation issue were entrenched and unlikely to reach a consensus, on April 28, 1997, the Board of Selectmen voted to appoint a special committee of unbiased and qualified people to study the fluoridation issue and to report back on their findings to the Board of Selectmen within approximately ninety (90) days. Hearings at two subsequent Selectmen's meetings produced a number of qualified, scientifically trained and experienced people and from that group, the Natick Fluoridation Study Committee (hereinafter NFSC) was formed by the Board of Selectmen. (2) The present document constitutes the report requested by the Board.

Recommendation and Charter of the NFSC

Mechanism of Approach to be Taken

Since an exhaustive literature search of the issues of fluoride and fluoridation could consume the resources of the committee for several months, the Board of Selectmen recommended that the committee obtain from the proponents and opponents of the issue suitable documentation with which to begin the pursuit of the resolution of the issue. (3)

The fluoridation proponents in Natick consist of the Natick Board of Health (hereinafter BOH) and those citizens of the town who are similarly disposed on the issue of fluoridation. A letter was sent by NFSC to the BOH on July 3, 1997 requesting that the BOH provide:

> Five sets of documents, each comprised of a maximum of six study reports which in the opinion of the BOH most clearly explain why the public water supply of Natick *should* be fluoridated.
>
> In addition, a maximum of six endorsements, letters and other non-data-intensive documents that support their contention that fluoridation is *both beneficial and lacking in undesirable side effects.*

On August 11, the BOH complied with this request by providing five copies of each of the above sets of documents (see Appendix A - Source Materials).

The opponents of the fluoridation issue in the town are represented by Ms. Shirley Brown of Megonko Road in Natick and Dr. Myron Coplan of Intellequity, Inc., also of Natick. Each of these individuals were also requested on July 3, 1997 to provide to NFSC similar packets of materials as follows.

> Five sets of documents, each comprised of a maximum of three study reports which in the opinion of the opponents most clearly explain why the public water supply of Natick *should not* be fluoridated.
>
> In addition, a maximum of three endorsements, letters and other non-data-intensive documents that support their contention that the *undesirable side effects* of fluoridation outweigh its beneficial effects.

The above materials were provided immediately by Ms. Brown and on July 9, 1997 by Dr. Myron Coplan (see Appendix A - Source Materials).

In this manner, it was anticipated that the most driving arguments for and against the issue would be immediately present, as would be the best reference list(s) of supporting original research papers. All of the above notwithstanding, the NFSC was not constrained to limit its search to the above materials and has instituted wide-ranging literature searches on the entire issue, often spending several hours per day in reading and/or pursuing further information.

Charter of the Fluoridation Study Committee

The Board of Selectmen, in appointing this committee, established the need to address several issues. (4) These are:

> *1. On the basis of the documentation provided to you by the proponents and opponents, do you believe that the potential side effects associated with fluoridating Natick's public water supply outweigh the potential benefits?*
>
> *2. If your answer to question 1 is "No" -- i.e. you believe Natick's water should be fluoridated -- do you believe that steps should be taken to establish the appropriate dosage before such fluoridation begins?*
>
> *3. If your answer to question 2 is "Yes", do you believe that an outside organization should be engaged to examine Natick school children and determine their DMFS (decayed, missing and filled surfaces) levels as an aid to selecting an optimum fluoridation level?*
>
> *4. If your answer to question 3 is "Yes", what organizations (identify at least two) are qualified to conduct such a survey, and what are preliminary estimates of the costs involved?*

Format of the Report

The format being used for this report is based upon the four *charges* of the Board of Selectmen with regard to the fluoridation issue. Each *charge* or question constitutes one section of the report. The individual issues addressed within each section result from the nature of the associated charge or question. In any publication of this nature, the issue of providing references must be addressed. On the one hand, material for public consumption is rarely well-received when numerous references are included. This is particularly so when the references take the form of foot-notes as opposed to end-notes. **On the other hand, nothing makes a report such as this more suspect than when no verifiable references are included and the reader is tacitly expected to accept the discussion and conclusions on faith alone**. We feel strongly enough about this issue to insist on the presence of references. The only relaxation of this requirement was an attempt to reduce the tedium that would be caused by having to constantly flip to the end of the report to find a reference or

explanatory note. This was accomplished by placing all of the references for each section immediately following that particular section. This has the necessary consequence of occasionally having more than one occurrence of the same reference but improves the readability and overall usefulness to the reader.

General Background of the Fluoridation Issue

Scientific information that has an impact on political and economic interests often generates controversy, even within the scientific community. This controversy is often magnified when the information is presented in the lay press. It is not that truly non-partisan reporting is unavailable, rather it is that the entrenched partisans of any such issue are only willing (able?) to accept those portions of the report which support their contentions. Be that as it may, there are some reviews of the fluoridation issue that are unbiased, chief among which is a review by Bette Hileman appearing in Chemical & Engineering News. (5) As background material, it is required reading for anyone interested in the issue of fluoridation. A brief portion of this review is quoted below in order to set the stage for further discussion. (Contrary to normal usage, direct quotes appearing within the text below are emboldened and not italicized in order to distinguish them from the remaining commentary.)

Throughout this report, the reader will note the recurrent use of the words ***"optimal", "optimum"*** and such phrases as ***"optimally fluoridated"***. It is important to understand that this usage is a direct contribution of the profluoridation argument and is therefore vigorously objected to by the antifluoridation contingent. In most cases, we have placed quotation marks around these words and phrases to indicate that the term is disputed and should be read as ***"so-called optimum"***.

> *".....The style of promotion that fluoridation's proponents have used from the very beginning probably made the issue more controversial than it need have been.*
>
> *The idea of fluoridating water supplies first arose from studies of dental mottling in areas, such as communities in Texas, where the water supply is fluoridated naturally. In the 1930s, H. Trendley Dean, a dental surgeon at the U.S. Public Health Service, correlated the occurrence of mottling or dental fluorosis with the fluoride content of water supplies in 345 U.S. communities. Fluorosis was most common in cities that had the highest concentration of fluoride in their water. He and his colleagues also unexpectedly found a lower incidence of dental caries in areas of endemic dental fluorosis.*
>
> *Dean concluded that the fluoride content of the drinking water causes a lower rate of dental caries. He also determined that the incidence of mottling was very minor when the fluoride content was 1 ppm or lower but rose linearly at higher concentrations. From this, PHS officials decided in 1943 that 1 ppm was an optimal level at which to artificially fluoridate water supplies in temperate climates. In areas where the fluoride*

content exceeded 2 ppm, they recommended fluoride be reduced to a level near 1 ppm.

In 1945, PHS initially planned to conduct 10-year studies of artificial fluoridation in two experimental projects, one in New York and one in Michigan. One city in each state would be fluoridated artificially and another would serve as a control. PHS officials intended to complete these projects before deciding whether to recommend fluoridation of drinking water as a general practice for all communities.

However, two public health officers in Wisconsin, Francis A. Bull and John Frisch, quickly became convinced of the effectiveness of fluoridation and launched a nationwide campaign to persuade PHS to endorse it. Also, results from the two projects that leaked out in 1950, after the trials had been going on for five years, revealed a sharp reduction in dental caries in the fluoridated cities. As a result of this disclosure and Bull's and Frisch's campaign, PHS officials endorsed fluoridation on June 1, 1950.

Several deficiencies in research by PHS were subsequently aired at Congressional hearings in 1952 and 1957. There had been almost no careful studies to assess the possible adverse health effects of lifelong consumption of fluoridated water. Aside from their dental health, the medical condition of residents of naturally fluoridated areas had been examined superficially, at best. In one of the fluoridation trials, research plans included a study of adverse effects of artificial fluoridation on children, but none on adults. No studies focused on malnourished children and infants, despite a warning in 1952 by Maury Massler, professor of pedodontics at the University of Illinois College of Dentistry, that **"low levels of fluoride ingestion which are generally considered to be safe for the general population may not be safe for malnourished infants and children, because of disturbances in calcium metabolism."**

Neither PHS nor anyone else had investigated potential carcinogenic effects, effects on pregnant women, or effects on people with chronic kidney impairment or other chronic diseases. Even in the early 1950s, enough was known of fluoride's toxicity profile to identify these as important topics to investigate.

From the beginning, the movement to fluoridate water was conducted more like a political campaign than a scientific enterprise. At a meeting of state dental directors with PHS officials in June 1951, Bull recommended tactics for promoting fluoridation. **"If it is a fact that some individuals are against fluoridation, you just have to knock their objections down. The question of toxicity is on the same order. Lay off it altogether. Just pass it over. 'We know there is absolutely no effect other than reducing tooth decay,' you say, and go on. If it becomes an issue, then you will have to take it over, but don't bring it up yourself."**

"The minute doubt is created in the minds of the public, any public health program is doomed to failure," *Bull later wrote in the Journal of the American Dental Association.*

The political role of dentists has been emphasized throughout the history of fluoridation. In 1970, even after 25 years of fluoridation, John W. Knutson, then

> *professor at the University of California Medical Center, advised dentists that when they discussed fluoridation with the public, they must realize that* **"they are propagandizing, not simply educating."** *This attitude, widely shared by political proponents, led early advocates to treat fluoridation campaigns as debates to be won with dogmatic assertions and attacks on the credibility of the opposition. To promoters, the debate has never been seen as a scientific search for truth.*
>
> *As a result, profluoridationists prepare booklets for the public that contain highly biased information. If scientific studies are cited, only those that support their side of the argument are mentioned. Those opposed to fluoridation counter with equally biased propaganda....."* (5)

According to many opponents of fluoridation, other tactics which were also widely used to denigrate any potentially negative effects include character assassination, inflammatory portrayal of the opposition, the widespread suppression of opposing results (see Appendix B) and the widespread use of sensationalism, etc. In the latter case at least, the antifluoridationists are no less culpable.

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Question 1 An Analysis of the Side Effects of Fluoridation

1. *On the basis of the documentation provided to you by the proponents and opponents, do you believe that the potential side effects associated with fluoridating Natick's public water supply outweigh the potential benefits?*

This is the main question to be addressed in this report. It also includes the reasons why the issue of fluoridation is so controversial. In spite of the other topics presented in this report, the main issue remains whether the benefits of fluoridation outweigh the risks. Moreover, it appears that the only significant benefit of fluoridation is the reduction of dental caries, this in spite of other past reports touting the applicability of fluoridation to osteoporosis as well (see the section on other positive effects of fluoridation). The *"profluoridationists"* have repeatedly asserted that there are no negatives associated with the process, or alternatively, that all of the negative reports are without scientific justification or merit. Because of this position, an examination of these negative reports tends to cast the examiners in the role of a "devil's advocate", the chief difficulty of which is that the examiners are then also perceived as being *"antifluoridationists"*, when in fact they are merely seeking to extract the truth from the polemics and hysteria of the issues and to expose this information to a critical and unbiased analysis. With this in mind we report on the following material.

The Beneficial Effects of Fluoridation

History of the Fluoridation Program in the United States

During the course of dental research conducted in the early part of this century on the condition then known as "Colorado Brown Stain" (a.k.a. "Texas Teeth" or dental fluorosis as it came to be medically known), it was discovered that individuals, living in areas where the water is known to contain elevated (relative to most water supplies) fluoride concentrations, exhibited a decreased rate of incidence of dental caries. (1) Several studies conducted during the decades prior to 1960 confirmed that when a small quantity (ca. 1 part per million, ppm) of fluoride was added to a community water supply, the incidence of tooth decay among the residents of those community decreased substantially. (2) The initial studies indicated a reduction in tooth decay of 50 to 60 per cent. (3) As a result of these achievements, the process of fluoridation of community water supplies has continued and resulted in more than half of the U. S. population being served by a fluoridated supply. (4) Numerous scientific papers have supported fluoridation throughout its history. (5-9) More recent studies , as interpreted by profluoridationists, indicate that reductions of between 20-40% are routinely achievable. (10-12)

Features of the Fluoridation Program

The desirability of the process of fluoridation of community water supplies, as maintained by the profluoridation community, is based upon the following reasoning: (13)

- Fluoridation is the least expensive and most effective way to reduce tooth decay.
- Fluoridation is safe.
- Fluoridation benefits both children and adults.
- Fluoridation benefits continue for a lifetime when fluoridated water consumption continues.
- Fluoridation is the surest way for everyone in the community to benefit.
- Fluoridation benefits everyone when they drink fluoridated water and consume foods and beverages prepared with it.

Supporters of Community Water Fluoridation

The following non-exhaustive list illustrates the wide-spread support for the fluoridation programs. (13, 14)

- Mass. Dept. of Public Health
- American Association of Public Health Dentistry
- American Dental Association
- Centers for Disease Control & Prevention
- American Medical Association
- World Health Organization

Other Positive Effects of Fluoridation

Proponents of fluoridation have also attempted to show that fluoride can be used to alleviate the symptoms of osteoporosis, and therefore that people living in fluoridated areas may be helped by the fluoride they are accumulating in their bones. Because fluoride increases bone mass, (see the section on Osteosclerosis) numerous patients have been given and are still being given large doses of fluoride as a treatment for osteoporosis. Recent data has not produced compelling evidence of beneficial results. The FDA has not approved the use of fluoride for osteoporosis. In spite of this, the National Osteoporosis Foundation reports that an FDA advisory committee has recommended that slow-release sodium fluoride be approved for the treatment of osteoporosis. (15)

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Refutation - Water Fluoridation today is at best only minimally effective

Fluoride was first investigated as an anti-caries agent because of the inverse relationship noted in many areas of the country between the prevalence of dental caries and the level of fluoride in drinking water. At first, scientists believed that the anti-caries activity of fluoride was the direct result of its incorporation into the apatite crystal of enamel, thus increasing its stability and reducing its acid solubility. The theory of pre-eruptive fluoride incorporation as the principal mechanism of caries prevention has been largely discounted. (1) Recent studies have suggested that the anti-caries action of fluoride may be related to the fluoride levels in the saliva and plaque fluids rather than the enamel surface itself, i.e., the action is topical rather than systemic. (2,3) Indeed, if one diligently searches the literature of fluoridation, it becomes clear that there are widespread differences of opinion among experts as to the actual mechanism. Moreover, it is significant that in one survey, only 66% of physicians thought that community fluoridation is very effective and only 37% think that dietary supplements are very effective. (4) This same survey reported that only a small percentage of physicians and dentists believe that topical fluorides are very effective preventive measures, so it is clear that even among *"those who should know"*, there is a large measure of discordant opinion.

The sources of fluoride intake for the U. S. population are primarily water, food, dental products and air (see Tables I & II). Children may also receive fluoride in supplements. Although fluoride exposure is generally greater in areas with fluoridated water than in areas with non-fluoridated or low-fluoridated water, populations in both areas are exposed to fluoride from food sources, drinking water, processed beverages and dental products. In one recently published survey, Dabeka and McKenzie have reported that the average intake of fluoride from food, averaged over all ages and sexes, was 1.76 mg/day. (5) Fluoride exposure differs markedly, depending upon several factors, e.g., lifestyle, dietary practices, age, gender and health status. It is clear however that drinking water provides minimal topical fluoride. The Agency for Toxic Substances and Disease Registry (ATSDR) sets the Minimal Risk Level (MRL) for ingestion of fluoride at 0.4 mg/kg/day. (6) In a 20 pound child this amounts to 3.6 mg/day and for a 50 pound child, the minimal risk level is about 9 mg/day. The MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure. However, to avoid an undesirable degree of dental fluorosis, children should consume no more than 0.10 mg of fluoride per kg of body weight per day.(7)

Table I - Fluoride Concentrations (ppm) in Food

Foods (Note A)	Mean (ppm)	Standard Deviation	Range (ppm)
Dairy Products	0.25	0.38	0.02 - 0.82
Meat Fish & Poultry	0.22	0.15	0.04 - 0.51
Grain & Cereal Products	0.42	0.40	0.08 - 2.01
Potatoes	0.49	0.26	0.21 - 0.84
Leafy Vegetables	0.27	0.25	0.21 - 0.84
Legumes	0.53	0.05	0.49 - 0.57
Root Vegetables	0.38	0.11	0.27 - 0.48
Fruits	0.06	0.03	0.02 -0.08
Oils & Fats	0.25	0.15	0.02 - 0.44
Sugar and Adjuncts	0.28	0.27	0.02 - 0.78
Nonclassifiable Foods	0.59	0.19	0.29 - 0.87

Note A The foods were ready to eat or prepared for eating. When preparation required the use of water (e.g. preparing juice from concentrate or boiling vegetables), the local water was used which contained 1 mg/L (1 ppm) of fluoride was used. Nonclassifiable foods included certain soups and puddings, among other items. (1)

Table II - Estimated Daily Fluoride Intake of Children (1)

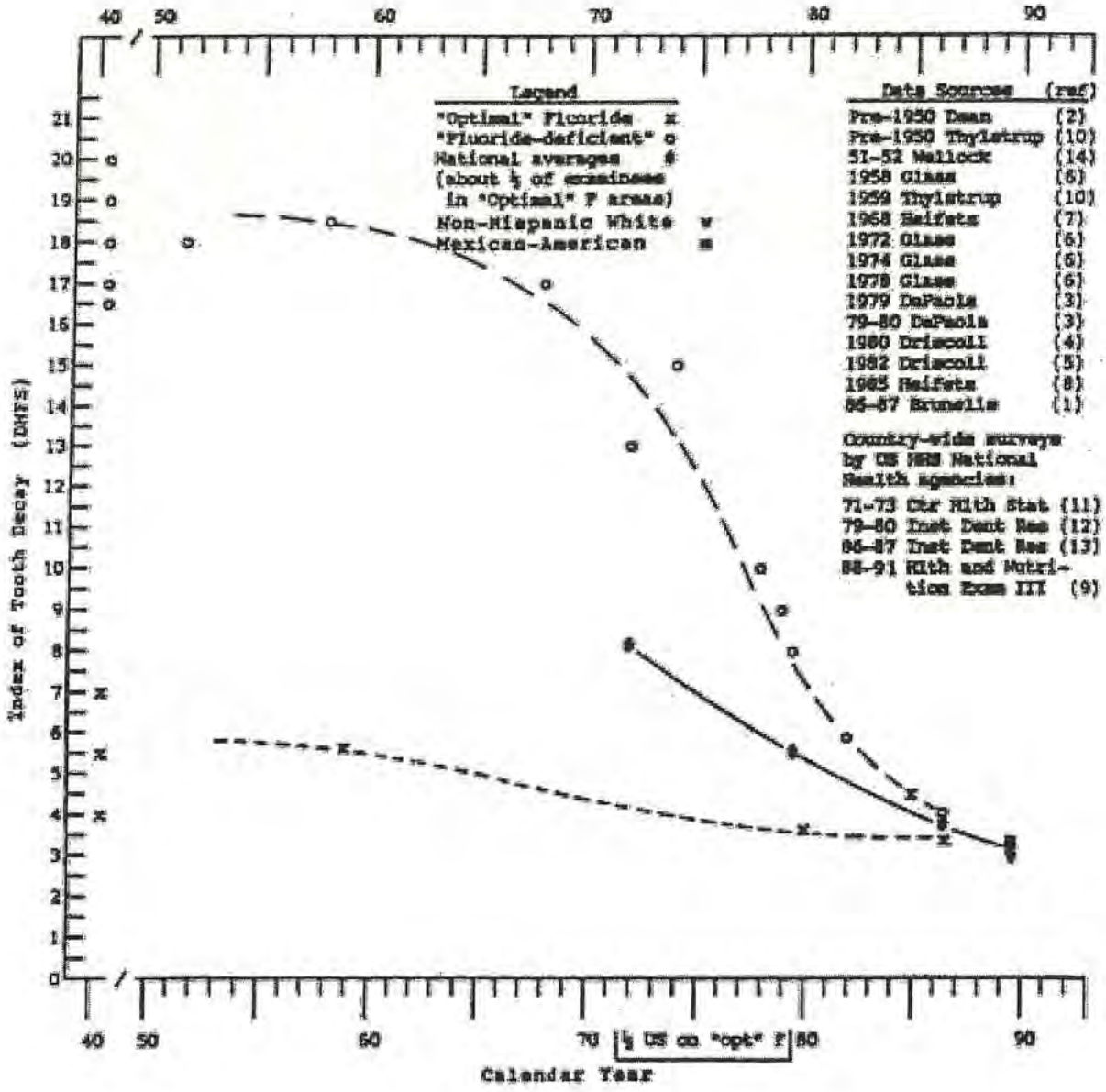
F Concentration in Water (ppm)	Intake from Food	Intake from Beverages	Intake from Dentifrices	Intake from F Supplements	Estimated Total Intake
<0.3	0.15 – 0.30	0.10 – 0.30	0.20 - 1.20	0.50	0.95 - 2.30
0.7-1.2	0.40 – 0.60	0.30 - 1.30	0.20 - 1.20	NR	0.90 - 3.60
>2.0	1.00 – 2.00	0.60 - 3.00	0.20 - 1.20	NR	1.80 - 6.20
Units	mg/day	mg/day	mg/day	mg/day	mg/day
Notes			(a)	(b)	

(a) Assumed that dentrifice used twice daily
(b) Assumed that dentrifice fluoride supplement taken daily

Studies show that tooth decay has declined in every country in the developed world. (2,3,9) The largest study in this country (the NIDR 1986-87 survey of 39,000 U.S. school children, (1)) showed no significant difference in dental caries in those living in fluoridated as opposed to those living in non-fluoridated communities. (11) See Fig. I.

Figure I - Decline in DMFS Index in 12-14 Year Olds (12)

50-YEAR HISTORY OF TOOTH DECAY PREVALENCE AMONG 12-14 YEAR-OLDS LIVING IN "OPTINAL FLUORIDE" AND "FLUORIDE-DEFICIENT" AREAS



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In British Columbia, only 11% of the population lives in areas containing fluoridated water, as opposed to 40-70% in other Canadian regions. However British Columbia has the lowest rate of tooth decay in Canada. (10) According to a 1987 report by Dr. Allan Gray, then director of the Division of Dental Health services for British Columbia, DMFT (decayed, missing or filled teeth) rates (see Appendix D) were falling drastically in both fluoridated and non-fluoridated areas. (11)

Mark Diesendorf, an applied mathematician and health researcher in the Human Sciences Program at Australian National University has found, by comparing results from about 24 studies of unfluoridated districts in eight countries, that reductions in dental caries are just as great in non-fluoridated areas as in fluoridated areas. (2)

One of the most significant factors in any comparison of the incidence of dental caries is the manner in which this is expressed (see Appendix D for a discussion of the methods of describing these incidence rates). In the early days of DMFS (decayed, missing or filled surfaces) scores (in the range of 18-20), a 20% difference (fluoridated vs. non-fluoridated) would indicate several cavities per child. However, given the current average DMFS scores (in the range of 2-4) it is clear that a 20% difference represents less than one cavity per child.

Summary

It seems clear that there is a link between fluoride intake and the reduction of dental caries. Although the mechanism is not fully understood, the effect is now thought to be due primarily to topical rather than systemic fluoride. In the early days of fluoridation, there were few other sources of fluoride in the daily diet. The introduction of fluoride into the daily diet (beverages prepared in communities with fluoridated water, toothpaste, food, supplements, etc.) starting in the 1950's has had the effect of reducing dental caries worldwide, even in those countries that do not fluoridate. In fact, fluoride is so widespread today that introducing it into public water supplies seems to have a very minimal effect in reducing dental caries. Current data seems to indicate little difference between the health of teeth in communities having fluoridated water supplies compared to communities having unfluoridated water.

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Toxic Effects of Fluoridation and Inorganic Fluoride

The next several sections deal with those issues which have been raised as being either detrimental or non-beneficial side-effects caused by the fluoridation process and/or the ubiquity of various forms of (chiefly) inorganic fluoride compounds in the environment or as used in the treatment of dental caries. Each section analyzes the scientific literature, paying particular attention to those primary research results which have appeared recently. Each section is comprised of both a discussion as well as a summary with respect to the particular issue treated.

The Acute Toxicity of Fluorine-Containing Materials

> *"Fluoride is a halogen with unique properties that enable it, in toxic quantities, to alter calcium availability to tissues, to effect changes to blood clotting parameters, to cause severe cardiac dysfunction, to alter bone and tooth structure, to cause severe anxiety in those who drink fluoridated water and to lead to death where it has been misused either accidentally , in suicide or in homicide."(1,2)*

The principal uses of fluoride include prophylaxis of dental caries, toothpastes, animal husbandry, timber preservation and pesticides. Both sodium fluoride and sodium fluorosilicate have been widely used as insecticides and rodenticides. (9) Ellenhorn and Barceleaux list the therapeutic dose at 0.25 to 0.50 milligrams per day (mg/d) and the oral lethal dose as 5-10 grams of sodium fluoride, (3) although less than 1 gram by mouth has caused severe poisoning. (4) Overfluoridation has resulted in mass intoxication (5) and death has resulted from ingesting 2 g of fluoride. (10)

Waldbott and others (6,7) have also remarked upon the extremely small safety margin between therapeutic doses and those causing toxic effects in humans, when compared to safety margins normally employed in medicinal products.

H. C. Hodge, a toxicologist of wide repute, notes that the minimum safety factors in the dietary regimen of any toxic material should be at least one hundred times the therapeutic dose. (8) Other workers in the field consider even this safety margin far too narrow.

Summary and Conclusions on Acute Toxicity

On the basis of acute toxicity, it is unreasonable to take a strong position against water fluoridation. On the other hand, there are questions about the nature and significance of chronic toxicity effects which must be addressed, as in the case of proper therapeutic quantities of fluoride. Moreover, the question which must be

resolved prior to taking such a position appears to be the determination of what constitutes the proper or safe and effective therapeutic dose. This question has great significance to many of the subjects treated in the following sections. Therefore, Appendix C is included to provide the responses to this question by a number of independent organizations as well as to provide guidelines relative to the total daily individual ingestion of fluoride for different age groups.

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Dental Fluorosis - An Undesirable Effect of Fluoridation

Varying amounts of fluoride are found naturally in the water supplies of many communities, including Natick. Natick's water has only a small amount of fluoride, on the order of about 0.1 ppm (0.1 mg fluoride per liter water). (1) If too much fluoride is ingested by children it results in a toxic dental condition known as dental fluorosis. This condition is marked by visible mottling and/or discoloring of tooth enamel, pitting of the enamel and disturbed tooth shape. (2, 3) Teeth with moderate dental fluorosis typically ***"....may have yellow and brown strains..... they are pitted, brittle, and susceptible to fracture."*** Severe dental fluorosis ***"...not only produces unattractive teeth but also may increase the risk of tooth loss because it destroys parts of the protective enamel."*** (4) Historically, dental fluorosis was first noted in children who grew up in areas where the drinking water supplies had a relatively high content of dissolved fluoride as shown in Table I which also lists communities with little or no dissolved fluoride in their water. Children in these latter communities had very little dental fluorosis. (5) It was also noted that children with dental fluorosis had fewer cavities. (2) Thus began the start of the "fluoride tradeoffs" which resulted in 80% to 90% of "treated" children with fewer cavities and 10% to 20% of those with dental fluorosis.

Table I - Dean's Survey(s) of Dental Fluorosis 1939-1940

Percentages of Children Experiencing Various Degrees of Dental Fluorosis

City	Year	N	F** (ppm)	Very Mild	Mild	Moderate	Severe	Total
Waukegan IL	1939	423	0.0	0.2	0.0	0.0	0.0	0.2
Oak Park IL	1939	329	0.0	0.6	0.0	0.0	0.0	0.6
Evanston IL	1939	256	0.0	1.6	0.0	0.0	0.0	1.6
Mi City IN	1940	236	0.1	0.0	0.0	0.0	0.0	0.0
Quincey IL	1940	330	0.1	0.3	0.0	0.0	0.0	0.3
Elkhart IN	1940	278	0.1	0.4	0.0	0.0	0.0	0.4
Portsmouth OH	1940	469	0.1	1.3	0.0	0.0	0.0	1.3
Middletown OH	1940	370	0.2	1.1	0.0	0.0	0.0	1.1
Zanesville OH	1940	459	0.2	1.5	0.0	0.0	0.0	1.5
Lima OH	1940	454	0.3	2.2	0.0	0.0	0.0	2.2
Marion OH	1940	263	0.4	5.3	0.8	0.0	0.0	6.1
Elgin IL	1939	403	0.5	3.5	0.7	0.0	0.0	4.2
Pueblo CO	1940	614	0.6	6.2	0.3	0.0	0.0	6.5
Kewanee IL	**1939**	**123**	**0.9**	**10.6**	**1.6**	**0.0**	**0.0**	**12.2**
Aurora IL	1939	633	1.2	13.9	1.1	0.0	0.0	15.0
Joilet IL	1940	447	1.3	22.2	3.2	0.0	0.0	25.3
E Moline IL	1940	152	1.6	29.6	2.0	0.0	0.0	32.0
Maywood IL	1939	171	1.6	29.2	4.1	0.0	0.0	33.3
Elmhurst IL	1939	170	1.8	30.0	8.8	1.2	0.0	40.0
Galesburg IL	1940	273	1.9	40.3	6.2	1.1	0.0	48.0
C Springs CO	1940	404	2.6	42.1	21.3	8.9	1.5	73.8

When, why and how does Dental Fluorosis occur?

Dental fluorosis occurs during early childhood while deciduous and permanent teeth and tooth enamel are still being mineralized and before they erupt within the mouth. (6,7) It is believed that dental fluorosis occurs because of the toxicity of fluoride to the enamel-forming cells of the teeth. (6) The degree to which a child experiences dental fluorosis depends on the amount of fluoride (s)he ingests. (2, 3, 6, 8) Dental authorities estimate that a child should ingest daily 0.03 mg to 0.07 mgs of fluoride per kg of body weight. When this amount is exceeded, dental fluorosis results. Moreover, the greater the fluoride overdose, the more severe is the dental fluorosis. Even with supervision, it is possible for a small child to overdose on fluoride each day with only one brushing with a fluoride tooth paste by swallowing much of it during the brushing process. (7)

The current model of fluorosis development proposes that *"....fluoride affects the forming enamel by making it porous. The degree and extent of the porosity depend on the concentration of fluoride in tissue fluids when the teeth are developing..."* and *"....the porosity and discoloration can vary in degree among different areas of the same tooth....."* (2) The ultimate result is the increasing porosity of the teeth and, in extreme cases, loss of the affected teeth. (9) Dental fluorosis is an excellent biomarker of excess fluoride ingestion and fluoride intoxication. (10) It is a visible, sometimes easily seen and noticed marker of fluoride intoxication. Unfortunately it tells us of excessive fluoride intake after-the-fact, i. e. after the newly emergent teeth have already been altered.

Why is the Prevalence of Dental Fluorosis Increasing?

There is now widespread recognition of the fact that the prevalence of dental fluorosis has increased substantially throughout those countries where fluoridation is practiced. (11-13) However, in spite of some reports to the contrary, (2) there does not appear to be general agreement within the dental community as to whether the severity of dental fluorosis has increased.

The nationwide increase of dental fluorosis was first recognized, documented and published by the National Institute of Dental Research (NIDR) after conducting (1986-1987) a survey that involved 32,241 U.S. school children. The total prevalence of dental fluorosis in this group of children was estimated to be 22.3 percent and included (mostly) very mild to mild dental fluorosis. (2) However some moderate to severe dental fluorosis was also found in approximately 1% to 2% of the children in "optimally" fluoridated water districts. (4) Another NIDR report published in 1988, studied four areas in Illinois with water concentration of one, two, three and four times the recommended "optimal" fluoride level. As of 1985, in the "optimally" fluoridated areas, twenty nine per cent of all tooth surfaces examined were reported to be affected by dental fluorosis. In those areas that had 2 to 4 times the optimal dose of

fluoride in the water supply, dental fluorosis affected close to seventy per cent of the teeth involved. (2) An even more recent study, published in 1990 (Table II) listed dental fluorosis in additional comparable cities in the United States and New Zealand with water systems "optimally" fluoridated and those with low fluoride. (14)

Table II - Percentages of Children with Dental Fluorosis

"Optimal" Fluoride Communities	Age Range of Children	F Conc. (ppm)	Percent of Fluorosis	Ref. (Note b)
Auckland (NZ)	7-12	1.0	**25**	(2)
Auckland Region	9	1.0	**25**	(3)
Hastings (NZ)	10	1.0	**23**	(5)
Kewanee, IL	**13-15**	**1.0**	**28**	**(12)**
Kerrville, TX	7-18	1.4	**16**	(14)
Angleton, TX	7-18	1.3	**33**	(14)
Alvin, TX	7-18	1.3	**29**	(13)
Kingsvlle, TX	7-18	1.0	**39**	(13)
Richmond, MI	6-12	1.2	**51**	(13)
Redford, IL	6-12	1.0	**48**	(13)
Hudson, MI	6-12	0.8	**32**	(13)
New York State	12-17	1.0	**27**	(15)
Low Fluoride Communities:	**Age Range of Children**	**F Conc. (ppm)**	**Percent of Fluorosis**	**Ref. (Note b)**
Richmond (NZ)	12-14	0.2	**6**	(I)
Auckland (NZ)	7-12	0.2	**4**	(2)
Auckland Region	9	0-0.2	**15 (Note a)**	(3)
Napier (NZ)	10	0-0.2	**3**	(15)
Iowa towns	8-16	0.0	**3**	(10)
San Antonio, TX	7-18	0.4	**2**	(14)
San Marcos, TX	7-18	0.3	**8**	(14)
N. Braunfels, TX	7-18	0.3	**9**	(14)
Cadillac, MI	6-12	0.0	**12**	(13)
New York State	12-17	0-0.3	**4**	(15)

Note a) 55% of the children received fluoride supplements.
Note b) References in the last column of the above table are taken from Ref. 14

In carefully comparing the data in Table 1 and Table 2, a number of observations can be made:

(1) the incidence of dental fluorosis in the children of Kewanee IL (selected because it is included in both studies and uses "optimally" fluoridated water) has increased from 1939 to 1990;

(2) the prevalence of dental fluorosis is greater in "optimally" fluoridated communities than in communities with fluoride-deficient water and;

(3) the percentage of dental fluorosis found today in "optimally fluoridated" communities approaches those found in communities with water containing 2, 3 and 4 times the "optimal level" of fluoridation 50 years ago.

The Anticipated Occurrence of Dental Fluorosis and Needed Corrective Measures

Now, as well as in the past few years, parents are being cautioned by the dental profession against excessive fluoride intake by infants and children by carefully regulating their total intake of fluoride in order to prevent dental fluorosis in developing teeth. This becomes increasingly more difficult as the infant/child grows older because of the ubiquity of fluoride in our country. Excessive amounts of fluoride can be ingested from a number of available sources: daily dietary fluoride supplemental pills, using fluoridated toothpaste, eating fluoride containing vegetables and fruits, other foods and drinks prepared with fluoridated waters and the application of topical fluoride products to teeth. (2, 7, 15) This is especially applicable if more fluoride is added to the communal water supply. Based on previously published data from other areas with drinking water fluoridated to about 1 ppm or 1 mg/liter ("optimal") our most optimistic scenario will show a minimum of one child out of every ten showing evidence of some degree of dental fluorosis. (2,5) However, if care is not exercised in preventing excessive fluoride intake, two to three children out of ten may develop dental fluorosis. The problem is exacerbated by the permissible fluoride levels in drinking water established by the U.S. Environmental Protection Agency's Safe Drinking water Act of 1974, in which the EPA set, on April 2, 1986, drinking water regulations for fluoride as follows:

1. ***"Primary Maximum Contaminant Level (MCL) of 4 mg F/L to protect against crippling skeletal fluorosis,"*** and

2. ***"Secondary Maximum Contaminant Level of 2 mg F/L to protect against moderate to severe dental fluorosis".***

This suggests that water-based consumer products should be made with water containing 2 ppm of fluoride or less.

The most effective corrective measure is to have children with developing teeth, especially permanent teeth, avoid the intake of toxic quantities of fluoride. This may be difficult to do for several reasons. Firstly, a child can unknowingly and unintentionally get unwanted fluoride from dental products, foods and drinks as mentioned above. Secondly, the expressed symptoms of dental fluorosis are not identical for all children exposed to the same dose of fluoride. Therefore there is difficulty in predicting fluorotic effects. Thirdly, there is also difficulty in diagnosing very mild to mild dental fluorosis by dental clinicians thereby missing opportunities to aesthetically correct objectionable fluorosis. (2)

The severity of the dental fluorosis and the psycho-socio-economic status of a child afflicted with dental fluorosis will determine if corrective action will be taken. Corrective procedures, when required or desired, include vital bleaching, abrasion and bonded veneers. These corrective procedures are performed by dental clinicians. However, at the present time dental fluorosis is considered to be a cosmetic effect (2) and therefore these corrective procedures are not covered by most dental insurance companies. Moderate-to-severe fluorosis results in unattractive misshapen teeth and probably will result in psychological damage to the affected child. However little research on the psychological effects of dental fluorosis on children has been conducted. (9)

Summary and Conclusions

Excessive fluoride intake by children causes a toxic dental condition known as dental fluorosis which is marked by visible mottling/discoloring of tooth enamel, pitting of the enamel and disturbed tooth shape. Dental fluorosis occurs during early childhood while the baby and permanent teeth and tooth enamel are still being mineralized and before they erupt in the mouth. The severity of the dental fluorosis is directly proportional to fluoride ingested in excess of 0.03 mg to 0.07 mg fluoride/kg of body weight/day. The ultimate result is the increased porosity of the teeth and, in extreme cases, loss of afflicted teeth. The prevalence of dental fluorosis is increasing in communities that have water supplies that are "optimally fluoridated" and in those with fluoride deficient doing water because of the ubiquity of products containing fluoride. However the prevalence and severity of dental fluorosis is greater in "optimally fluoridated" communities than those with fluoride-deficient water. Parents are being advised to protect against excessive fluoride intake by infants and children by carefully regulating their total intake of fluoride. It is anticipated that fluoridation of the Natick water supply to 1 ppm or 1 mg/L will result in dental fluorosis to some degree in at least one child out of every ten. However if care is not exercised in preventing excessive fluoride intake, two to three children out of every ten may develop dental

fluorosis. Corrective procedures, when required, can be performed by dental clinicians. However, the cost of teeth rehabilitation will be borne, most likely, by the individual/parent since dental fluorosis is considered to be a cosmetic defect and therefore is not covered by most dental insurance plans.

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Skeletal Fluorosis, Osteosclerosis & Related Disorders

Two diseases of the skeletal system are osteosclerosis and osteoporosis. Osteosclerosis is a disease involving an increase in bone density (and thickening) accompanied by an increase in bone brittleness. Osteoporosis is a disease involving a decrease in bone density (due to loss of calcium) leading to decreased bone strength. (1) Osteofluorosis is an osteosclerosis caused by prolonged overdoses of fluoride.

Osteoporosis

Prolonged or increased ingestion of fluoride is known to result in increased bone mass. However, the bone formed in response to these high and/or prolonged doses of fluoride is reported to exhibit both reduced strength and increased fragility. (2, 3, 4) The abnormal bone resulting from fluoride is of poor quality and while the increased mass helps compressive strength, it generally leads to weakness of tensile strength. Thus, tests of fluoride "treatment" for osteoporosis finds a decrease in vertebral compression fractures but an increased incidence of hip and long bone fractures, compared to control patients. Other researchers have advised abandoning fluoride as a legitimate treatment for osteoporosis for that reason as well as for the well known toxicity of fluorides. In fact, in a 1987 review of fluoride therapy for osteoporosis, Louis V. Avioli, professor at the Washington University School of Medicine, concludes: ***"Sodium fluoride therapy is accompanied by so many medical complications and side effects that it is hardly worth exploring in depth as a therapeutic mode for postmenopausal osteoporosis, since it fails to decrease the propensity toward hip fractures and increases the incidence of stress fractures in the extremities."*** (5)

Skeletal (Osteo)Fluorosis

Osteofluorosis is a complicated disease with a number of stages. The first two stages are preclinical, that is, the patient feels no symptoms but changes have taken place in the body. In the first preclinical stage, biochemical changes occur in the blood and bone composition; in the second stage histological changes can be observed in bone biopsies. Some experts call these changes harmful because they are precursors of more serious conditions. Other experts say they are harmless. (6) Most admit that the effects of long term ingestion of fluoridated water on bone are poorly understood. (6)

The clinical stages of osteofluorosis includes pain in the bones and joints, muscle weakness, fatigue, calcification of ligaments and bone spurs. Most experts in skeletal fluorosis agree that ingestion of 20 mg of fluoride per day for 20 years or more can cause crippling skeletal fluorosis and doses as low as 2 to 5 mg per day over the same time period can cause the preclinical stages. (7) Moreover, the total quantity of

fluoride ingested is the single most important factor in determining the clinical course

> ***Post Publication Correction:*** The correct figures for the development of crippling skeletal fluorosis should be 10-20 mg/day for 10-20 years. (See:

of osteofluorosis. (8) The severity of the symptoms correlates directly with the level and duration of exposure. For almost 40 years, investigators in the United States have searched for evidence of osteofluorosis. The U. S. Public Health Service (8) reports that:

> *"....Radiographic changes in bone indicative of skeletal fluorosis, changes in bone mass, and effects on skeletal maturation were not observed at water fluoride concentrations of 1.2mg/l for 10 years and from 3.3 to 6.2 mg/l for a lifetime. In a survey of 170,000 radiographs of patients living in Texas and Oklahoma with water fluoride levels between 4 and 8 mg/l, Stevenson and Watson (1957) found 23 cases of radiographic osteosclerosis, but no evidence of skeletal fluorosis."* (references deleted.)

Nevertheless, large numbers of people in Japan, China, India, the Middle East and Africa have been diagnosed with skeletal fluorosis. (9) In India, Tanzania and South Africa, crippling forms of skeletal fluorosis have been reported in pediatric age groups as well. (8)

Hip and other Fractures

In clinical practice, the occurrence of atraumatic minor compression fractures of vertebra is common in postmenopausal osteoporotic women and is frequently asymptomatic, being found only by radiographs, though the patient may have noted a slight decrease in height over time. The more morbid consequence of osteoporosis is hip fracture which has the potential for seriously disabling patients. It has been suggested that sodium fluoride could be used as a treatment for osteoporosis since it is associated with *'bone thickening'*. Dr. C. Y. C. Pak and others are conducting a USPH funded and FDA approved study using slow-release sodium fluoride in the management of postmenopausal osteoporosis. (10) In this study, Dr. Pak is administering about 25 mg of fluoride per day in a slow release form to post menopausal women in order to raise their serum fluoride levels from 50 ng/ml to slightly over 100 ng/ml while avoiding fluoride's known gastric inflammatory effects such as mucosal erosions, ulcers, and bleeding which regularly accompany usual oral fluoride supplementation at this dosage. However, according to a critical review of this study appearing in the journal, "Fluoride", Dr. John Lee states that: *"....*(Dr. Pak's study) *seems limited to demonstrating the obvious, i.e., that excessive fluoride causes osteofluorosis."*(10)

The one interesting finding in Pak's interim report is the fact that fluoride supplementation did not cause any reduction in vertebral fractures in women on estrogen supplementation compared to controls. Among estrogen-treated women, the fracture-free rate of the placebo (no fluoride) group compared to that of the fluoride group was 75.0% and 76.9% respectively, an inconsequential difference
In a national study of ecological design (11), Jacobsen et al., examined the association between water fluoridation and the incidence of hip fractures. For the period 1984-1987, a total of 218,951 eligible hip fracture cases were studied. (12) Raheb characterized the results of Jacobsen's study as *"...A small, statistically significant, positive association was found between fluoridation and fracture incidence rates."* (13) However, a careful review of the data of Jacobsen and his co-workers show an eight (8%) percent increase in women [±2 percent] and a seventeen (17%) percent increase for men [±4 percent]. A more recent study on a smaller population (which was restricted to Mormon communities in Utah to correct for confounding factors such as smoking and/or use of alcohol) showed an increased incidence of hip fractures of 27% in women and 41% in men, albeit with a larger 95% confidence interval. (14) While four other studies indicate either no effect or a negative effect of fluoridation, these studies involved a total of only 6,874 subjects as opposed to positive correlation in the case of 781,575 subjects.

Summary

Well controlled studies have not demonstrated a beneficial effect of the use of high doses of fluoride in reducing osteoporosis and related bone fractures. However, there has been shown to be a positive relationship between water fluoridation and increased hip fractures in persons 65 years of age and older. Human crippling osteofluorosis is endemic in several countries of the world, but is extremely rare in the United States. A number of factors govern the amount of fluoride deposited in the skeleton. The important factors include:

1) age at exposure
2) duration of exposure
3) dose of fluoride
4) nutritional status
5) renal status
6) individual biological variation.

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Carcinogenicity

Whether or not fluoride causes cancer in humans has been a subject of heated debate. Based on the journal articles and other documents reviewed by this committee, the debate has not been resolved and appropriate epidemiology studies have yet to be conducted. This section of the committee report summarizes the two major animal bioassays that have tested fluoride for its carcinogenic potential. It also reviews some of the human epidemiology studies that have been published.

Animal Studies

The US National Toxicology Program (NTP) tested sodium fluoride for carcinogenicity in a 2 year bioassay using rats (F344/N) and mice (B6C3F1) (1) The most often cited result of this study is that it provided "equivocal" evidence of carcinogenicity based on a statistically significant elevation of osteosarcoma (a type of bone cancer) in male rats. No bone cancer was observed in female rats and male or female mice. However, a careful review of the data presented in the published report of this study (1) shows that one additional osteosarcoma was observed, but not counted in male rats. In addition, one male and one female mouse treated with fluoride also developed osteosarcoma, although these were not included in the summary table, only in the footnotes.

In addition to bone cancer, the NTP study showed marginal differences between control animals and dosed animals with respect to cancer of the oral mucosa, thyroid gland and uterus of rats and the hematopoietic system and liver in mice. While several liver tumor types were observed in male and female mice, two tumor types were considered highly unusual and worthy of note: hepatoblastoma and hepatocholan-giocarcinoma (1).

In general, animal bioassays test chemicals at doses much higher than received by humans, and the results are then extrapolated to lower doses. In the NTP study, sodium fluoride was administered in drinking water at 25, 100 and 175 ppm. A special low fluoride diet was formulated. A measure of cumulative dose (dose taken in over a long period) for fluoride is the ash content in the bone. Comparison of the fluoride in the bones of the animals in the high (125 ppm) dose group with humans drinking water with fluoride concentrations above 2 ppm show that older humans have more fluoride in their bones than the high dose animals (1,2). In other words, this study was conducted using fluoride doses that humans actually receive.
The NTP study has been criticized by all sides: some charge the study understates the cancer potency, others say it overstates the potency and is irrelevant to humans (2,3,4).

William Marcus, an EPA toxicologist, compared the rate of osteosarcoma in historical controls (control animals used in previous toxicology testing studies) with those

obtained in the dosed animals in the fluoride study. He assumed the fluoride dose in the historical controls was due to ingestion of normal rat chow, and represented a dose between the 25 ppm and 100 ppm dose group in the fluoride drinking water study (the NTP agrees with this calculation). Marcus found that the observed "historical" control rate of osteosarcoma fit exactly where expected based on the fluoride dose (2). Marcus also reports that the original pathologist's classifications of the liver tumors in the rat, oral tumors in the rat, and adrenal pheochromocytomas were consistently downgraded by a review panel (2). This would serve to underestimate the evidence for cancer based on the animal study. Marcus recommended that EPA assemble an independent panel of pathologists to review the slides from the NTP study.

James Bawden, representing the American Associations for Dental Research (AADR) at a peer review panel of the NTP study, claimed that the types of bone tumors observed in the rat differ from osteosarcoma observed in humans, and thus the NTP study has no relevance to humans (4). However, Bawden's statement represents a fundamental misunderstanding of the purpose of the 2 year bioassay: it is not a model for a specific cancer at any particular site in the body. The purpose of the bioassay is only to show the potential for a chemical to induce cancer; correlation of cancer sites in the animal and humans is not required and indeed, is rarely observed. John Stamm, representing the American Dental Association at the peer review panel (5), expressed concern about how the NTP did its statistical analysis and suggested the data were not strong enough to call fluoride an "equivocal" carcinogen.

Proctor and Gamble (P&G) sponsored a 2 year bioassay in which rats (Sprague - Dawley) were administered sodium fluoride in food. Rats were fed 1.8, 4.5, or 11.3 mg fluoride per kg body weight each day in a low fluoride semi-synthetic diet. There were two control groups, one fed the low fluoride semi-synthetic diet and one fed normal rat chow (fluoride content not determined). The fluoride content of the drinking water was not reported. The study ended early because too many animals died in both the fluoride fed and control groups. There were multiple problems with this study, including that the semi-synthetic diet may not have provided the nutrients for normal growth and development and a virus is likely to have infected the animals (6).
The original laboratory conducting the P&G rat study reported one osteosarcoma in a low dose female and a few other tumors. The carcinogencity assessment committee of the FDA reassessed the data and found another osteosarcoma in a low dose female and one in a high dose male. Moreover, not all the animals were carefully examined for bone cancer and thus other tumors may have been missed. The FDA review concluded that ***"...there are flaws and uncertainties in the studies that keep them from providing strongly reassuring data"*** (6).

The P&G study was also conducted with mice. Osteomas (non-malignant bone tumors) were observed in all groups with a significantly higher incidence in the high fluoride dose group. However, the mouse study hasn't been deemed useful for risk assessment because the mice in both the treatment and control groups were infected

with a virus (C-type retrovirus), and it is suggested that the tumors were formed via an interaction between the virus and fluoride (7).

Human epidemiology studies

Many epidemiology studies examining possible associations of fluoride and cancer have been conducted. Some studies examine bone cancer or cancers at particular sites, others examine overall cancer incidence rates or cancer mortality rates. Few of the studies are of individuals; rather they look at effects of populations who are assumed to be exposed or not exposed to fluoride or fluoridated water. Only a few will be summarized here.

Early epidemiology studies compared cancer mortality (death) rates in cities with and without water fluoridation. An analysis by Yiamouyiannis and Burk in 1977 found 4-5% lower death rates in non-fluoridated cities (comparison of 10 largest US cities with fluoridated water and 10 largest US cities without fluoridated water). At about the same time, three British scientists completed an analysis of the same 20 cities and found no effect of fluoridation on mortality rates. A review of these two studies by the US National Research Council concluded that the differences could be explained by use of different data sets and analytical methodologies; the differences showed the relative insensitivity of the data and measurements (3). Yiamouyiannis disputes this and claims that the British scientists omitted data and made mathematical errors (12). Freni and Gaylor examined international trends in bone cancer based on incidence (not mortality) data in a study published in 1992 (8). In general, they found no relationship between water fluoridation and bone cancer with the possible exception of an increased risk for females in fluoridated areas of the United States. The study was weakened by lack of good exposure data; non-differential misclassification of exposure will lead to an underestimate of an effect. Freni and Gaylor (8) also demonstrated that mortality data is a far less reliable measure of bone cancer than incidence data.

Several small case control studies examining the relationship between fluoride and bone cancer have been conducted, with mixed results. One small study in New Jersey found that males under age 20 years who resided in communities with fluoridated water at the time of diagnosis had a higher osteosarcoma rate than those who resided in nonfluoridated communities (9). A small case control study of osteosarcoma and water fluoridation (among other factors) conducted in Wisconsin showed no association between osteosarcoma and residence in an area with fluoridated water at time of diagnosis (10). Both of these studies suffer from lack of explicit exposure data. Exposure classification is based on residence at time of diagnosis, which may or may not reflect exposure to fluoridated water for any period of time.

A larger case control study examining the association between fluoride intake (and water fluoridation) and childhood (less than 25 years old) osteosarcoma was conducted in New York State (11). This study included contacting both cases (or their parents) and controls, and asking questions related to fluoride exposure. The study found no association between total fluoride exposure and osteosarcoma for either males or females. A statistically significant risk (odds ratio) for osteosarcoma was found at the lowest level of water fluoridation for females, and for males and females combined (but not for males alone). However, the risk did not increase with increasing exposure to fluoride in the drinking water, and the risk at the higher water fluoridation exposure was not significantly elevated (11).

Yiamouyiannis examined the relationship between incidence of and mortality from bone cancer in males and water fluoridation using several US data sets (12). He reported an association between water fluoridation and bone cancer incidence and death from bone cancer among males under the age of 20. He also suggested there is a 30-60% increase in oral cancers because of fluoridation. Several problems are apparent with this paper. For example, for bone cancers, he assumed that only males would have bone cancer linked to fluoride, and then used females as an unaffected reference population. The validity of this assumption has not been proven; indeed, other data reviewed here suggest this is an incorrect assumption.

A recently published study from Okinawa, Japan reports a relationship between fluoride concentration in drinking water and mortality from uterine cancer (13). However, it does not seem that important variables, such as water chlorination, were appropriately taken into account. It is also noted that, in Okinawa, the fluoridated water ranged between 0.19 and 0.37 mg/l fluoride. These waters would generally be considered non-fluoridated in the U.S.

Summary of cancer data

The animal study conducted by the National Toxicology Program (NTP) provides evidence that fluoride causes osteosarcoma, a malignant bone tumor. Although the NTP concluded that its study gave "equivocal" results with respect to cancer, the background memos and documents suggest that the results are actually stronger than suggested by the report. Similarly, the Procter and Gamble study likely gave stronger evidence of carcinogenicity, notably bone cancer, than suggested in the summary statements.

That fluoride is associated with bone cancer is reasonable from the point of view of what is known about the effects of fluoride: fluoride causes the division of immature bone cells (proliferation of osteoblasts) and fluoride accumulates in the bone and thus can cause damage there. Fluoride has been shown to be genotoxic in numerous test systems which is another property that is associated with carcinogens (1,5). In other

words, the biochemistry, pharmacokinetics, and other toxicology studies support the view that fluoride maybe a bone carcinogen.

Epidemiology studies examining cancer in general and bone cancer in particular have been inconsistent. Studies using ecologic designs (the studies are based on cancer incidence or mortality for given geographic areas, not for individuals) have given conflicting results for cancer in general, for all bone cancer, and for osteosarcoma. The larger case-control studies do not show an association of fluoride or water fluoridation with bone cancer although at least one small study has shown an association. Most of these studies are handicapped by completely inadequate measures of exposure which would mask any effects that may be there because of non-differential misclassification of exposure. Given the widespread deliberate exposure of humans to water fluoridation and the suggestive animal data regarding cancer, especially osteosarcoma, it is incomprehensible why a large case-control epidemiology study with good measures of fluoride exposure has not been initiated.

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Metabolic & Enzymatic Effects

This section discusses the impact of fluoride on metabolic and enzymatic processes. Included in these areas are the direct action of the fluoride anion as well as that of other inorganic fluorine-containing materials related to the process of fluoridation. In addition, the impact of fluoride on biochemical pathways and/or enzymatically controlled processes based upon either the formation of fluoride-metal complexes or upon the interference caused by fluoride in the interruption of enzyme-substrate spatial arrangements is discussed.

Background Material

Although it is beyond the scope of this report to present a pedagogical background in biochemistry or the chemistry of fluorine-containing compounds, a few principles should be discussed in order to understand the issues involved and the degree to which these issues would have had an impact on the widespread introduction of fluoride into the human food chain.

Fluorine is contained in significantly fewer than 10 % of more than 700 minerals. Of these, only 5 or 6 minerals are truly common and almost all of these are either insoluble or have very limited solubility in water of neutral pH, although some exhibit enhanced solubility in water in the lower pH (acidic) range. (1)

In those areas of the world where there is an abundance of the common fluorine-containing minerals in contact with either ground or surface water below pH 7, dissolved fluorine-containing minerals will be present in the indigenous water supplies. As a result, those areas will have an increased presence of fluorine in the vegetable and animal food-stuffs produced there. The fluorine that does enter the human food-chain, whether naturally occurring or as a result of artificial fluoridation, corresponds primarily to the sodium salt of the fluoride anion (F^-) and either sodium fluorosilicate or fluorosilicic acid. Clearly it is the nature of these materials which most concern us in this section and, in addition, the nature of the biological materials with which these interact. (2)

Characteristics of Fluorine and Fluoride Ion

The primary action of fluoride in metabolic and enzymatic reactions is related to the formation of *"complexes"* in one form or another. The fluoride anion has the highest charge density of any negative ion. (3) As a result of this, it is now known that fluoride forms an exceptionally strong hydrogen bond (> 148 kJ/mol.) with substrates in amide-fluoride systems. (4) Strong hydrogen bonding is now recognized as being clearly distinguishable from normal hydrogen bonding.

Another related characteristic of fluoride ion is that it exhibits an affinity for many metal ions, especially magnesium, manganese, aluminum, and calcium and therefore it can effect the bioavailability of these ions either separately or may cause either inhibition or otherwise interact with any enzyme system which requires one of these metals as a co-factor. (5,6)

Structural Susceptibility of Biomolecules

The impact of strong hydrogen bonding is that proteins, which consist of a repetitive sequence of amide linkages, are particularly susceptible to this type of hydrogen bonding. The end results of this type of interaction are two-fold. The lesser effect is that the carbonyl-nitrogen (amide) bond in proteins may become more susceptible to cleavage even though fluoride itself is a less nucleophilic anion. The second, and probably enormously greater, effect is that the spatial arrangement or macromolecular structure of these materials depends heavily upon normal hydrogen bonding to produce the secondary stereochemical structure required for appropriate enzymatic activity to take effect. (7) This has been demonstrated by Edwards and co-workers, who studied the perturbations caused by fluoride on the structure of Cytochrome C peroxidase. (8) Further, *ab initio* calculations by Emsley et al. lead to the conclusion that the fluoride ion may completely disrupt the Thymine-Adenine linkage in DNA. (9) A survey of the literature reveals no shortage of supporting research results. (10) The conclusions reached in several of these studies are listed below.

- Fluoride inhibits metalloproteins (12)
- Fluoride inhibits DNA polymerase (13)
- Fluoride induces chromosome aberrations (14)
- Fluoride effects the adenyl cyclase system (15)
- Fluoride inhibits yeast enolase (16)
- Fluoride inhibits protein synthesis enzymes(17)
- Fluoride inhibits gycolytic enzymes (18)
- Fluoride inhibits cell growth enzymes (19)
- Fluoride inhibits testosterone synthesis (11)

It is of interest to note that the latter interaction may be responsible for those deleterious effects of fluoride which appear to be restricted to males (e. g. testosterone is involved in bone growth in males but not in females). (11) The above list is by no means exhaustive. Rather, it should be taken to indicate that there is sufficient evidence to warrant more extensive research into this area. However, over all, the results described in the above references *"suggest that sodium fluoride is potentially dangerous to humans." (14)*

Fluoride and Calcium Metabolism

The interaction of fluoride in those metabolic processes involving calcium are also of great significance. This type of interaction may have been responsible for the recent observation that even when calcium is supplemented in osteoporotic patients, a large number of those who have also been treated with fluoride still show evidence of calcium deficiency. (20) The lack of availability of calcium, either as a result of precipitation by fluoride or the formation of fluoroapatite, may result in hypocalcemia which may have other widespread and, as yet, poorly understood effects on bone formation and other regulatory mechanisms of the body.

Summary and Conclusion

The information above and the references cited illustrate that fluoride can seriously disturb the balance of enzymatically activated biochemical reactions. These effects clearly were not well-known at the commencement of fluoridation activities. However, the recent literature contains many references (e.g. 21-25) to original research results that illustrate that fluoride effects the metabolism of a number of common oral bacteria, (e.g., Streptococcus mutans) so that, equally clearly, this phenomenon of fluoride effects on enzymes should be as well known to the proponents of fluoridation as to anyone. Thus, while there can be no doubt that fluoridation has contributed to the reduction of dental caries in the past, there is likewise little doubt that the continuation of the fluoridation process in the light of recent evidence outlined above is inappropriate without first answering the serious and potentially health-effecting questions raised.

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Hypersensitivity and "Allergic" Reactions

Some humans appear to be hypersensitive to fluoride, although there is some question about whether the reaction is allergic. At the very least, some components of the hypersensitivity appear to be allergic (i. e. an immune system effect); other portions may be related to the central nervous system or altered fluoride metabolism (pharmacokinetics). However, from the viewpoint of the sufferer of these effects, it is a moot question since the end result is virtually the same. With apologies to the sufferers and those physicians who use the terms *"allerg-(ies,ic)"* in their discussions or papers, this section refers to the manifestations of these effects as hypersensitivity.

Hypersensitivity reactions to fluoride, including fluoridated water, have been known to and reported by medical practitioners for decades (1). A search of the recent literature identified several references to occupational asthma induced by fluoride exposure in the aluminum industry (aluminum potroom asthma)(2), but no references to environmental exposures. While this absence of recent literature suggests that this is not an active area of current research, it does not invalidate the older observations.

George Waldbott, M. D., summarized both the medical literature and his own observations on the allergic reactions to fluoride in a 1964 article in the Journal of Asthma Research. He reported six cases of urticaria (hives) due to fluoridated water. The urticaria was accompanied by other fluoride associated health effects, including paresthesias, cephalgia (headaches), arthritis in the lower spine, gastrointestinal and urinary disturbances. For at least some of these patients, the association of the urticaria with fluoride was demonstrated in double blind challenge tests. These patients appeared to retain more fluoride than most individuals, putting them at higher risk of fluoride-associated health effects. In the same journal article, Waldbott also described other effects on fluoride on the skin of sensitive individuals. These included atopic dermatitis and contact dermatitis, including on the fingers of dentists after applying sodium fluoride to patients.

Summary

This paper demonstrates that there is a sub-population of adults that is hypersensitive to even low doses of fluoride such as those in water fluoridated to 1 ppm. While the size of this sub-population is unknown, there appears to be at least one Natick resident who is hypersensitive to fluoridated water.

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Central Nervous System: behavioral and IQ effects

Several papers published in the last few years report that fluoride has adverse effects on the central nervous system (CNS), including intelligence and behavioral patterns. These papers encompass biochemical, histological, animal, and human studies and give a consistent picture regarding previously untested adverse consequences of fluoride exposure. Four important features of the animal toxicology and human studies are:

- 1) the fluoride doses are in the range that some humans actually receive; the animal studies are in the range of the upper end of fluoride food and water intake in the U. S.;
- 2) for some effects, the timing of the dose is critical, prenatal and early life exposures appear to be the critical periods for IQ deficits and some behavioral changes,
- 3) the adverse effects due to prenatal exposures are not reversible, and
- 4) the adult onset symptoms may be reversible if fluoride exposure is eliminated.

Animal Toxicology Studies

Dr. Phyllis Mullenix and co-workers published a study on the neurotoxicity of sodium fluoride in rats in 1995. (1) The study used behavioral methodology that focused on behavioral repertoire, responses to novelty, and the temporal or sequential organization of spontaneous behavior. This methodology had been previously used to study alterations in CNS function and behavioral alterations including cognitive deficits (mental retardation) due to chemotherapy for childhood acute lymphoblastic leukemia (2), amphetamine induced hyperactivity, and triethyltin-induced hypoactivity. (1) Thus, the methodology used to test the sodium fluoride should be considered a validated one.

In all, 532 rats (Sprague-Dawley, male and female) were used in the study. Fluoride was given at different doses and three life stages: prenatal, weanling and adult. Prenatal doses were administered via subcutaneous injections of the pregnant dam on either gestational days (GD) 14-18 or GD 17-19. The dose schedules produced plasma fluoride peaks of 0.15 to 0.2 ppm. Weanling and adult rats were dosed via incorporation of 75 ppm, 100 ppm or 125 ppm sodium fluoride in drinking water for 6 - 20 weeks. While these drinking water concentrations are higher than would be used in water fluoridation, the cumulative exposures to fluoride, as measured by the concentrations of fluoride in plasma, are similar to plasma levels observed in humans with high end exposures. For example, the plasma level of fluoride in the rats ranged from 0.059 - 0.640 ppm. Plasma fluoride levels of 0.076-0.25 are found in humans ingesting 5-10 ppm fluoride in drinking water and plasma fluoride levels of 1.44 ppm

have been found in children 1 hour after receiving topical applications of acidulated phosphate fluoride gel.

The prenatal exposures (also referred to as *in utero* exposures) during GD 17-19 altered the behavioral outcome in male (but not female) offspring in a manner correlated with hyperactivity. There was no overt toxicity to the dams or offspring based on reduced body weight, suggesting the behavioral alterations were not secondary to another toxicity. [Note that absence of overt toxicity in the dam is a key test in defining a developmental toxicant according to EPA guidelines.] (3) Plasma fluoride concentrations at 3 and 9 weeks of age were not elevated, suggesting that the damage occurred *in utero* and as a result of a short term exposure. It should be noted that the nervous system develops throughout gestation and during the early postnatal period, and that the higher cognitive functions develop towards the end of gestation (gestation period is 20-21 days in rats) and during the postnatal period. It is also a common feature of developmental toxicants that adverse effects are observed if a toxicant is given during one period of gestation, but not during others (other adverse effects may or may not be observed at other periods).

Weanling exposures to fluoride affected the behavior of both males and females in a dose dependent manner (based on plasma fluoride levels), although the female rats were affected at lower doses. These doses also induced slight toxicity as judged by body weight gains. The behavioral changes for both sexes and at all doses were consistent with respect to the controls, and were different from the behavioral changes observed in male rats exposed prenatally. The observed behavioral changes are associated with cognitive deficits in other studies (1).

Adult rats were exposed for 6 weeks to 100 ppm fluoride in addition to the no fluoride control. No toxicity was associated with this dose based on differences in body weight. Female (but not male) rats showed behavioral changes, and these changes were similar to those observed in the weanling exposures, namely cognitive defects (1).

A study by Liu published in 1989 (4) appears to show behavioral changes associated with *in utero* exposure to fluoride, although only the abstract is available in English. Female Wistar rats were given 0, 30 or 60 ppm NaF in drinking water, apparently for 85 days before and during pregnancy. Their offspring were tested (age 33-42 days) for pain reaction and conditioned reflex. The response time for the fluoride exposed offspring was longer than for the no-fluoride control animals.

Behavioral Changes in Humans Associated with Exposure to Fluoride as Adults

Spittle (5) summarized several studies that reported central nervous system effects in humans following occupational or environmental exposures to fluoride. About 25% of workers exposed to fluoride from cryolite (a fluoride-containing mineral) who had

skeletal fluorosis also had central nervous system effects including fatigue, headache and giddiness. A similar proportion of aluminum smelter workers with skeletal fluorosis also reported psychiatric disturbances including depression, mental sluggishness and memory disturbances. Although these observations are reported for people with high fluoride exposure, the effects from occupational exposures are often used to forewarn of hazards that may also occur, but be harder to measure, at lower doses such as those that may result from environmental exposures.

There are also several studies where behavioral changes or other CNS symptoms are associated with lower fluoride exposure. Waldbott (summarized in Ref. 5) reported generalized progressive fatigue associated with a distinct decline in mental acuity in persons residing within 3 miles of an enamel factory emitting hydrogen fluoride. Waldbott also reported CNS symptoms (lethargy, memory impairment) in several patients exposed to fluoridated drinking water. Some of these studies are also described in this report in the section titled hypersensitivity. (5 ,6)

Rotton and coworkers (7) subjected adult volunteers to experiments that tested their attention and error rate on primary and secondary tasks (tracking objects and responding to flashing lights). The individuals were given one drop of sodium fluoride in water (0.1, 1, 10, 100 ppm) sub-lingually. The administration of sodium fluoride did not affect the primary task; tracking a moving target. However, the sodium fluoride increased the error rate (missed responses) of the secondary task and the highest two doses resulted in an increase in the latency (response time) between the secondary stimulus and the subject's response.

Decreases in IQ in humans exposed to fluoride *in utero*

Cognitive deficits due to fluoride exposure, in the form of a population-wide decrease in intelligence in children, have been reported in several different populations in China in the last few years. Two of the studies were published in English (8,9), and they are summarized here.

Zhao and co-workers (8) studied the differences in IQ in children aged 7-14 in two villages in Shanxi Province of China. The villages were similar with respect to occupations, living standards and social customs, but differ with respect to the fluoride content of drinking water. Sima has a fluoride content of 4.12 ppm, 86% of the population has clearly evident dental fluorosis, and 9% have clinically diagnosed skeletal fluorosis. Xinghua has a fluoride content of 0.91 ppm, 14% of the population has dental fluorosis and no skeletal fluorosis has been diagnosed. In each village, 160 (80 male, 80 female) randomly selected children were given standard IQ tests. The only constraint was that the children's mothers lived in the village being studied during their pregnancy. The mean IQ in the Sima (high F) and Xinghua (low F) were 97.7 and 105.2, respectively, which is a statistically significant difference. The range was also generally lower in the higher fluoride area.

Table I - IQ Ranges for Different Exposures to Fluoride (8)

Village	Mean IQ	Range
Sima (high F)	97.7	60-133
Xinghua	105.2	69-141

The IQ distribution of children in Sima was lower than in the low F village of Xinghua, leading to fewer children in the superior intelligence category and more children in the low intelligence category in the high fluoride village. There was no difference in IQ between males and females. As expected, within each village, IQ correlated with parents' educational levels.

The high F village of Sima had fluoride concentrations only just above that allowed by US standards (MCL of 4.0 ppm) and the low F village of Xinghua had a fluoride content (0.91 ppm) slightly lower than the proposed fluoridation level in Natick. However, these data do not suggest that 0.91 ppm is without effect, as no village with lower drinking water fluoride concentrations were tested. It is also important to note that the study required *in utero* exposure to these levels of fluoride.

Li and co-workers (9) conducted IQ tests on children living in four areas of the Guizhou Province of China. The areas differed with respect to endemic fluorosis caused by coal burning for grain drying, but were otherwise similar in cultural and socioeconomic aspects. Children aged 8-13 were tested (total of 907) using a Chinese IQ test for children in rural areas. Dental fluorosis was measured using Dean's scale of DMF. Urinary fluoride was also measured and correlated with the measured dental fluorosis (Table II, below).

The results of this study in terms of mean IQ scores and the distribution of IQ scores are summarized in the two tables below.

Table II

Mean IQ scores for children in areas with different prevalence of fluorosis (9)

Degree of Fluorosis	none	slight	medium	severe
No. of children	226	227	224	230
Dental Fluorosis Index	<0.4	0.8	2.5	3.2
Urinary F (mg/L)	1.02	1.81	2.01	2.69
IQ (mean±SD)	89.9±10.4	89.7±12.7	79.7±12.7	80.3±12.9

Table III
Distribution of child IQ scores from areas of differing fluorosis prevalence (9)

	IQ Range						
Fluorosis Status	<70	70-79	80-89	90- 109	110- 119	120- 129	>129
none	2.6%	9.7%	37.1%	46.8%	3.9%	0.8%	0
slight	3.1%	15.9%	29.1%	47.1%	3.1%	1.3%	<0.4
medium	25.4%	23.7%	29.9%	20.5%	0.4%	0	0
severe	20.9%	26.6%	26.9%	25.2%	0.4%	0	0

Inspection of the first table indicates that there is a 10 point IQ drop in the medium-severe fluorosis area compared to the non-slight fluorosis areas. Inspection of the second table shows that the decrease in IQ is throughout the "bell shaped" IQ curve. There is a marked increase in the percentage of children with IQ less than 70 in the medium-severe fluorosis areas (approximately 3% to more than 21%) and a marked decrease in the percentage of children in the higher IQ ranges (for example, IQ greater than 110 decreases from approximately 5% to 0.4%, a ten-fold decrease).

No correlation was observed between IQ decrement and age of the children. As pointed out by the authors, this suggests that early exposure (in utero or early postnatal) to fluoride is critical to the production of the adverse effect.

Biochemical studies of the brain

The findings of central nervous system effects (behavior changes and decreased IQ) in the human and animal studies following fluoride exposure is supported by biochemical data that show that fluoride accumulates in both fetal and adult human brain tissues. In other words, it can be shown that the fluoride reaches the brain tissue, and thus is available to exert an effect.

Mullenix and coworkers (1) measured the concentrations of fluoride in various regions of the brain of both weanling and adult animals exposed to fluoride in their behavioral studies. They detected increased fluoride concentrations in the hippocampus of females, but not males exposed as adults, and of both females and males exposed as weanlings. This pattern of elevation of fluoride in the hippocampus is the same as the pattern of behavioral changes. Several studies have linked hippocampal damage and hyperactivity and cognitive deficits.

Alterations in the hippocampus section of the brain following ingestion of sodium fluoride in drinking water have also been reported by other researchers (10) using a different rat strain (Long Evans) and different measurement endpoints [abnormalities

in the hippocampus and alterations in biochemical reactions in the brain such as beta amyloid and IgM antibody].

Additional studies along these lines have been published in the Chinese literature, but only abstracts or other summaries are available in English. Higher concentrations of fluoride have been found in human embryonic brain tissue obtained from termination of pregnancy operations in areas where fluorosis due to coal burning was prevalent. Detailed studies of these tissues showed that differentiation of brain nerve cells was poor and brain development delays (cited in reference 9). Li (4) reports that the brain of rat pups whose mothers had been exposed to 60 ppm NaF in drinking water had higher nerve cell density in the brain and mild degeneration of organelles of the nerve cells compared to pups from control dams.

Summary and Conclusions Regarding CNS Effects

The study conducted by Mullenix et al., shows central nervous system changes in rats that are likely to be observed as hyperactivity and decreases in IQ or other cognitive (thinking) functions in humans. The observed change depends upon whether the fluoride was administered prenatally or after the pups were born. The observed changes also depended upon whether the animal was male or female. This is a very well conducted study using a previously validated test system, and fluoride doses within the range that humans receive. A lot of weight should be placed on the results of this study.

The two Chinese epidemiology studies suggest that fluoride exposure sufficient to produce moderate to severe dental fluorosis also results in substantial IQ decrements if the fluoride exposure occurs in utero or during the early postnatal period. Taken together, the studies indicate that total fluoride exposure is critical: the IQ decrements were observed due to both drinking water and inhalation exposures. These findings are quite consistent with the animal toxicology data published by Mullenix et al. (1)

Biochemical and histological studies show the accumulation of fluoride in fetal and adult brain tissue and fluoride-induced changes of the stucture of brain tissue. These studies support the animal and human studies that fluoride adversely effects human behavior and cognitive function by showing that flouride reaches brain tissue and alters its appearance.

The Chinese IQ studies (8, 9), the animal toxicity study by Mullenix et al. (1), the studies summarized by Spittle (5), and the biochemical and histological studies together very strongly support the proposition that fluoride has adverse effects on the human central nervous system. Moreover there is good evidence that fluoride is a developmental neurotoxicant (1, 8, 9), meaning that fluoride effects the IQ and behavioral patterns of the developing fetus at doses that are not toxic to the mother.

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Reproductive and Developmental Toxicity (other than nervous system)

Reproductive toxicity is the study of toxic effects on the reproductive capacity of males and females. Animal toxicity tests to determine whether or not a substance is a reproductive toxin include:

1) alterations in sperm count and quality;
2) number of litters and number of conceptuses/litter when male or female animals are exposed to a potential toxicant prior to mating; and
3) number of live births when male or female animals are exposed to a potential toxicant prior to mating.

Human epidemiology studies of birth rates may also give insight into reproductive toxins.

Developmental toxicology is the study of conditions (including chemical substances) that lead to abnormal development. Manifestations of developmental toxicity include structural malformations (birth defects), growth retardation, functional impairment and death of the organism. The study of developmental functional deficits, including neurobehavioral effects has emerged in the last twenty years (1), and is thus still in its early years of elucidation.

Reproductive Toxicity

There have been a number of studies of the effects of fluoride ingestion and of water fluoridation on reproductive capability of humans and animals. In its influential 1991 review of water fluoridation, the US Public Health Service (US PHS) (2) found that fluoride may affect reproduction in animals, although some data were contradictory. [It should be noted that the US PHS report was provided to this committee by both Myron Coplan and the Natick Board of Health.]

Several laboratory studies of rodents (rats and mice) exposed to fluoride in food or drinking water showed reduced fertility. Heifers exposed to 5 ppm fluoride in water during four breeding seasons calved at a rate that was only 30% of normal. At higher fluoride doses, the effect was earlier and more severe, which is strongly indicative that the effects observed were due to fluoride and not a confounding factor (cited in 2, 7).

In screech owls, chronic dietary intake of 40 ppm sodium fluoride resulted in significantly smaller egg volume, which is considered a slight-to-moderate reproductive disorder. No gross abnormalities were apparent. (3) Pastel mink fed up to 230 ppm fluoride in their diet did not show adverse reproductive effects such as changes in breeding, gestation, whelping or lactation. However, there was only a 14% survival rate of kits whelped by females fed 385 ppm fluoride. (4)

Several animal studies have examined the effect of fluoride on sperm count, motility and other sperm quality parameters. Narayana and Chinoy (5) fed albino rats 10 mg/kg sodium fluoride for 50 days, and examined the structure and metabolism of the sperm. They observed biochemical alterations that manifest themselves in reduced sperm motility and lower sperm count. Both of these are considered adverse reproductive effects. Withdrawal of sodium fluoride reversed most, but not all of the observed alterations. Addition of ascorbic acid and calcium to the rat diet after withdrawal of the sodium fluoride produced full recovery from the adverse effects of the sodium fluoride.

Susheela and Kumar (6) fed rabbits 10 mg/kg sodium fluoride for 18 or 29 months. At the end of the exposure period, the animals were sacrificed and the structure of the testis, epididymis and vas deferens studied by microscopy. Deleterious changes were observed after 18 months, including absences of mucus droplets in the vas deferens and changes in the epithelial cells lining the lumen of various structures. Spermatogenesis ceased in animals treated for 29 months, but not 18 months, suggesting that longer exposures to fluoride result in more severe effects.

Because of the lack of any human epidemiology studies, Stan Freni, a participant in the US PHS (2) review, initiated an epidemiological study of the possible association of fluoride concentrations in community water supplies and US birth rates. (7) Freni calculated the annual total fertility rate for white women in the age range 10-49 years for the period 1970-1988 in 30 regions (somewhat equivalent to counties) in 9 states. He compared the total fertility rates with measures of fluoride concentrations in drinking water (up to 10 ppm in some individual systems, but averaged over all the drinking water in the county), the percentage of people drinking highly fluoridated (>3 ppm) water, and various socioeconomic factors that are known to affect fertility rates. After accounting for the socioeconomic and other demographic factors, Freni found an association of decreasing total fertility rate (low birth rates) with increasing water fluoride concentrations for most, but not all, of the regions examined. (7)

Developmental Toxicity (other than neurotoxicity)

This subsection describes two animal experiments that tested the effect of fluoride given to pregnant mice. The endpoints studied were various aspects of health, growth and birth defects of the fetus. However, neither study examined functional deficits, such as neurobehavioral deficits. These are fully described in the neurotoxicity section of this report.

Collins and coworkers (8) published a study on the effects of sodium fluoride in drinking water provided to pregnant rats on the health of the fetuses. In this study, they dosed pregnant female rats with drinking water containing 0, 10, 25, 100, 175 or 250 ppm NaF every day throughout gestation. The NaF did not appear to produce any change in fetal growth or affect the development of specific bones. However at

the highest dose (250 ppm NaF), there was a significant increase in the average number of fetuses with three or more skeletal variations.

Heindel and coworkers (9) evaluated the effects of sodium fluoride in drinking water provided to pregnant rats and rabbits on the health of the fetuses at the end of gestation. In this study, they dosed rats with 0, 50, 150, or 300 ppm NaF in drinking water during gestational days 6-15; rabbits were dosed on GD 6-19 with 0, 100, 200 or 400 ppm NaF in drinking water. The animals were killed and the fetuses were examined at the end of gestation (GD 20 for rats, GD 30 for rabbits). No clear signs of maternal toxicity were noted at the 150 ppm level and lower. No developmental effects, manifest as post-implantation loss, mean fetal body weight/litter, external, visceral or skeletal malformations were observed.

Summary

Regarding fluoride and reproductive effects: Taken together, the studies summarized here raise serious concerns about the impact of fluoride on human reproduction, even at water fluoridation levels currently considered "safe". The human epidemiology study conducted by Freni (7) does not prove that fluoride in drinking water decreases fertility. However, the association observed in the study is a serious cause of concern, especially because of its consistency with some observations in laboratory and farm animals. It clearly shows the need for careful studies that are designed to ascertain if water fluoridation decreases human fertility.

Regarding fluoride and developmental effects: These two studies do not show any fluoride associated developmental effects such as malformations (birth defects), post-implantation loss, or death of the fetus or infant at drinking water doses up to 175 ppm sodium fluoride. There may be some effects above 250 ppm sodium fluoride, particularly skeletal related effects. The experimental protocols used in these two studies do not test for cognitive or neurobehavioral changes following *in utero* exposure, such as those observed by Mullenix and coworkers described in the neurotoxicity section.

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Lead Contamination

This section discusses the impact of specific concerns about the role of fluoridation relative to the issue of lead contamination.

Lead Contamination

Lead contamination is a recognized concern in water supplies. As such, it is regulated by the EPA. This regulation stipulates that certain action(s) must be implemented if the lead concentration exceeds 15 parts per billion (ppb) which is termed the action level. The 1986 Safe Drinking Water Act Amendment prohibits the use of lead pipes and limits the lead content in brass plumbing components to 8%. Older facilities are likely to have a larger amount of lead used throughout the water distribution system.

Although the lead level in ground and surface water supplies may be low, the level of lead can increase to the action level (as specified by the EPA) depending on chemical and physical factors. The factors affecting the amount of lead contamination are: 1. The corrosiveness of the water which is dependent on pH, alkalinity (or buffering capacity), and mineral content, 2. Age of lead-soldered joints and other lead components, 3. Quantity and surface area of lead materials, 4. Time and temperature of water in contact with lead surfaces. (7)

The health effects of lead can be as severe as coma and possible death at high blood levels (exceeding 80 ug/dL). Severe effects are unlikely to occur from drinking water. Low level lead exposure (as determined by blood lead levels greater than 10 ug/dL) is more likely to occur from drinking water. Low level lead exposure is associated with adverse effects on the central nervous system such as decreased intelligence and impaired neurobehavioral development. (7)

Corrosion is one of the mechanisms by which lead contamination increases in the water supply. The fluoridating agents Natick intends to use, fluorosilicic acid and sodium fluorosilicate, are both corrosive in certain conditions. As stated in the Water Fluoridation Manual for Plant Operators (1):

> *".....Under certain water-quality conditions, a small increase in the corrosivity of potable water that is already corrosive may be observed after treatment with alum, chlorine, fluorosilicic acid, or sodium fluorosilicate. This increase in corrosivity is caused by a depression of pH resulting from these treatments and occurs in potable water with a low buffering capacity. The increase in the corrosivity of potable water as a result of the addition of the fluorosilicic acid or sodium fluorosilicate is negligible for most water systems, but where it is significant, it can be reduced by adding small amounts of lime or caustic soda".*

An example of the relationship between increased lead levels associated with fluoridation occurred in 1992 in Tacoma, WA. Data from the City of Tacoma water treatment plant show water sampling parameters from the same neighborhood before and after the fluoridation equipment broke down (2). The pH was identical at 6.6 in both cases, yet with fluoridation 20% of the homes exceeded the EPA action level for lead, whereas 10% of the homes exceeded the level without fluoridation.

An important aspect of this corrosivity effect requires a consideration of the dissociation of sodium fluorosilicate, which takes place as follows: (1, 5)

$$Na_2SiF_6 \rightarrow 2Na^+ + SiF_6^{2-}$$

Further dissociation of fluorosilicate ion takes place utilizing different pathways:

$$SiF_6^{2-} + 2H_2O \rightarrow 4H^+ + 6F^- + SiO_2\ (s)$$

$$SiF_6^{2-} \rightarrow 2F^- + SiF_4\ (g)$$

$$SiF_4\ (g) + 3H_2O \rightarrow 4HF + H_2SiO_3$$

$$SiF_4\ (g) + 2H_2O \rightarrow 4HF + SiO_2\ (s)$$

$$HF \rightarrow H^+ + F^-$$

Similar to sodium fluorosilicate dissociation, fluorosilicic acid dissociation occurs as follows:

$$H_2SiF_6 \rightarrow 2HF + SiF_4\ (g)$$

$$SiF_4\ (g) + 2H_2O \rightarrow 4HF + SiO_2\ (s)$$

$$SiF_4\ (g) + 3H_2O \rightarrow 4HF + H_2SiO_3$$

$$HF \rightarrow H^+ + F^-$$

These reactions do not occur at equal rates, however the end products are fluoride ion (F^-), silicon compounds, and hydrogen ion (H^+; hydrated). The rates of reaction are of concern when considering corrosion effects, since the initial reaction will release some hydrogen ions which will lower the pH, increasing the acidity. The initial pH drop can be neutralized at the water plant; however, the slower breakdown of intermediate products like silicon tetrafluoride will happen gradually, quite likely after leaving the plant. As a result of this delayed hydrolysis of the silicon compounds, increased acidity will be experienced thoughout the water distribution system. (6)

A results of a study of water fluoridation agents (if any) and the venous blood lead levels of children of ages 0 to 4 years is shown in Table I, below. (3, 4, 8, 9)

These data illustrate that the reported blood levels exceeded the limit of 10 ug/dL in 0.75% of the children in non-fluoridated communities while more than twice as many (1.53%) of the children in the fluorosilicic acid-fluoridated communities exceeded the recommended limit. (3) The communities represented are comparable in size to Natick, ranging from 15,000 to 50,000.

Table I
Fluoridation and Venous Blood Lead Levels in MA Children Aged 0-4 Yrs.

Percent of Children with Venous Blood (VB) Levels Greater than 10 Micrograms/Deciliter (3,4,8,9)

Number of Communities	Total Population	Number Screened	Number with VB > 10 ug/dl	Incidence n/N	Fluoridation Agent Used
	(thousands)	N	n	%	
36	882.8	40669	306	0.75	None
20	416.0	17441	181	1.04	NaF
30	865.3	36804	564	1.53	H_2SiF_6

Summary

Fluorosilicic acid and sodium fluorosilicate are acknowledged to have corrosive abilities. Even when maintained diligently, an increase in lead levels should be anticipated at point-of-use in homes after exposure to the distribution lines where lead solder and valves are in contact with water. The use of fluorosilicic acid and sodium fluorosilicate poses a specific risk since they have been associated with increased blood lead levels.

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Non - Health Issues

Although the Committee was not specifically asked to address any other issues related to fluoridation, the members felt that certain of the following matters would/should bear upon the decision of the Natick Board of Selectmen vis-à-vis the overall question of fluoridation.

Forced Medication/Freedom of Choice

It has been said that ***"....fluoride, at the levels recommended by profluoridationists for reducing tooth decay is not an essential nutrient; is not a natural substance for babies or for most adults; is not a compulsory medication; but is an expensive-to-avoid medication with an uncontrolled dose; and is harmful to some people."*** (1) As a result of these characteristics, the issue of fluoridation cannot be properly discussed without a concomitant discussion about the ethical issues involved. (1-3). In spite of this, the position taken by most profluoridationists is that the questions involved are of a scientific nature only and therefore should be addressed only by those well-versed in these issues. (4-6) However, a powerful case can be made that there are indeed ethical questions involved and that **perhaps these issues should be addressed first and not merely by the technically elite.** The obligation that this imposes upon any government official, including the Board of Selectmen, cannot be minimized. It is therefore appropriate that the Committee recommend that a careful reading of reference (1) below should precede **any action** taken in the resolution of the Natick fluoridation issue.

Economic Issues of Fluoridation

The issue of economic costs in a massive program such as the fluoridation of a public water supply is difficult to treat exhaustively. Moreover, the Committee has no great degree of financial expertise. Nevertheless, we would be remiss if we failed to identify those costs which come to mind. These are treated in the following sections.

Direct Process Costs

The Natick water department expects to pump approximately 1.5 **billion** gallons of water this year to Natick consumers. Of this amount, approximately 60 **thousand** gallons will be used **weekly** (0.208 per cent) in the preparation of food and for drinking purposes. (7) Fluoridation of 1.5 billion gallons of water to the 1 ppm level will require 15,720 lbs. of H_2SiF_6. (7) The projected annual cost for fluoridation of the Natick wells is believed to be a minimum of $35,000 per year. (8) Other estimates range from $30,000 to $50,000 per year. Moreover, it is not clear whether these costs

take into consideration such factors as training of personnel, amortization costs, repairs and replacement of both privately owned and town-owned equipment due to increased corrosion (unrelated to lead issues) which is known to occur. (9) From these facts, two interesting pieces of data emerge. These involve the issues of environmental impact and the reputed cost effectiveness of the fluoridation effort.

Cost-effectiveness of Fluoridation

Public health officials have always considered fluoridation of public water supplies to be a cost effective approach to giving children fluoride. (10) However, the economics have changed in the last few decades. We feel that it deserves a closer look and we note some concerns below.

Two important factors need to be taken into consideration when considering the 'savings' attributable to reduction in caries caused by fluoridation. First is the fact that modern DMFS scores (explained in Appendix D) are much lower in all communities than was the case when fluoridation was first started. (11) This means that there are fewer cavities in the population and that any percentage reduction in the incidence of cavities involves many fewer incidences than was previously the case. The second salient fact is that 55% of the children in communities having unfluoridated water are cavity free. (12) Clearly, fluoridation is not cost effective for this segment of the population.

The calculation of cost effectiveness of fluoridation is very complex. One has to consider the savings due to (possibly) fewer cavities in some children and the cost to treat those children. However, it is also true that there will be increased treatment costs due to dental fluorosis (between 10-30% of children in communities that fluoridate develop some form of dental fluorosis-see section on dental fluorosis). Although these costs are not borne by the community at large, they should be considered in any assessment of cost-effectiveness. (13)

It is beyond the scope of this committee to make such complicated calculations but is seems clear that there will be a greater increase in fluorosis than there will be a reduction in cavities.

Indirect Costs

The committee has also identified indirect costs that should be included in the cost effectiveness calculations. These include the costs borne by individual Natick residents who choose not to drink fluoridated water and individual Natick residents who may incur medical or dental costs due to drinking fluoridated water. Finally, there are other costs to the town such as amortization, repair, etc., of equipment necessary

to the program. These cost include (but are not limited to) the following identifiable items:

- Increased dental costs (not covered by insurance) to treat fluorosis
- Purchase of unfluoridated water from other sources ($3-4 per week)
- Purchase of fluoride removal equipment
- Increased medical costs
- Legal costs to the town to defend against lawsuits (see below)
- Increased plumbing costs resulting from corrosion. (9)

Liability Ramifications

If the town fluoridates its public water supply, there is a possibility that legal culpability may result from any number of sources. For example, if continued research into the correlation between fluoride and diminished IQ (or other factors) substantiates the research results described above (see the sections on Central Nervous System effects and Lead Contamination), the town may well be held liable. (That the town in this case would have unlimited company may be of little consolation!) In addition, in spite of the best efforts of the town, a hazardous spill may occur, as has already occurred in several other communities. In at least one of these incidents, multiple lawsuits have been filed. (14)

Environmental Impact

In the process of fluoridation of Natick's water supply, 15,680 pounds of fluoride per year will enter the environment (assuming 50% fluoride retention within the human body). This fluoride will be dispersed via a number of mechanisms into a variety of paths including incorporation into locally grown foods and locally raised livestock. It is easy to see how the presence of fluoride has become so endemic that many researchers have postulated that it is no longer possible to determine whether fluoride in public water supplies has any value, without considering how to quantify this effect. (13)

In order to place this issue in proper perspective, it is insightful to consider that the effect of having the entire United States being served with fluoridated water will result in the dumping of at least 100 million pounds of fluoride annually within the United States alone! Needless to say, for any other material, beneficial or otherwise, a far greater public outcry would be raised.

Political Ramifications of Referenda and Plebiscites

The town of Natick appears to be deeply divided on the subject of fluoridation. The issues of a "*binding*" vs. a "*non-binding*" referendum; the **staledatedness** of a popular

vote and the question of whether a small majority of a larger number of voters is more valid than a larger majority of a smaller number of voters in a plebiscite are questions that interest all of the parties concerned, including the members of the Committee. However, it seems clear that since neither the Committee nor the various parties in the fluoridation issue will resolve these questions, we will not speculate on the legal opinions which may be rendered thereon. On the other hand, certain valid points can be made.

First of all, the information and research about fluoridation and the effects of fluoride has grown tremendously in the past few years. When the first vote on fluoridation was taken in Natick, much less was publicly known about the possible negative effects of fluoride and the decreasing impact of fluoridation of drinking water. For this reason we feel that more attention should be paid to the latest vote in Natick in which the voters failed to support the fluoridation of Natick drinking water.

Secondly, even if there is a possible reduction of caries in Natick due to fluoridation, this has to be weighed against the possible harm caused to some number of residents of the town due to increased fluoride in their diets. It has been argued that fluoridation of the drinking water is the most cost effective method of getting additional fluoride into the diet of children. However, this cost savings (if indeed there is a savings) has to be weighed against the increased cost of medical care for those who may be negatively effected by an increase in fluoride.

Summary of Non-Health Issues

Fluoridation of the Natick water supply has multiple implications beyond the risk vs. benefit considerations that were the primary focus of this report. There are environmental impacts, unquantifiable potential costs, liability and political ramifications that must be addressed. Further, the issue of cost-effectiveness must be more fully explored before an intelligent decision can be rendered. To varying degrees, all of the matters addressed in this section of the report would tend to argue against fluoridation in Natick. That is to say that all of these factors, quite aside from the main issue of the benefits of fluoride, do not cast the matter in a positive light.

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Question 2 The Establishment of an Appropriate Dosage

2. If your answer to question 1 is "No" -- i.e. you believe Natick's water should be fluoridated -- do you believe that steps should be taken to establish the appropriate dosage before such fluoridation begins?

Discussion

Conclusion and Committee Response to Question 2

In view of the Findings, Conclusion and Recommendations given on p. 3, this is not an easy question to answer. One of the most troublesome aspects of this entire investigation involves the determination of total fluoride intake within the community. Moreover, both the sources and the quantities of ingested fluoride may be all but impossible to determine except in the simplest cases. In fact, it is precisely this inability to identify and quantify the uncontrollable sources of fluoride, particularly in the young, that argue against adding a so-called "controlled" dose of fluoride into any public water system.

It has been suggested that the increased incidence of dental fluorosis is, to a large degree, the result of this "uncontrollable" fluoride ingestion. Nevertheless, Appendix C has been included to provide some insight and guidelines with regard to recommended maximum fluoride intakes for various age groups.

Question 3 The Advisability of a Dental Survey

3. *If your answer to question 2 is "Yes", do you believe that an outside organization should be engaged to examine Natick school children and determine their DMFS (decayed, missing and filled surfaces) levels as an aid to selecting an optimum fluoridation level?*

Discussion

Conclusion and/or Committee Response to Question 3

From a pragmatic view, the debate on fluoridation in Natick may continue regardless of either the conclusions reached in this report or the actions taken by the Board of Selectmen. If Natick decides to go ahead with fluoridation, then those who are opposed will probably continue to lobby for the cessation thereof and if Natick decides not to fluoridate then the profluoridationists will probably continue to lobby for fluoridation.

Nevertheless, it is patently clear that all of the past and current debate has taken place without any hard data about the incidence of dental caries in Natick. It seems equally clear that unless we know whether there is any real need for fluoridation in Natick the question of benefit vs. risk, whether perceived or real, can never be answered. For this reason we recommend that a study of the incidence of dental caries and the signs of fluorosis in the youth of Natick be undertaken regardless of whether the water is fluoridated or not. Without this baseline we will never know the possible benefits to fluoridation in Natick.

Question 4 Source of the Survey and Probable Costs

4. *If your answer to question 3 is "Yes', what organizations (identify at least two) are qualified to conduct such a survey, and what are preliminary estimates of the costs involved?*

Discussion

Conclusion and Committee Response to Question 4

The Committee consensus is that this question cannot be easily answered without additional further study. Some have suggested that such a study could take as long as the time taken to produce the current document. The Committee recommends that if such a study is undertaken, a neutral organization should be selected and appropriate oversight provisions should be made.

Appendix A Source Materials

During the planning stage of this report, the committee had considered including copies of all of the supporting documents received. During the ensuing weeks, it has become clear that such an approach would merely ensure that this report would be less useful to the Selectmen as well as the community at large. Therefore, this section is comprised of a list of those materials provided by each requested party. A separate binder(s) containing all of the materials provided or referenced will also be prepared and this will be provided to the Morse Library, as will a bound copy of this report.

As can be seen from the following lists, proponents and opponents of the fluoridation issue have provided (often substantially) more than had been requested by the committee. These materials are listed below. [Note: All of the concerned parties also submitted additional materials later in the study which are not listed below.] Since the committee, in addition, reserved the right to search the literature on their own, no attempt was made to limit the selections provided. However, "propaganda" pieces such as those characterized by hysteria and/or polemics, and in particular, those documents which contained no referential material generally were less favorably received by the committee than were peer-reviewed and/or clearly unpartisan attempts to summarize the field. The numbers assigned to any of the provided materials listed below should not be construed to indicate correspondence with the references noted in any of the sections of this report. They are merely used to enumerate the documents provided.

Materials Provided by the Board of Health

1. Wade, R., Cover memo to Fluoride Study Committee
2. Ripa, L., *A Half-century of Community Water Fluoridation....Review and Commentary*, J. Pub. Hlth. Dent., **53**; 17 (1993)
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5. Harte, D. B., *Letter from the Mass. Dental Society to Board of Selectmen*, Feb. 10, 1997
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13. American Dental Association, *Fiftieth Anniversary of Water Fluoridation*, Press Release, Apr. 1995

14. Todd, J. S., *Letter from the A. M. A. to T. J. Ginley*, Apr. 30, 1990
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17. Center for Desease Control and Prevention, *Fluoride: The Benefits Can Last a Lifetime*, Press Release (?), (undated)
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19. Taddeo, A. C., et al. *Letter from the Natick Board of Health to E. Dlott*, Mar. 21 1996
20. Breda, D. J., et al., *Letter from the Natick Board of Health to J. Moran*, Feb. 10, 1997
21. Natick Board of Health, Press Release, Mar. 11, 1997
22. Natick Board of Health, Press Release, Apr. 29, 1997
23. Delli Colli, P. A., *Letter from the Natick Board of Health to the Natick Bulletin*, Mar. 18, 1997
24. Various other materials presented at a later date. Though not enumerated here, this material will be included in the reference package to be provided to the Morse Library.

Materials Provided by Shirley Brown

1. Hileman, B, *Flouridation of Water*, Chem. & Eng. News, **66**:26, (1988) (several copies)
2. Agency for Toxic Substances and Disease Registry U.S. Public Helath Service, *Toxicological Profile for Fluorides, Hydrogen Fluoride, and Fluorine*, TP-91/17, (l993) (selected pages)
3. Glass, G, *Water Fluoridation: The Enhanced Toxicity Factor?*, The Australian Fluoridation News, 31:6, (1995)
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23. Health Alliance International, *Water Fluoridation* (press release) May 5, 1997
24. Various other materials presented at a later date. Though not enumerated here, this material will be included in the reference package to be provided to the Morse Library.

Materials Provided by Myron Coplan

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2. Coplan, M. J., *The Massachusetts Lead/Fluoridation Connection in 1992*, (unpubl.)
3. Coplan, M. J., *Fifty Years of Tooth Decay and Fluoridation*, (unpubl.) (1997)
4. Report of the Ad Hoc Committee, *Review of Fluoride Benefits and Risks*, USPHS, (1991). (Only 89 pages of this document were provided.)
5. DePaola, P., et al., *Changes in Caries Prevalence of Massachusetts Children over Thiry Years*, (paper presented at 1981 IADR)
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10. Various other materials presented at a later date. Though not enumerated here, this material will be included in the reference package to be provided to the Morse Library.

Materials Provided by NFSC Members

These materials are listed throughout this report. A collection of these references is being prepared and will be provided to the Morse Library.

Appendix B The Difficulty of Research on Fluoridation

This committee encountered many problems in conducting a literature search on the subject of fluoridation. The topic has become a very emotional one and proponents on both sides of the issue (profluoridationists and antifluoridationists) are guilty of not giving full credit to research from their opponents. However, since the 'power' and money lies mostly within the profluoridation side of the issue, (the larger, powerful organizations such as the AMA and ADA historically have been in favor of fluoridation) the antifluoridationists have been given 'short shrift' due their inability to get approval and funding for studies which lead to, or may lead to, conclusions that are in opposition to fluoridation. In fact, some profluoridation reviews do not even record or address these opposing views. It is therefore very important that when one looks into the literature regarding fluoridation, one keep in mind the biases that exist on this issue. This matter has been addressed in Hileman's review of fluoridation (which can fairly be described as non-partisan). We have included an excerpt from this review to give the reader some insight into the problems of research on the fluoridation issue. (1)

"Ever since the Public Health Service (PHS) endorsed fluoridation in 1950, detractors have charged that PHS and the medical and dental establishment, such as the American Medical Association (AMA) and the American Dental Association (ADA), have suppressed adverse scientific information about its effects.

Some of those who generally support fluoridation make similar charges. For example, Zev Remba, the Washington Bureau editor of AGD Impact, the monthly publication of the Academy of General Dentistry, wrote last year that supporters of fluoridation have had an"unwillingness to release any information that would cast fluorides in a negative light," ***and that organized dentistry has lost*** **"its objectivity - the ability to consider varying viewpoints together with scientific data to reach a sensible conclusion."**

The dozen or so scientists C&EN was able to contact who have done research suggesting negative effects from fluoridation agree on this aspect. They all say that fluoridation research is unusual in this respect.

If the lifeblood of science is open debate of evidence, scientific journals are the veins and arteries of the body scientific. Yet journal editors often have refused for political reasons to publish information that raises questions about fluoridation. A letter from Bernard P. Tillis, editor of the New York State Dental Journal, written in February 1984 to Geoffrey E. Smith, a dental surgeon from Melbourne, Australia, says: **"Your paper ... was read here with interest," but it is not appropriate for publication at this time because "the opposition to fluoridation has become virulent again."** *The paper poses the question: Are people ingesting increasing amounts of fluoride and can they do so with impunity?*

Sohan L. Manocha, now a lawyer, and Harold Warner, professor emeritus of biomedical engineering at Emory University medical school in Atlanta, received a similar letter in 1974 from the editor of AMA's Archives of Environmental Health. The editor rejected a report Manocha and Warner submitted on enzyme changes in monkeys who were drinking fluoridated water because of reviewers' comments such as: **"I would recommend that this paper not be**

accepted for publication at this time" because "this is a sensitive subject and any publication in this area is subject to interpretation by anti fluoridation groups."

These papers were subsequently published in prestigious British journals, Science Progress (Oxford) and Histochemical Journal. Many other authors have reported similar difficulties publishing original data that suggest adverse effects of fluoridated water. Most authoritative scientific overviews of fluoridation have omitted negative information about it, even when the oversight is pointed out. Phillips Grandjean, professor of environmental medicine at Odense University in Denmark, wrote to the Environmental Protection Agency in June 1985 about a World Health Organization study on fluorine and fluorides: **"Information which could cast any doubt on the advantage of fluoride supplements was left out by the Task Group. Unless I had been present myself, I would have found it hard to believe."**

In his 1973 Ph. D. thesis on the fluoridation controversy, Edward Groth, III, a Stanford biology graduate student at that time, concluded that the vast majority of reviews of the literature were designed to promote fluoridation, not to examine evidence objectively. Groth also noted a number of anti fluoridation reviews that were equally biased.

According to Robert J. Carton, an environmental scientist at EPA, the scientific assessment of fluoride's health risks written by the agency in 1985 **"omits 90% of the literature on mutagenicity, most of which suggests fluoride is a mutagen."**

Several scientists in the U.S. and other countries who have done research or written reports questioning the benefits of fluoridation or suggesting possible health risks were discouraged by their employers from publishing their findings. After their paper had been rejected by the editor of Archives of Environmental Health, Manocha and Warner were told by the director of their department not to try to publish their findings in any other U.S. journal. NIDR had warned the director that the research results would harm the cause of fluoridation. Eventually, Manocha and Warner were granted permission to publish their work in a foreign journal.

In 1982, John A. Colquhoun, former principal dental officer in the Department of Health in Auckland, New Zealand, was told after writing a report that showed no benefit from fluoridation in New Zealand that the department refused him permission to publish it.

In 1980, Brian Dementi, then toxicologist at the Virginia Department of Health, wrote a comprehensive report on "Fluoride and Drinking Water" that suggested possible health risks from fluoridation. This 36-page study has been purged from the department's library even though it is the only one the department has prepared on the subject. According to current employees, no copy exists anywhere in the department. Spokesmen say the report was thrown away because it was old but also say the department will be preparing another report on the subject soon.

An ADA white paper written in 1979 states: **"Dentists' nonparticipation [in fluoridation promotion] is overt neglect of professional responsibility."** *An ADA spokesperson says this is still the association's official policy. In recent years, several dentists who have testified on the anti fluoridation side have been reprimanded by their state dental officers.*

ADA and PHS also have actively discouraged research into the health risks of fluoridation by attacking the work or the character of the investigators. As part of their political campaign, they have over the years collected information on perceived anti fluoridation scientists,

leaders, and organizations. Newspaper articles about them are stored in files, as are letters about them from various proponents of fluoridation. Little or no effort has been made to verify the accuracy of this information. It is used not only in efforts to counteract arguments of the antifluoridationists, but also to discredit the work and objectivity of U.S. scientists whose research suggests possible health risks from fluoridation.

One example is the false information about the late George L. Waldbott, founder and chief of allergy clinics in four Detroit hospitals, that ADA disseminated widely to discredit the validity of his research. Rather than deal scientifically with his work, ADA mounted a campaign of criticism based largely on a letter from a West German health officer, Heinrich Horning. The letter made a number of untrue statements, including an allegation that Waldbott obtained his information on patients' reactions to fluoride solely from the use of questionnaires. ADA published Hornung's letter in its journal in 1956 and distributed a news release based on the letter. ADA later published Waldbott's response to this letter. But the widely disseminated original news release was not altered or corrected, and continued to be published in many places. As late as 1985, it was still being quoted. Once political attacks effectively portrayed him as "anti fluoridation," Waldbott's work was largely ignored by physicians and scientists.

In November 1962 and 1965, ADA included in its journal long directories of information about anti-fluoridation scientists, organizations, leaders, and others known to be opposed to fluoridation. Listed in alphabetical order were reputable scientists, convicted felons, food faddists, scientific organizations, and the Ku Klux Klan. Information was given about each, including quotes from newspaper articles, some of which contained false data. The information was published for use by proponents of fluoridation in local fluoridation referenda.

John S. Small, information specialist at the National Institute of Dental Research, is quite willing to talk about the files he keeps on anti fluoridation organizations and their leaders. **"Of course, we gather information,"** *he says.* **"These people are running all over the country opposing fluoridation. We have to know what they are up to."** *Consumer advocate Ralph Nader has a different view of this activity. He calls it an* **"institutionalized witchhunt."**

It is easy to understand why research on risks of fluoridation has never been more vigorously pursued. Most of the individuals and agencies involved have been promoting fluoridation publicly for nearly 40 years. Research that suggests possible harm threatens them with a loss of face. For example, PHS has historically been the principal source of funds for fluoride research; but ever since June 1950, PHS has been officially committed to and responsible for promoting fluoridation. Thus, the agency has a fundamental conflict of interest.

Colquhoun, now teaching the history of education at the University of Auckland, offers another explanation for what appears to be the suppression of research. He notes that the editorial policy of scientific journals has **"generally been to not publish material which overtly opposes the fluoridation paradigm."** *Scientific journals employ a referee system of peer review. But when the overwhelming majority of experts in an area from which the referees are selected are committed to the shared paradigm of fluoridation, Colquhoun notes, the system lends itself to preservation and continuation of the traditional belief that fluoridation is safe and effective. This results in* **"single-minded promotion, but poor quality research, and an apparent inability to flexibly reassess in the presence of unexpected new data,"** *he says....."* (1)

Reference

(1) Hileman, B, *Fluoridation of Water*, Chem. & Eng. News, 66:26, (1988)

Appendix C Recommended Dosage of Fluoride

The following table provides the currently recommended fluoride supplementation for children living in a community with fluoride level in the ranges shown. (1) These doses were revised downward in 1995.

Table I
Recommended Fluoride Supplementation (mg/day) for Children

Range of Child's Age			Fluoride Concentration of Drinking Water (ppm)		
Child Age		PPM	<0.3	0.3-0.6	0.6
From	To	Units	mg/day	mg/day	mg/day
0	6	months	0	0	0
6	36	months	0.25	0	0
3	6	years	0.5	0.25	0
6	1	years	1.0	0.5	0

For example, from the shaded box above, one can determine that the fluoride supplement suggested above for a 6-36 month-old child living in a community with public water fluoride concentration below 0.3 ppm, amounts to 0.25 mg per day.

Table II below illustrates the estimated tap water intake in milliliters per day (ml/d) for each of the age groups shown related to the fluoride intake (mg/d) for 1 ppm fluoride and for 0.7 ppm fluoride in the tap water.

Table II Estimated Tap Water and Fluoride Intake for Children

Daily F & H_2O Intake			Water Consumption		F Intake (1.0 ppm)		F Intake (0.7 ppm)	
Child Age			Mean*	90 pct*	Mean*	90 pct*	Mean*	90 pct*
From	To	Units	ml/day	ml/day	mg/day	mg/day	mg/day	mg/day
0	6	mos	?	?	?	?	?	?
6	36	mos	470	890	0.47	0.89	0.33	0.62
3	6	yrs	550	930	0.55	0.93	0.38	0.65
6	16	yrs	700	1160	0.70	1.16	0.49	0.81

- The "Mean" value is equivalent to the arithmetic average value. "90 pct" refers to the 90th percentile. In the above example, 10 per cent of the children in the 6-36 month-old group will ingest more than 0.89 mg of Fluoride per day. The hypothetical average child in this age range will ingest about 0.47 mg/day.

The shaded boxes in Table II under Water Consumption indicate **that the same 6-36 month old child** who drinks 470 ml/d or the upper 90th percentile child who drinks 890 ml/d will have received 0.47 and 0.89 mg/d of fluoride if the water is fluoridated at the level proposed for Natick. Clearly such a child **will have ingested between about 2-3.5 times as much fluoride as recommended by the American Academy of Pediatrics, American Dental Association and the American Academy of Family Physicians.**

Reference

(1) American Academy of Pediatrics, Committee on Nutrition, Pediatrics, 95:777, (1995); Also endorsed by the American Dental Association and the American Academy of Family Physicians.

Appendix D The Measurement of Fluoridation Parameters

The following material is provided so that persons unfamiliar with the literature of the fluoridation issue can gain some insight into the methods used by workers in the field to express dental caries prevalence and reductions thereof. (1,2) The process used and the specification of the results obtained are described below. The second section of this appendix describes the method used to arrive at a measure when comparing the prevalence of dental fluorosis.

The Dental Caries Reduction Measurement

Measurements are taken by trained examiners using specified lighting and dental instruments to examine the teeth of subjects for evidence of:

- untreated decay ("D" or "d"),
- tooth lost due to decay ("M" or "m"),
- filling in place ("F" or "f").

Capital letters are used when the tooth is permanent and lower case is used when the tooth is deciduous. Most scores do not mix the two types of teeth in a common number. However, sometimes "d" appears without "m" because it is not always possible to know after the fact if a missing baby tooth was carious when it was lost. Initially, the scoring system used to relate an individual score was determined by the number of decayed, missing and filled teeth found, so "DMFT" (or "dmft") was the term used. Later on, counting the number of tooth surfaces implicated in a carious tooth became more popular, so "DMFS" (or "dmfs") appears more often in the literature after 1975.

For epidemiologically statistical purposes, the scores of all the individuals in a group are added and the sum is divided by the number of individuals to give a group score. Note however, that this says nothing about the distribution of poor teeth within the group as a whole. In addition, it has been widely recognized that fluoridation is responsible for the phenomenon of delayed dentition. Szelag has evaluated the "dental age" of a number of children and has reported that not only is there evidence of delayed dentition but that the greater the dosage of fluoride taken up during development, the greater was the retardation of dentition. (3) Opponents of fluoridation have pointed out that when due consideration is given to this fact, the reported DMFT and DMFS scores fail to reflect a significant difference between fluoridated and unfluoridated areas. For example, Coplan states:

> *"....When the basic scoring system was adopted, a small filling or decayed tooth area added one unit to a DMFT score with the same weight as a tooth seriously enough involved to have required extraction or major repair. Thus, a*

badly decayed extracted molar added no more to a score than a small filling in a cuspid. Since this lack of discrimination could mask real effects, the practice was adopted of weighting decay severity by counting implicated surfaces, not just implicated teeth.

A molar extracted due to decay adds five units to a score, a small filling adds one unit, a two-surface filling on a cuspid adds two units, etc. This may leave some room for judgment calls but it definitely makes DMFS scores numerically larger than DMFT scores for the same actual caries status and has the numerical consequence that DMFS = 1.6 x DMFT as a general rule." (2)

As the incidence of caries has declined, however, and DMFS scores have fallen into the low single-digit region, the interpreter of group DMFS values should be cautioned to be wary of the potential for subtle distortions. For example, a 10% difference in group DMFS scores in the region of approximately 3.0 can mean any number of things, not the least of which may be due to slight variations in examining protocol. Possible sources of such differences may actually be due to variations in treatment schemes from time to time, dentist to dentist or location to location. (2)

As an example, decisions to extract a decayed first molar because it seems to be interfering with the eruption of other teeth, but which might have been saved by filling, or to decide whether a particular tooth gets a 3-surface crown or some less complicated repair will obviously have some impact on these scores. Waldbott (1) has commented on this variability as follows:

"...Another crucial factor to be considered in evaluating caries statistics is the variability and possible bias of the examiner. One investigation demonstrated, for example, that repeated examinations of the same tooth by the same examiner yielded widely varying caries scores from one examination to another.(58) In a different study, when each of the 33 patients was examined by three of eight different dentists, a deviation of 89% in the number of cavities was recorded.(59) In one case two of the dentists found 12 cavities. while the third found only five. In another case one dentist found 13 cavities, the second found six, and the third found only five. Overall, the average difference in assessment for the 33 patients was 4.2 carious teeth and 5.8 carious surfaces. With such large and glaring discrepancies, it is obvious that any conclusions based on differences of only two or three DMF teeth, as is often the case in fluoridation studies, has only marginal value at best. Realistically speaking, such conclusions are highly questionable...."

Socioeconomic factors may also play a decisive (albeit unapparent) role, especially in comparisons of areas wherein these factors may vary greatly, obviating otherwise identical factors. In such an area, how much does DMFS depend on diet, willingness to seek medical help, ability to pay, delay in getting proper, early and regular

professional attention? When one considers that close to half of the student population in current studies is caries-free, one is forced to recognize that "*...the poor dentition resides in a smaller segment of the population, probably starts very early and benefits little if at all from fluoridated water.*" (2)

Nevertheless, with DMFS values as low as 2.3, what is the significance of a 10 % reduction in practical terms? In a school system with DMFS value of 2.8 averaged across 5-17 year-olds, the statistically average child who comes into the school system with no cavities will leave with a DMFS of 7.01 (fluoridated public water supply) vs. 8.59 (unfluoridated public water supply), a difference of only 1.5 treated surfaces over the 12-year school experience. (4) An 18% reduction in DMFS as far as the student is concerned is less than one cavity and therefore can hardly be called significant.

The Quantification of Dental Fluorosis

This measurement, also termed the Dental Fluorosis Index, was postulated and defined as a means to respond to the problem of determining the optimal fluoride concentration in a public water supply that would produce the soundest teeth without the disfigurement of mottling. The parameters for this metric are illustrated in Table I.

Table I Degrees of Dental Fluorosis (5,6)

Category	Description of Aberration	Weighting Factor
None (Normal)	Enamel smooth, glossy, pale creamy white translucency	0.0
Questionable	Slight aberrations from translucency with occasional white fleck or spots	0.5
Very Mild	Small, opaque, paper-white areas involving less than 25% of the surfaces of the two most affected teeth; may acquire brown stains in adulthood	1.0
Mild	More extensive dull white opacities involving less that 50% of the surfaces of the two most affected teeth; Brown staining often present	2.0
Moderate	All enamel surfaces affected; distinct brown staining frequent	3.0
Severe	Teeth show marked hypoplasia, attrition and pitting; brown or black staining widespread	4.0

According to Dean, the preferable community index should not exceed 0.4 and at 0.6 *"...it begins to constitute a public health problem."* Waldbott comments (5) on both the weighting factors used and the justification thereof by stating:

> *".....Although in theory such calculations are attractive, in reality they are misleading. The community index of dental fluorosis does not accurately represent the true state of mottling in a community. It gives the same weight to eight questionable (0.5) cases as to one severe case (4.0); it counts three mild (2.0) or six very mild (1.0) cases as equal to two moderate (3.0) ones ..... For the individual with an unsightly degree of mottling, it is of no comfort to know that the community index of dental fluorosis is below 0.6 or even below 0.4! This dilemma was clearly perceived by Cox, who first explicitly advocated fluoridation, when he wrote: `...***With the threat of the Scylla and Charybdis of dental caries and mottled enamel, great caution must be observed in the means of administration of fluorides and in the control of such procedures as may be adopted'...***"

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Members of the Committee

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*The committee wishes to acknowledge the participation of Dr. Benedict J. Gallo and to recognize his contributions during the course of this study. Although Dr. Gallo resigned from the committee during the final preparation of this report, he remained in complete agreement with the Findings, Conclusion and Recommendations of the Natick Fluoridation Study Committee.

Curricula Vitae of the Committee Members

The following material is being provided at the specific request of the Board of Selectmen of the town.

Curriculum Vitae of Benedict J. Gallo

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EDUCATION

Ph.D., University of Michigan, Ann Arbor, Michigan, 1977.
Major, Botany; Minor, Chemistry.
M.S., Eastern Michigan University, Ypsilanti, Michigan, 1970.
Major, Biology; Minor, Education.
M.S., University of Michigan, Ann Arbor, Michigan, 1962.
Major, Geology; Minor, Zoology.
B.A., University of Connecticut, Storrs, Connecticut, 1958.
Major, Geology; Minor, Zoology.

EMPLOYMENT

1979 – Retirement 1996

Research Microbiologist at the U.S. Army Research, Development and Engineering Center, Natick,, Massachusetts 01760-5020. Conducted basic research involving the search and development of inexpensive sources of microbial enzymes for use in Chemical Defense and munitions biodegradation.

1976 – 1979

Research Microbiologist, National Research Council Research Associate at the U.S. Army Research, Development and Engineering Center, Natick, Massachusetts 01760- 5020. Conducted basic research in the bioconversion of ligno-cellulosic urban wastes and agricultural residues into the power fuel ethanol.

1970 – 1976

Teaching Fellow and Research Associate, University of Michigan, Ann Arbor, Michigan. I instructed degree candidates in Botany, Genetics and Microbiology and conducted independent research involving the genetics, biochemistry and microbiology of several microbial enzyme systems.

1960 – 1970

Instructor, Biology, Central Connecticut University, New Britain, CT; Secondary School Science Teacher at St. John School, Jackson, Michigan and Bentley Senior High School, Livonia Public School System, Livonia, Michigan, Pharmaceutical Sales, Westerfield Labs, Cincinnati, OH

PUBLICATIONS

Journal papers, DOD reports and U.S. Patents.

AWARDS

1989 USANRDEC Silver Pin for Research,
1989 Soldier Sciences Directorate Outstanding Project Officer Award.

Curriculum Vitae of Jason Kupperschmidt

Jason Kupperschmidt
17 Greenwood Road
Natick, MA 01760

Education
University of Illinois, Urbana-Champaign, IL
B. S., Chemical Engineering, May 1992
Northeastern University, Boston, MA
M. S. Student, Environmental Engineering, September 1995 to Present

Professional Affiliations
Commonwealth of Massachusetts Engineer-in-Training, Certificate # 16409
American Institute of Chemical Engineers, Member

Experience
Armstrong Pharmaceuticals, Boston, MA
September 1994- Present
Process Engineer

- Enhance existing manufacturing processes through optimization of critical steps.
- Interact with the other department supervisors to ensure production under GMP guidelines.
- Oversee personnel training, preventive maintenance, and calibration programs.
- Implement process changes in chemical aspects of production.
- Maintain all support equipment including: refrigeration, pumps, and tanks.

General Chemical Corporation Framingham, MA
November 1993 - January 1995
Environmental Chemist

- Analyzed hazardous waste primarily for chlorinated solvents and PCB contamination.
- Evaluated whether chlorinated solvent waste can be reclaimed.

U.S. Army Natick Research, Development and Engineering Center, Natick, MA
May - August 1990, May - August 1991, July 1992 - February 1994
Biochemistry Research Assistant

- Conducted and analyzed biochemical experiments for food research applications.
- Designed diagnostic thermal processing devices to optimize heat exposure.
- Utilized analytical instrumentation and computer software in data analysis.

Dupont/Merck Pharmaceutical Company, Billerica, MA
March - August 1994
Contract Assignment

- Production Engineer Technician
- Regulated and disposed of radioactive waste.
- Generated the radiopharmaceuticals used as imaging agents.

Curriculum Vitae of Norman R. Mancuso

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Dr. Norman R. Mancuso possesses a broad and diversified background in Engineering and the Sciences. Trained and educated at such institutions as St. Bonaventure University, State University of New York at Buffalo and the Massachusetts Institute of Technology, he has over thirty years of in-depth, hands-on experience in a wide range of Chemical, Engineering and Computer related projects. He is the author or co-author of over one hundred Chemical, Scientific and Engineering publications and has extensively served both the domestic and European high-tech communities.

Educational Background

Postdoctoral Fellow
Massachusetts Institute of Technology
Ph.D., Chemistry, SUNY at Buffalo, Buffalo, NY.
M.S., Chemistry, St. Bonaventure University, Olean, NY.
B.S., Chemistry/Mathematics, St. Bonaventure Univ. Olean, NY.

Academic and Industrial Honors

Dupont Teaching Fellow
National Institutes of Health Postdoctoral Fellow
Product Innovation Award - Dennison Mfg. Co., Inc.

Academic and Industrial Positions

NORMAN R. MANCUSO ASSOCIATES, Framingham, MA, Consulting Engineer
AVERY DENNISON, INC., Imaging Systems Division, Hopkinton, MA, Group Leader
DENNISON MFG. CO., INC., Corporate R&D, Framingham, MA, Senior Engineer
INFOREX, INC., Advanced Development Group, Burlington, MA, Senior Engineer
MASSACHUSETTS INSTITUTE OF TECHNOLOGY, Dept. of Chem., Cambridge, MA

- Postdoctoral Fellow and Research Staff Member
- Director of the Computer Facility at the NIH Mass Spectrometry Center

NORTHEASTERN UNIVERSITY, Boston, MA, Lecturer/SOA Engineering Program

Dr. Mancuso pioneered the use of high-integration embedded microprocessors, high density programmable logic and field programmable gate arrays at Dennison and introduced various CAE tools into the Dennison R & D environment.. He also planned, implemented, and directed an Automatic Test facility for PCB testing. He received a Corporate Productivity Award for the design and development of high integration embedded microprocessor PCB's used in several product lines. He also developed various product/process specifications, including technical documentation for a number of engineering companies. A strong proponent of continuing education, he was instrumental in arranging and administering employee Professional Development courses and other programs increasing technical employee involvement, productivity and morale.

As an Apollo Program Project Scientist, Dr. Mancuso was responsible for the development of an interlock system enabling the organic analysis of lunar samples while maintaining and protecting the integrity of the terrestrial biosphere. Other analytical instruments developed include a laser-based web flaw detector for the Dunn Paper Co. as well as Comparator/Spectrophotometers and real-time data acquisition systems for the measurement of Mass Spectrometric photographic plates. While serving as a consultant to Karolinska Institute (Stockholm) he developed a real-time data acquisition systems incorporating multi-channel non-coincidence amplifier systems.

Dr. Mancuso is a member of the Metrowest Chamber of Commerce, the Institute of Electrical & Electronic Engineers, the Committee of Concerned Engineers and the American Chemical Society. He also served on the Natick Underground Storage Tank Removal Committee.

Curriculum Vitae of Alfred J. Murray

Alfred J. Murray
Framingham, MA 01702
Email murray@meol.mass.edu

Education:

- Bridgewater State College, 1960. B.S.
- Major; Mathematics and Chemistry
- Colby College, 1973. M.S.T.
- Major; Chemistry

Massachusetts Teaching Certification:

- Secondary school principal, mathematics, chemistry and general science

Work Experience:

- 1991-1997 Dean College. Instructor of Chemistry
- 1963-1997 Natick High School, Teacher and Dept. Chairman
- 1990,91,94 U.S. Army Natick Labs. Research
- 1966-1971 Framingham Union Hospital, Lab Technician
- 1962-1964 Lawrence General Hospital, Lab Technician
- 1963-1964 Longwood Hospital, Lab Technician
- 1960-1962 United States Army, Clinical Lab Technician

Publication:

Mental Deficiency, *Dwarfism and Decreased Segmentation of Neutrophilic Leucocytes*; Journal of Mental Deficiency Research, **11**(4) December 1967

Awards:

- U.S. Army Special Act Award (as a food chemist)
- Edison Citation for Distinguished Service
- Certificate of Honor, Westinghouse Science Search
- Tandy Outstanding Educator Award (1993 &1994)

Curriculum Vitae of Harlee S. Strauss

Harlee S. Strauss, Ph.D.
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Dr. Strauss is the President of H. Strauss Associates, Inc. (HSAI), a consulting firm she founded in 1988. Dr. Strauss works on a broad range of projects, from site specific human health risk assessments, to in-depth evaluations of the toxicity of individual chemicals, to the development of risk assessment methodology. She has conducted projects related to identifying gender biases in risk assessment, how to apply risk assessment methodology to childhood cancer, and how to establish risk assessment frameworks with respect to microorganisms, including bioremediation. Dr. Strauss initiated and, for its first year, lead a multi-million dollar study to investigate the potential links between the environment and breast cancer on Cape Cod, Massachusetts.

Dr. Strauss has been a member of the Society for Risk Analysis since 1987. She served on the Management Committee for the residential exposure assessment project and on the Advisory Committee for SRA Workshop *"Key Issues in Carcinogen Risk Assessment Guidelines."* Dr. Strauss is a long time member and former president of the New England Chapter of SRA. She initiated the SRA-NE monthly newsletter, *"Back of the Envelop"* and was its editor for several years. Dr. Strauss received an Outstanding Service to Society award from the SRA in 1996.

Dr. Strauss's other activities include serving her second, two-year term on the Army Science Board (ASB). She was a member of the ASB work group on Management and Abatement of Lead Based Paint at Army Sites and is currently a member of two ASB study panels: 1) Evaluation of the Effectiveness of Existing Groundwater and Soil Treatment Systems in the US Army and 2) a study related to Chemical/biological Weapons Defense.

Harlee Strauss earned a Ph.D. in molecular biology from the University of Wisconsin-Madison (1979) and an A.B. in chemistry from Smith College (1972). She was a postdoctoral fellow in biology at MIT (1979-81), sponsored by the NIEHS) and a Congressional Science Fellow sponsored by the Biophysical Society (1981-83). Dr. Strauss has also held the positions of special assistant for government affairs at the American Chemical Society (1983-84), special consultant at ENVIRON Corporation (1984), research associate at the MIT Center for Technology, Policy and Industrial

Development (1985-86), senior associate at Gradient Corporation (1986-88), and executive director of Silent Spring Institute (1994-95).

Errata

Page	Correction
8	"....**Dosage** of Fluoride..."
11	"....**Selectmen** with regard...."
12	"....normal **usage**, direct..."
	"....this **usage**....."
12, 16	"....**profluoridation**..."
14, 15	"....**antifluoridationists**.."
15, 16	"....**profluoridationists**..."
	"....**a.k.a.**...."
16	"....**desirability**..."
18	"....**pre-eruptive**..."
18	"....**e. g., lifestyle**...."
18	"....**desirable**...."
24	"....**dysfunction**...."
	"....**insecticides**...."
	"....**minimum**...."
25	"....**independent**...."
27	"....**mgs** of fluoride per **kg** of...."
	"....**nationwide**... "
29	"....**Agency's**...."
33	"....**postmenopausal**...." (2x)
36	"....**carcinogenicity**...."
43	"....**enzymes** (17)...."
48	"....**triethyltin-induced**...."
52	"....**than** 110...."
53	"....**prenatally**..."
	"....**observed**...."
	"....**findings** are...."
56	"....**epidemiological** study..."
60	"....**negligible**...."
	"....**occurred** in 1992...."
61	"....sodium **fluorosilicate** are...."
76	Definitions corrected and clarified.
82	"....M**A**"
89	"....murray@meol.**mass.**edu"
	"....**Army**...."

Report of the Fairbanks Fluoride Task Force

April 25, 2011

Prepared for the Fairbanks City Council
Fairbanks, Alaska

Contents

Chapter 1

Introduction

In response to concerns expressed by community members, on February 8, 2010, the Fairbanks City Council passed a resolution (Appendix A) establishing a committee charged with the responsibility to examine evidence related to fluoridation of public water supplies and to provide the City Council with a report containing analysis and recommendations. The committee was to obtain documentation provided by both proponents and opponents of fluoridation and to supplement this documentation with information from other appropriate sources. The committee was to make its final report to the City Council by early July, 2010, but the committee was unable to meet this deadline due to the complexity of the assignment and the schedules of the committee members.

The committee, referred to in this report as the Fairbanks Fluoride Task Force (FFTF), is composed of the following members:

Paul Reichardt, Ph.D. (Chair)
Professor of Chemistry Emeritus
University of Alaska Fairbanks

Richard Stolzberg, Ph.D.
Professor of Chemistry Emeritus
University of Alaska Fairbanks

Rainer Newberry, Ph.D.
Professor of Geochemistry
University of Alaska Fairbanks

Bryce Taylor, D.D.S.
Dentist
Fairbanks

Joan Braddock, Ph.D.
Professor of Microbiology Emeritus
University of Alaska Fairbanks

Beth Medford, M.D.
Tanana Valley Clinic
Fairbanks

The FFTF met for the first time on March 4, 2010, and continued to hold public meetings approximately twice a month through March 8, 2011. At the invitation of the FFTF, both proponents and opponents of fluoridation of the Fairbanks water system (operated by Golden Heart Utilities) made presentations at the March 16, 2010, meeting. Public testimony was received at each of the ten public meetings during the period March 16, 2010, through June 22, 2010. Numerous comments and pieces of information were submitted to the FFTF electronically. Members of the FFTF supplemented this information with relevant articles from the professional literature and results of personal interviews and research.

All documents and information received by the FFTF during the period in which public testimony was being accepted are cited on the References section of the FFTF website (www.ci.fairbanks.ak.us/boardscommissions/fluoridetaskforce/fluoridetaskforcereferencematerials.php). While FFTF members considered the entire body of information submitted and collected, only some of the materials listed on the References website were used in preparing this report. Those materials are listed as references at the end of this report. There is a massive amount of relevant information on this topic. For example,

in 2008, C. A. Yeung did a review of the efficacy and safety of fluoridation that began with over 5,000 relevant citations. The approach the FFTF took to assessing and using this information was to rely on reviews and studies published between 2000 and 2008 to assess the evidence for and against fluoridation of drinking water as it existed up to 2008 and to supplement this body of literature with key professional articles published in the last several years.

Although the FFTF examined all aspects of water fluoridation, it focused most of its review of the literature on exposure of individuals to fluoride, the efficacy of fluoridated water in caries prevention, and the risks associated with consumption of fluoride. While the task force's major concerns were about populations exposed to 0.7 to 1.2 parts per million (ppm) fluoride in their water supplies, it did examine and consider evidence related to populations receiving both higher and lower concentrations of fluoride in their drinking water. The FFTF's review and analysis of relevant information was organized around the topics that became the chapters of this report. After a series of discussions and work sessions in which all members voiced their observations and concerns about each of the topics, assignments were made to individual task force members for lead responsibility in producing an initial draft of each chapter. The entire task force was subsequently engaged in the process of chapter revision that led to a draft report, which went out for public review and comment. After consideration of comments submitted electronically as well as at two public hearings (March 29 and 31, 2011), the task force made corrections and edits at its meeting on April 5, 2011. The subsequent final report (including recommendations) will be submitted to the City Council.

Some technical terms and abbreviations are used throughout this report. In an attempt to make the report more readable for the general public, a few key definitions are given below:

concentration: the relative content of a component, often expressed as amount in a given volume (e.g., ppm)
DMFS: decayed, missing, and filled surfaces in permanent teeth
DMFT: decayed, missing, and filled permanent teeth
dmft: decayed, missing, and filled deciduous (baby) teeth
dose: measured quantity of an agent to be taken at one time
g (gram): 0.001 kg
kg (kilogram): a basic unit of mass and weight equal to 2.2 pounds
mg (milligram): 0.001 g
L (liter): a basic unit of volume equal to about a quart
LD_{50} (lethal dose, 50%): dose of a toxin required to kill 50% of a group of test organisms
ppm (parts per million): a unit of concentration, defined for this report as one mg/L

Chapter 2

Recommendations

The Fairbanks Fluoride Task Force makes a set of four recommendations. We anticipate that the community's focus will be on Recommendation #1, but as a committee we feel strongly that Recommendations 2, 3, and 4 should be implemented along with Recommendation #1 as part of a cohesive plan to address dental health issues in our community.

1. Primarily because (1) the ground water used for Fairbanks public water contains an average of 0.3 ppm fluoride, and (2) higher concentrations of fluoride put non-nursing infants at risk, the task force recommends that supplemental fluoridation of the Fairbanks public water supply be terminated. The task force further recommends that the Fairbanks community be informed of possible dental health implications from not fluoridating the water.
   Rationale: Not fluoridating Fairbanks water will reduce the fluoride content from 0.7 ppm to 0.3 ppm, which is the fluoride concentration of the raw water used by Golden Heart Utilities (GHU). This will reduce, but not eliminate, the risk of significant incidence and severity of fluorosis, especially fluorosis associated with the use of GHU water to prepare infant formula. Doing so will also address ethical concerns raised during the task force's public testimony. However, the effect of this reduction in fluoride concentration on the caries rate in the Fairbanks community, while most likely small, is unknown and unpredictable. Those who depend on 0.7 ppm fluoride in tap water for their dental health need to be informed of the possible adverse consequences to their dental health caused by reducing the fluoride content of Fairbanks tap water from 0.7 ppm to 0.3 ppm and of the measures that can be taken to address these possible adverse consequences.

   The task force has made this recommendation to terminate fluoridation of GHU water with full knowledge of and respect for the positions of the American Dental Association (ADA), the Centers for Disease Control and Prevention (CDC; part of the U.S. Department of Health and Human Services), the World Health Organization, and the Alaska Department of Public Health in support of fluoridation of public water supplies. While the task force members agree that water fluoridation may be an important element of an effective dental health program in many communities, the majority of members are not convinced that it is necessary in Fairbanks because of the fluoride content of the city's ground water and the alternate sources of fluoride available in the community. Five task force members, with various degrees of conviction, support this recommendation, while one member (Dr. Taylor) supports continuing fluoridation at 0.7 ppm.

2. The Fairbanks City Council's decision-making process on fluoridation should involve representatives of the Fairbanks North Star Borough government.
   Rationale: At least 25% of area residents who receive GHU water reside outside the city limits.

3. Local dentists and physicians should be encouraged to provide their patients with up-to-date information on the benefits and risks associated with fluoride.
   Rationale: If nothing else, the recent notice that the secretary of the U.S. Department of Health and Human Services has proposed a new recommendation on fluoridation of public water supplies

indicates that the citizenry should be informed about the state of contemporary research findings and analysis related to the role of fluoride in dental health. All of the members of the task force went into this project with incomplete and in some cases incorrect information about the issue. We suspect that we are not unique in that respect.

4. The Fairbanks City Council should encourage the local school system to review and modify, as appropriate, its approach to promoting good dental health practices.
   Rationale: The local schools have an excellent opportunity to help all families in the community to learn about and to implement good dental health practices, which can include optional opportunities at school for topical fluoride treatment (in the form of rinses and tooth brushing, for example) as well as techniques for minimizing unnecessary and/or unwanted exposure to fluoride.

Chapter 3

History of Fluoridation of Public Water Supplies

Fairbanks

A version of Fairbanks City Code dated July 1, 1959, contained a section (Article III, Section 10.301) that authorized and directed the Municipal Utilities System to develop and implement a fluoridation plan that fulfilled the requirements of the Alaska Department of Health. A slightly rewritten version of Article III, Section 10.301 of the City Code was adopted on January 12, 1960, and on August 21, 1962, the mandated fluoridation of city water was implemented in the city of Fairbanks. In 1996, the city water plant was sold by the Municipal Utilities System to Golden Heart Utilities (GHU). The fluoridation program continued under the auspices of GHU, and in 1999 the rewritten Fairbanks General Code (FGC 82-1) continued the mandate for fluoridation under the administration of Golden Heart Utilities. The present version of the Fairbanks City Code retains the language of Section 82-1 as it existed in 1999.

The only formal attempts to discontinue the fluoridation program took place in 2008. On February 25 of that year a proposed ordinance to prohibit the addition of fluoride to the GHU water supply failed in a vote of the City Council. In July 2008, a city resident submitted an application for an initiative proposing that FGC 82-1 be repealed and reenacted to read:

> Fluoride should not be added to City community water systems. Water utilities that own or operate community water distribution systems in the City shall not add fluoride, in any form, to the water system. All water utilities owning or operating community water systems in the City shall conduct periodic water quality testing.

The required signatures were not submitted by the deadline of August 12, so the initiative did not go on the October ballot. The city took no additional action on the fluoridation issue until February 8, 2010, when the City Council passed Resolution No. 4398, establishing a task force to research issues related to the fluoridation of the municipal water supply.

United States

In the early 1900s, research, largely by dentist Frederick McKay and Dr. G. V. Black of the Northwestern University Dental School, documented that many residents in several areas of the western U.S. had mottled teeth and, in severe cases, brown stains ("Colorado brown stain") on their permanent teeth. McKay also noticed that the mottled teeth were resistant to decay. By the 1930s it had been determined that these conditions (today known as fluorosis) were caused by high concentrations of fluoride (ca. 4–14 ppm) in drinking water. In the ensuing years, Dr. H. Trendley Dean conducted a series of epidemiological studies and reported that (1) fluoride concentrations of up to 1.0 ppm in drinking water did not cause the more severe forms of dental fluorosis and (2) a correlation existed between fluoride levels in drinking water and reduced incidence of dental decay

(Dean et al., 1941). Dean's work led Dr. Gerald Cox and associates to publish in 1939 the first paper in which fluoridation of public water supplies was proposed (Cox et al., 1939).

In the 1940s, four classic, community-wide studies were carried out to evaluate the addition of sodium fluoride as a caries-reduction strategy in Grand Rapids, MI; Newburgh, NY; Brantford, Ontario; and Evanston, IL. Based on the overwhelmingly positive evaluations of these pilot studies by scientists and dental professionals, water fluoridation programs were instituted in a number of large U.S. cities in the following two decades. In addition, alternative methods of administering fluoride to combat caries were developed, the most notable being the introduction of fluoridated toothpaste in 1955.

However, as water fluoridation programs spread, so did opposition to the practice. In 1965, the first lawsuit in the U.S. contesting the legality of fluoridation of public water supplies was settled by the New York State Supreme Court, which denied the plaintiff's case "at least until some proof is advanced that fluoridation has harmful side effects" (Graham and Morin, 1992, p. 215). In the ensuing years a number of lawsuits contesting fluoridation of public water supplies have been pursued, but in no case have the plaintiffs been successful in stopping the practice (see Legal/Ethical Issues, chapter 4).

The relevant federal, state, and professional organizations have endorsed and promoted the fluoridation of public water supplies for the past fifty years. As a result, in 2008, forty-six of the country's fifty largest cities provided fluoridated water, and approximately 60% of the U.S. population consumed fluoridated water (Fagin, 2008). The U.S. Public Health Service (USPHS) has set a goal of "at least 75% of the U.S. population served by community water systems should be receiving the benefits of optimally fluoridated water by the year 2010" (U.S. Department of Health and Human Services [HHS], 2000, p. 205). However, the actions of communities on this front are mixed. One summary (Juneau Fluoride Study Commission, 2006) indicates that from 1998 to 2005 approximately two hundred communities in the U.S. moved to fluoridated water or decided to retain it while approximately one hundred chose to discontinue the practice. The situation in Alaska, where the fluoridation of public water systems is encouraged by the Alaska Department of Public Health (www.hss.state.ak.us/dph/targets/ha2010/PDFs/13_Oral_Health.pdf), roughly mirrors the national picture. In 2006, 64% of the Alaska population received fluoridated water, up from 47% in 1993 (Whistler, 2007). However, today's statewide figure may be below that of 2006 because Juneau discontinued its fluoridation program in January 2007.

International

According to the British Fluoridation Society (British Fluoridation Society, 2010), over 400 million people in sixty countries were served by fluoridated public water supplies in 2004. Countries and geographic regions with extensive water fluoridation programs include the U.S., Australia, Brazil, Canada, Chile, Columbia, Ireland, Israel, Malaysia, New Zealand, Hong Kong, Singapore, Spain, and the United Kingdom. However, especially during the period of 1970 to 1993, Japan and a number of European Countries (Federal Republic of Germany, Sweden, Netherlands, Czechoslovakia, German Democratic Republic, USSR, and Finland) discontinued water fluoridation programs. In 2003, Basel, Switzerland, ended its water fluoridation program, and in 2004 Scotland rejected plans to fluoridate water supplies.

In most or all of these situations, dental health continued to improve following cessation of water fluoridation (Ziegelbecker, 1998), presumably due to factors including enhanced dental hygiene programs, fluoride-containing table salt, fluoridated toothpaste, and improved diets. There are data to support the contention that in recent years caries rates in many areas have declined irrespective of the concentrations of fluoride in water supplies. World Heath Organization (WHO) data (Peterson, 2003: Fig. 7) indicate substantial declines in DMFT among twelve-year-olds in developed countries (from about 4.7 to about 2.5) during the period 1980 to 1998 but little change among this age group in developing countries (from about 1.8 to about 2.3). Nevertheless, the World Health Organization continues to consider community water fluoridation to be an effective method to prevent dental caries in adults and children. However, it recognizes that other approaches, including fluoridated salt and milk fluoridation, have "similar effects" (www.who.int/oral_health/strategies/cont/en/index.html). It also recognizes the value of fluoridated toothpaste and fluoride-containing mouth rinses and gels.

For Alaska communities, perhaps the most relevant international situation is that in the neighboring country of Canada. According to the Health Canada website (www.hc-sc.gc.ca), each Canadian municipality retains the authority to decide on fluoridation of its water supply; in 2005, 43% of the Canadian population was served by fluoridated water supplies (Federal-Provincial-Territorial Committee on Drinking Water, 2009). The Guidelines for Canadian Drinking Water Quality set a maximum allowable fluoride concentration of 1.5 ppm in drinking water, a level at which Health Canada believes there are no undue health risks (Health Canada, 2010). Although Canadian provincial and territorial governments regulate the quality of drinking water in their jurisdictions, Health Canada has recommended to communities wishing to fluoridate their water supplies that "the optimal concentration of fluoride in drinking water to promote dental health has been determined to be 0.7 mg/L" (Health Canada, 2010).

The Controversy

From the very beginning of efforts to implement water fluoridation programs in 1945, there has been controversy (Connett et al., 2010). By the 1950s the sides were pretty well drawn. On one side were dentists and scientists from government and industry, who promoted the addition of fluoride to drinking water as a protection against dental decay. On the other side were mostly activists who contended that water fluoridation was essentially compulsory mass medication, thus a violation of individual rights, and that the risks of fluoridation had not been studied adequately. The advocates of fluoridation won the argument, in part by ridiculing the unlikely arguments of some of the opponents (e.g., the John Birch Society, which contended that fluoridation was a communist plot to poison the citizens of the USA).

A series of court cases from the mid-1960s through the mid-1980s established that local and state governments have the constitutional authority to implement fluoridation programs. These decisions were based largely on the principle that the "government interest in the health and welfare of the public generally overrides individual objections to health regulation" (American Dental Association [ADA], 2005, pp. 47–49). In light of these decisions, the argument against "compulsory mass medication" has emphasized ethical rather than legal issues (see, for example, Bryson, 2004).

During this same period, a number of scientific investigations into potential adverse effects of drinking fluoridated water were undertaken. None of these studies produced results that were generally accepted as demonstrating serious adverse health effects of water containing "optimal levels" of fluoride ion (0.7 to 1.2 ppm). However, a number of them raised significant questions about potential risks by showing some adverse health effects at fluoride concentrations of greater than 2 ppm (for example, Kurttio et al., 1999; Freni, 1994).

Around the turn of the century, a comprehensive review of the scientific literature related to water fluoridation was undertaken under the auspices of York University in the United Kingdom. The report from this review (McDonagh et al., 2000), often referred to as the York Report, noted the generally poor quality of the evidence for both beneficial and adverse effects of fluoridation. The resulting uncertainties about the benefits and risks of consuming fluoridated water fueled the controversy in that it allowed each side to discount the opposition's arguments because of the "poor quality" of the evidence on which positions were based. While there are many examples of the arguments put forward by the two sides, two representative accounts are an antifluoridation article by Colquhoun (1998) and a profluoridation article by Armfield (2007).

Another key review of the effects of fluoride in drinking water was published by the U.S. National Academy of Sciences in 2006 (National Research Council, 2006). This review and associated recommendations were focused on EPA standards for drinking water (Maximum Contaminant Level, MCL, of 4 ppm and Secondary Maximum Contaminant Level, SMCL, of 2 ppm) and did not directly address the USPHS regulations on the lower concentrations in fluoridated public water supplies in the U.S. (0.7 to 1.2 ppm). Nevertheless, the report contains information and data relevant to the safety of fluoridated water. Evidence in the scientific literature led the review committee to conclude that water containing 4 ppm fluoride "puts children at risk for developing severe enamel fluorosis" and was "not likely to be protective against bone fracture" (National Research Council, 2006, p. 2). This review also contains analyses of a number of other adverse health effects that have been alleged to be related to fluoride ingestion, but the authors found that these allegations were either not supported by good evidence or required further study before any meaningful conclusions could be drawn. As with the York Report, the uncertainties about the risks of fluoride-containing water (compounded, in this case, by uncertainties about how conclusions based on consideration of fluoride concentrations of 2 ppm or higher relate to lower concentrations) have given both advocates and opponents of fluoridation data and arguments that they have selectively employed in supporting their opposing positions.

As time has gone on, particularly since the publication of the York and National Research Council reports, a number of professionals with expertise in dental health and toxicology have joined the opposition to fluoridation. They include dental researchers who were originally supporters of fluoridation (e.g., Colquhoun, 1998; Limeback, 2000), dentists (e.g., Osmunson, 2010a), and EPA employees (e.g., Thiessen, 2006, 2009a, 2009b, 2010; Hirzy, 2000). A "Professionals' Statement to End Fluoridation" (www.fluoridealert.org/prof_statement.pdf) had over three thousand signers as of July 2010 (although many of the signers are not identified with respect to their areas of expertise, so it is not clear that all these "professionals" have expertise in relevant areas). However, professional and governmental organizations remain supportive of water fluoridation, and to our knowledge, the majority of dental health practitioners in the United States continue to support it.

There is no shortage of information; the literature search for a recent review of the efficacy and safety of fluoridation turned up over five thousand citations. However, after application of exclusion/inclusion criteria related to the quality of the research and after review of the full text of each remaining article, the author of the review selected just seventy-seven citations for inclusion (Yeung, 2008). Why has so much of the fluoridation literature been deemed to be of less than high quality? There are at least four difficulties inherent in these studies:

1. as with all epidemiological studies, those focused on the safety and efficacy of water fluoridation are complicated by a multitude of confounding variables (e.g., Taubes, 2006), not the least of which is the tremendous variability in water consumption and related fluoride dose of individuals (EPA, 2004);
2. in many cases the data cannot be interpreted without the application of sophisticated statistical methods, and even then statistical correlations do not necessarily imply causative relationships (e.g., Sigfried, 2010);
3. some of the alleged adverse effects of fluoride are associated with very rare conditions (e.g., osteosarcoma), making it difficult to detect small, but potentially significant, differences in study populations;
4. the results from studies with laboratory animals are often not complicated by confounding variables, but their relevance to humans and the concentrations of fluoride in public water supplies is often difficult to determine (Hayes, 2008, pp. 330–332).

In recent years, the difficulties associated with critical evaluation of research findings and associated conclusions have been exacerbated by the widespread use of the internet as a medium for distributing information and opinions. The opponents of fluoridation in particular have used the internet to advance their arguments and point of view. Although many of these sites contain useful information and cogent arguments, the sites and the information on them are not uniformly of high quality. In many instances it is difficult to evaluate the quality of material posted on websites focused on fluoride and fluoridation without a fairly thorough knowledge of the peer-reviewed literature.

While these scientific issues continue to be debated, it appears that within the general public the major concern is related to ethics, not quality of the research on benefits and adverse effects of water fluoridation. Thus, many opponents of water fluoridation would remain opposed to "mass medication" even if the safety and efficacy of the practice were clearly documented. So, today the controversy continues unabated. The situation is described quite well in a recent journal article:

> Plans to add fluoride to water supplies are often contentious. Controversy relates to potential benefits of fluoridation, difficulty in identifying harms, whether fluoride is a medicine, and the ethics of a mass intervention. We are concerned that the polarised debates and the way that evidence is harnessed and uncertainties glossed over make it hard for the public and professionals to participate in consultations on an informed basis. (Cheng et al., 2007, p. 699)

Findings

Throughout the United States, and in many countries around the world, the incidence of tooth decay has decreased significantly over the past several decades. Although claims have been made that adding fluoride to drinking water has been one of the main reasons for this decline, the data indicate that in many countries and communities progress in preventing caries has been made without fluoridated water.

For many years professional organizations and federal, state (including Alaska), and local governments in the United States have promoted the fluoridation of public water supplies, and these organizations and relevant government agencies still strongly support the practice. However, there has also been opposition to the practice since its inception in the 1940s. Although it appears that most dental practitioners and researchers still support fluoridation of municipal water supplies, it also seems that the number of practitioners and researchers who oppose the practice has increased. At this time the claims most often cited by opponents of fluoridation of water supplies are:

- lack of definitive evidence for efficacy,
- evidence indicating risk of adverse effects, and
- ethical issues related to mass medication.

Chapter 4

Legal and Ethical Issues

As indicated by testimony to the Fairbanks Fluoride Task Force, legal and ethical issues are perhaps the biggest concerns of the local residents who are opposed to fluoridation of Fairbanks' public water supply. The testimony received by the task force was overwhelmingly against fluoridation. During the ten task force meetings at which public testimony was invited, sixty-two testimonies were presented by thirty individuals (at the extremes eighteen individuals presented testimony just once, and one individual submitted testimony on six different occasions). The positions of the testifying individuals, as described by themselves or ascertained by the task force from the nature of the testimonies, were twenty-six against fluoridation, three in favor, and one with no clearly stated opinion. The major concerns voiced by the opponents of fluoridation were:

1. toxic and harmful effects of fluoride;
2. lack of high-quality evidence that fluoride in public water supplies effectively prevents dental caries;
3. unethical aspects of "mass medication," including lack of informed consent;
4. fluoridation of public water supplies interferes with freedom of choice, infringes on individual rights, and results from an overreach of governmental powers; and
5. the risk that fluoridation of public water supplies may do more harm than good.

While testimony and evidence on all five of these concerns were presented to the task force, concerns 3, 4, and 5 were highlighted for the task force by both the frequency and passion of testimonies related to them. They have also been voiced in the larger debate over water fluoridation. The "mass medication" argument is that fluoridation of public water supplies administers medication to an unaware and in some cases, unwilling public (see, for example, www.fluoridedebate.com/question34.html; Cross and Carton, 2003). The "individual rights" concern (#4) is related to the previous concern in that it questions governmental authority to implement the "mass medication" (Cross and Carton, 2003). The concern that water fluoridation may do more harm than good brings into the argument the "first, do no harm" precept of medical ethics. This precept basically says that in a given situation it may be better to do nothing if the action to be taken may cause more harm than good.

The legal concerns brought to the task force were considered in light of a rather lengthy history of legal challenges to fluoridation of public water supplies (Graham and Morin, 1999). Although fluoridation has been challenged numerous times in at least thirteen states, and while cases decided primarily on procedural grounds have been won and lost by both proponents of and opponents to fluoridation, no final ruling in any of these cases has stopped a proposed fluoridation program or ruled in favor of elimination of an existing program (Block, 1986; ADA, 2005; Pratt et al., 2002). In the process, the U.S. Supreme Court has declined to review fluoridation cases at least thirteen times (ADA, 2005).

In contrast to the legal question, which has repeatedly been addressed by the courts, the ethical issues remain problematic. On the one hand, opponents of fluoridation cite concerns about the propriety of forced "mass medication" and the integrity of at least some of the individuals and organizations that promote the practice (see, for example, Bryson, 2004; Cheng et al., 2007; Connett et al., 2010). On

the other hand, some proponents have argued that those who potentially have the most to gain from fluoridation of public water supplies—the economically and educationally disadvantaged and those with limited access to proper health care—do not have a voice in the development of health policies and practices unless those in power are looking out for their interests (McNally and Downie, 2000). Cohen and Locker (2001), observe that the conflict between beneficence of water fluoridation and autonomy remains unresolved and that "there appears to be no escape from this conflict of values, which would exist even if water fluoridation involved benefits and no risks" (p. 578). Further, they argue that although recent studies indicate that water fluoridation continues to be beneficial, critical analysis indicates that the quality of evidence provided by these studies is generally poor. Thus, they argue that from an ethical standpoint, past benefits of fluoridation cannot be used to justify continuation of the practice, and they call for new guidelines that "are based on sound, up-to-date science and sound ethics" (p. 579).

Chapter 5
Exposure

Fluorine, which exists in its elemental form as fluorine gas, is one of the most reactive elements. Its chemical reactivity is characterized by its propensity to accept electrons and to undergo reduction to the fluoride ion. While elemental fluorine is found in just one form, the fluoride ion exists in a number of compounds, including the common minerals fluorite and especially fluorapatite. Fluorine is also found in a group of compounds called "organic fluorides," compounds in which fluorine is chemically bonded to carbon. Some pharmaceuticals, consumer products, and pesticides are organic fluorides.

Concerns about the safety and efficacy of artificially fluoridated water revolve around one species, the fluoride ion—often referred to in this report as fluoride. Fluoride is easily absorbed in the human alimentary tract, is distributed to most—if not all—tissues, and is cleared from the blood and tissues by uptake into bone and by excretion (Whitford, 1996; National Research Council, 2006). It is capable of inhibiting certain enzymes (Scott, 1983, p. 166; National Research Council, 2006) and of affecting bacterial metabolism, including reducing the capability of plaque-forming bacteria to produce acid (Featherstone, 2000; Jones et al., 2005), which is the bacterial product responsible for caries. Given that fluoride has these biochemical properties, it is not surprising to find that it is toxic. The acute toxic dose of fluoride is 5 to 10 grams for a 155-pound person (Hodge and Smith, 1965; ADA, 2005). More precise determinations of toxicity have been performed with pure chemicals and laboratory rats, and these studies indicate, for example, that sodium fluoride is about ten times less toxic than sodium cyanide and about fifty times more toxic than sodium chloride (table salt).

The fluoride-containing compound of most interest in the Fairbanks situation is sodium fluorosilicate, the compound that Golden Heart Utilities (GHU) uses to fluoridate the water it distributes. Sodium fluorosilicate is toxic; for rats its LD50 is 125 mg/kg (that is when laboratory rats were given single doses of 125 mg of sodium fluorosilicate per kg of body weight, 50% of the test animals died). According to the National Institute of Health's TOXNET website (http://toxnet.nlm.nih.gov/cgi-bin/sis/search/a?dbs+hsdb:@term+@DOCNO+770), the acute toxic dose of sodium fluorosilicate for a human is between 3.5 and 35 grams. However, the low concentration of this compound in treated water (around 1.5 mg per liter) ensures that there is no acute toxicity threat associated with the treated GHU water. Nevertheless, concerns have been voiced about risks related to the use of sodium fluorosilicate in water fluoridation programs. In particular, a correlation was reported between use of sodium fluorosilicate to fluoridate water in various locales in the state of New York and levels of lead in the blood of children residing in these communities (Masters and Coplin, 1999; Masters et al., 2000). However, this correlation was not verified in a subsequent study (Macek et al., 2006). Furthermore, a causative link between the use of sodium fluorosilicate and elevated lead levels in blood of children who consume the fluoridated water would require that sodium fluorosilicate incompletely dissociates when it dissolves in water, a proposition put forward by Westendorf (1975) but which is inconsistent with the best contemporary evidence (Urbansky, 2002).

Because fluoride is found in a number of common minerals, it is not surprising to find that it is naturally present in water. The concentration of fluoride in the oceans is approximately 1.3 ppm (Turekien, 1969). In the United States, fluoride concentrations in wells, lakes, and rivers range from below detection to 16 ppm (National Research Council, 2006). For example, Lake Michigan's fluoride level is 0.17 ppm, wells in Arizona have concentrations up to 7 ppm, and groundwater in Bauxite, Arkansas, has up to 14 ppm fluoride (ADA, 2005). In Alaska, a voluminous DEC data sheet (Alaska Department of Environmental Conservation, 2010) demonstrates that although many natural water systems around the state have undetectable levels of fluoride, one area (Wales) has 2 ppm fluoride in groundwater, and several sources of groundwater in the Fairbanks area have from 0.1 to 0.3 ppm fluoride. Several independent studies of domestic, commercial, and monitoring wells in the greater Fairbanks area show that fluoride is present at concentrations ranging from 0.1 to 1.6 ppm (Fig. 5.1; USGS, 2001; Mueller, 2002; Verplanck et al., 2003).

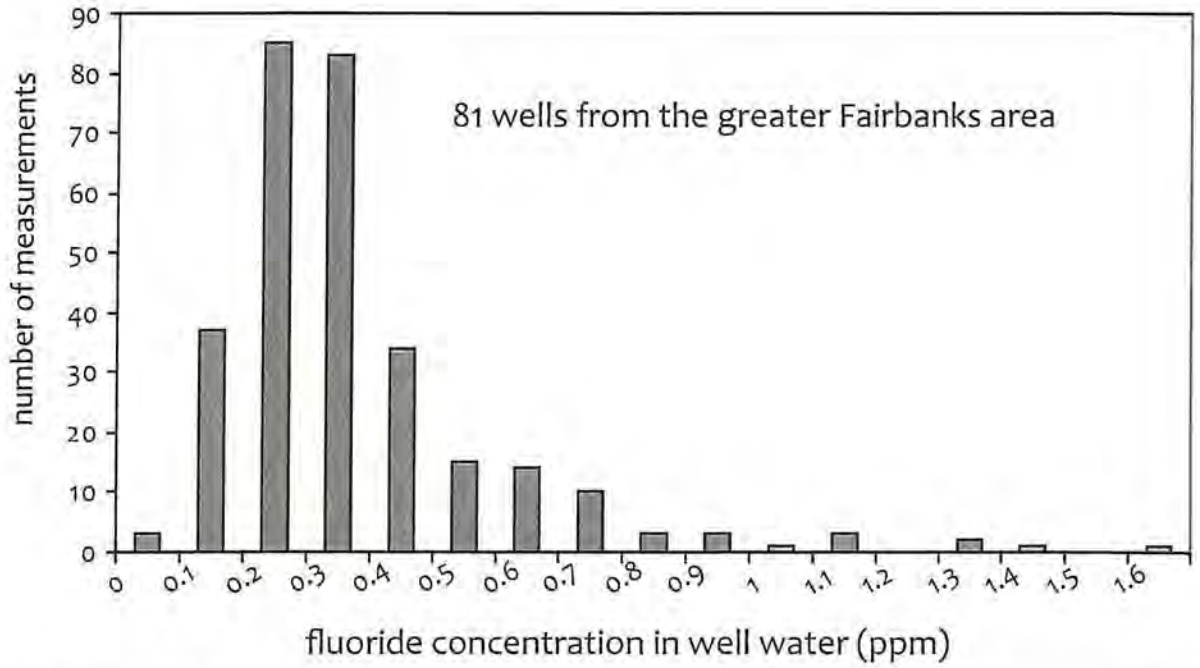

Figure 5.1. Histogram of fluoride concentrations in 81 wells in the Fairbanks area. The median value is between 0.2 and 0.3 ppm, and the bulk of values are between 0.1 and 0.7 ppm. Wells in metamorphic rocks contain the higher fluoride concentrations; those tapping the sedimentary aquifer have values of 0.2 to 0.4 ppm. Data from USGS, 2001; Mueller, 2002; Verplanck et al., 2003; and Alaska Department of Environmental Conservation, 2010).

Wells employed for Fairbanks city water are at depths greater than 100 feet below the surface and tap the sedimentary aquifer of the Fairbanks floodplain. The several hundred feet of sediment is essentially uniform in mineralogy and mineral compositions, hence, by reaction with groundwater it creates water with an essentially constant composition. The fluoride content of raw water from these wells has been tested numerous times between 1987 and 2008 yielding an average fluoride concentration of 0.34 ± 0.1 ppm (Fig. 5.2). Given the constant substrate for groundwater in the Fairbanks floodplain, there is every reason to consider this fluoride concentration to be the same for a very long time to come.

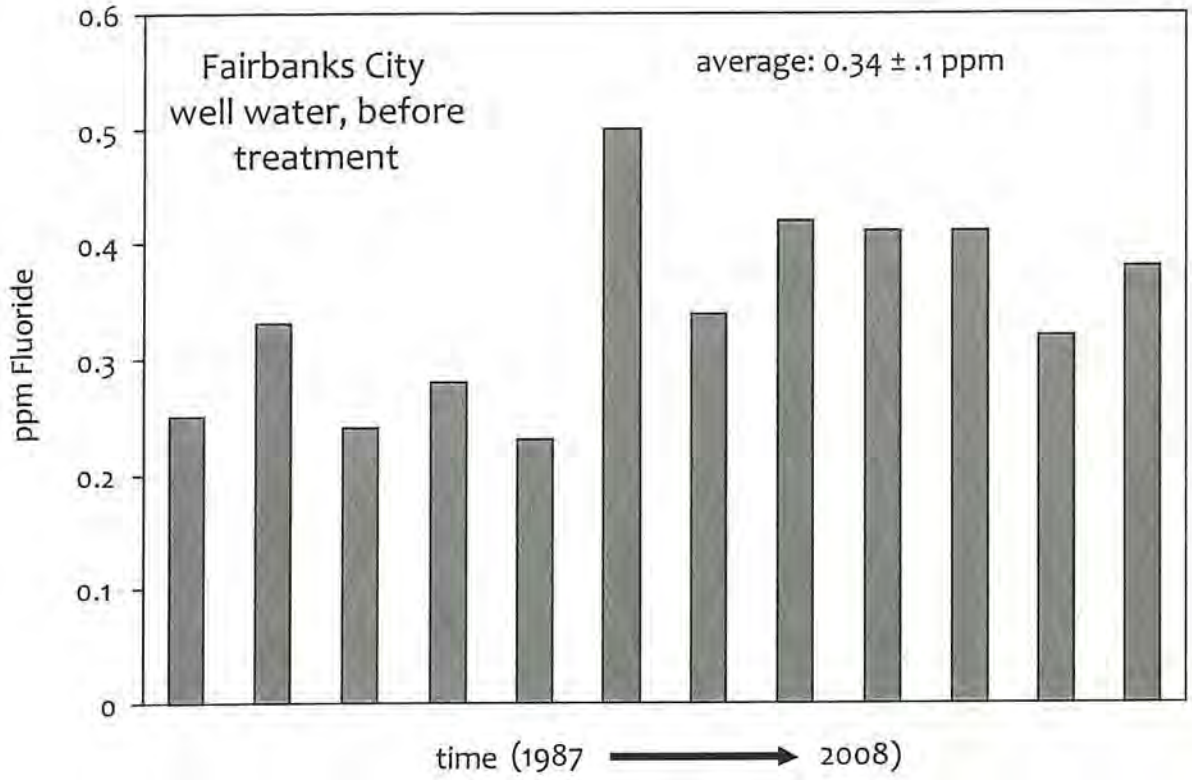

Figure 5.2. Fluoride concentrations in Fairbanks municipal raw well water prior to treatment and fluoridation. Each bar represents a single measurement. Based on checks of fluoride standards, the uncertainty of a given measurement is approximately 0.05 ppm. Data supplied by GHU.

A major source of exposure to fluoride for many Americans, including those who receive GHU water, is drinking water. While this exposure is clearly related to the concentration of fluoride in the water, it is important to distinguish between concentration and dose. The amount of fluoride (dose) an individual receives from drinking water depends on the concentration of fluoride in the water and the amount of water consumed. Thus an individual who drinks one liter of water containing 0.5 ppm fluoride receives the same dose of fluoride as another individual who drinks two liters of water containing 0.25 ppm. Various surveys have found that the amount of drinking water consumed by individuals varies considerably. For example, an EPA report (2004) states that the results from surveys done in the 1990s indicate that very young children consume an average of about 0.3 liter of drinking water per day and adults about 1 liter, as opposed to earlier EPA and WHO estimates of 1 liter and 2 liters, respectively. More importantly, the ranges of consumption are enormous: among the study subjects, infants less than one year old had water consumptions ranging from 0.03 liter to 1.5 liters, and the range among adults was from 0.1 liter to over 4 liters. The situation is further complicated by the fact that certain metal ions present in many water supplies can react with fluoride ions (before consumption) in a way that alters the uptake of fluoride from drinking water by humans (Institute of Medicine, 2000; Urbansky, 2002). For example, in seawater about one-half of the total fluoride is actually present as the MgF^+ complex ion (Bethke, 1996). Therefore, it is very difficult to determine how much fluoride any individual actually consumes from drinking water on a daily basis. Furthermore, "average consumption" is meaningful for a relatively small segment of the population (see Fig. 5.3 for one representation of the situation).

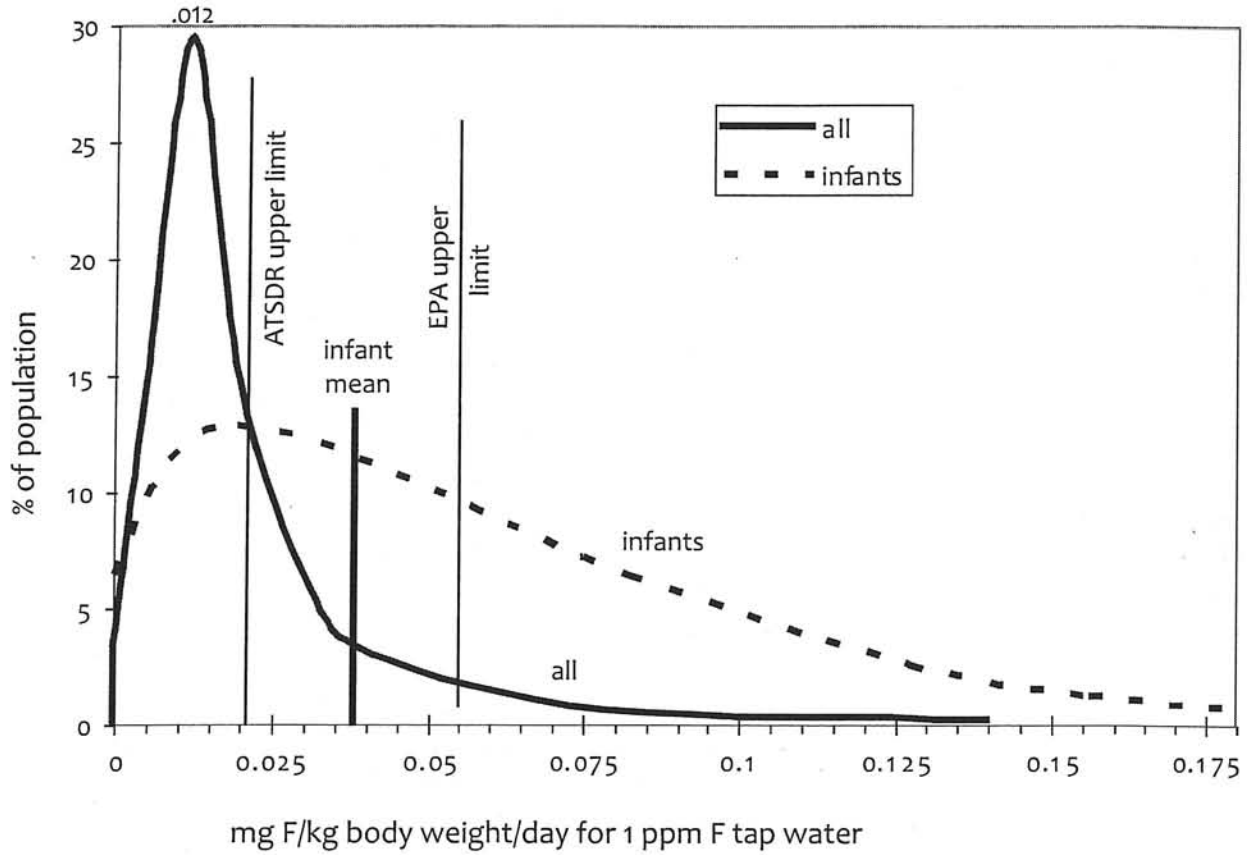

Figure 5.3. Fluoride consumption from tapwater distribution for total population (dark, solid curve) and for infants (dashed curve). Note that only a small proportion of the population receives the target dose from tap water and that a large proportion of infants receive a dose considerably higher than the target dose. Graph constructed from data in EPA (2000).

Agencies of the U.S. federal government, taking into account information that documents the adverse effects of human consumption of large doses of fluoride, have issued regulations and recommendations on the concentrations of fluoride ion in drinking water. The U.S. Environmental Protection Agency (EPA) has set a maximum contaminant level (MCL) for fluoride at 4 ppm and a secondary maximum contaminant level (SMCL) of 2 ppm (to provide a margin of safety against development of fluorosis from exposure to fluoride in drinking water—see Chapter 7). In 1962 the U.S. Public Health Service adopted standards that call for fluoride concentrations between 0.7 ppm and 1.2 ppm in public water supplies that have been "artificially fluoridated" or have "adjusted" levels of fluoride. This range of concentrations was selected based on estimates of water consumption that take into account differences based on climate and the assumption that people in warmer climates drink more tap water than do residents in cooler climates.

In January 2011, just as the Fairbanks task force was finalizing the first draft of its report and recommendations, two federal agencies initiated formal processes to change policy and regulations related to fluoride exposure. In early January, the secretary of the U.S. Department of Health and Human Services (HHS) issued a notice that HHS was seeking public comment on a proposed new recommendation that communities that are fluoridating or choose to fluoridate their public water

supplies adjust the fluoride concentration to 0.7 ppm (http://www.hhs.gov/news/press/2011pres/01/pre_pub_frn_fluoride.html). This recommendation is based on the considerations that (1) scientific evidence indicates that water fluoridation is effective in preventing dental caries, (2) fluoride in drinking water is now just one of several sources of fluoride, (3) the prevalence and severity of dental fluorosis has increased in recent years, and (4) the water consumption of children and adolescents is independent of ambient temperatures. At this writing, the HHS action is limited to initiating the public comment period and does not constitute a formal change in the HHS recommendation. A few weeks later, the EPA initiated a "Registration Review" of the pesticide sulfuryl fluoride. This chemical, used for controlling insect pests in a variety of stored agricultural products, breaks down during application to release fluoride ions. Although the fluoride residue from sulfuryl fluoride contributes negligibly to the fluoride exposure of individual humans, this proposal is based on the EPA's assessment that "aggregate fluoride exposure is too high for certain identifiable subpopulations in the United States, in particular children under the age of seven who live in areas with higher fluoride concentrations in drinking water resulting from natural background sources" (EPA, 2011; Office of Pesticide Programs, 2011). Under the Federal Food, Drug, and Cosmetic Act, EPA must withdraw sulfuryl fluoride under these circumstances, and the action initiated at this point (invitation for public comment) is the first step in the withdrawal process.

The exposure of a given individual in the Fairbanks area to fluoride from drinking water is very difficult to assess because of the various sources of drinking water available in the area. However, for the purposes of this report, we will focus on individuals who are served by the Golden Heart Utilities water system. This distribution includes about 30,000 people (approximately 6,500 hookups) in the city of Fairbanks and an additional 10,000 to 25,000 individuals (approximately 2,200 hookups, including several water delivery services) in the surrounding area served by College Utilities. Until January of 2011 the drinking water supplied to these individuals contained, on average, 1.0 ppm fluoride. The GHU records examined by the task force demonstrated that over an extended period of time, the range of fluoride concentration in the distributed water was from 0.8 to 1.1 ppm. The variability in the concentration of fluoride was probably due to measurement uncertainties and to the fluctuation in fluoride concentration in the feed water for the GHU process—averaging 0.3 ppm but ranging from 0.2 to 0.4 ppm (Fig. 5.2). In response to the HHS action described in the previous paragraph, in January of 2011 GHU reduced the concentration of fluoride in distributed water from 1.0 ppm to 0.7 ppm. Thus the GHU fluoridation process presently raises the fluoride concentration from about 0.3 ppm in the groundwater to 0.7 ppm in the distributed water.

The process used by GHU to produce water containing 0.7 ppm fluoride is one of the two most common approaches used elsewhere in the United States. A calculated amount of sodium fluorosilicate (SFS) is added to the raw water in a rather sophisticated treatment process. The SFS originates at KC Industries in Mulberry, Florida, where it is manufactured and purified as a byproduct from the domestic phosphate fertilizer industry. Each lot of SFS is analyzed and verified as meeting or exceeding American Water Works Association standards of purity before it is shipped. The material used by GHU is shipped from Florida by truck and container ship to Univar in Anchorage then by truck to Fairbanks. Univar has on record the certificates of assurance for the purity of each lot of SFS that it receives (R. Holland, personal communication). A member of the Fairbanks Fluoride Task Force conducted a laboratory analysis of a sample of SFS provided by GHU and found it to be impressively pure (Table 5.1) relative to typical laboratory chemicals. When used in the fluoridation

process, the calculated concentrations of metal ions added from the SFS are in the parts per trillion range, well below limits set by the EPA. While there are no guarantees against accidents in which fluoride levels in distributed water could rise to a dangerous point, the GHU fluoridation process is well run and has controls in place to provide a high level of assurance of safe operation. Each year since 2006 GHU has received a "Water Fluoridation Quality Award" from the Alaska Oral Health Program (Alaska Division of Public Health). The fluoride concentration in drinking water is measured three times each day, and the concentrations of eleven metals and radionuclides are analyzed on schedules that range from every three to nine years.

Table 5.1a. Major elemental components of a random sample of KC Industries' sodium fluorosilicate[a]

Element	Weight %	Element	Weight %
Silicon	14.8	Fluorine	60.3
Sodium	24.9	Chlorine	0.24

Table 5.1b. Trace elements in a random sample of KC Industries' sodium fluorosilicate[a]

Element	ppm	Element	ppm
Aluminum	25	Arsenic	<4
Barium	<5	Bromine	132
Cobalt	<1	Chromium	<1
Copper	<5	Iron	35
Iodine	35	Nickel	<2
Phosphorous	34	Lead	<1
Antimony	<5	Thorium	<0.5
Vanadium	<1	Tungsten	<2
Zinc	<2		

Table 5.1c. Approximate concentrations of elements added to Fairbanks water after the fluoride concentration has been adjusted to 0.7 ppm

Element	ppm	Element	ppm
Silicon	0.1	Fluorine	0.4
Sodium	0.2	Chlorine	0.002
Element	**ppt[b]**	**Element**	**ppt[b]**
Aluminum	21	Arsenic	<4
Barium	<4	Bromine	11
Cobalt	<1	Chromium	<1
Copper	<4	Iron	28
Iodine	28	Nickel	<1
Phosphorous	28	Lead	<1
Antimony	<4	Thorium	<0.4
Vanadium	<1	Tungsten	<1
Zinc	<2		

a. Analysis by XRF at the University of Alaska Fairbanks, Advanced Instrumentation Lab; R. Newberry, analyst
b. ppt = parts per trillion

Exposure of individuals to fluoride from dental products was not an issue when fluoridation of public water supplies was first introduced in the 1940s. Fluoridated toothpaste became commercially available in 1955, and it rapidly became widely accepted as an agent for caries prevention. However, inadvertent intake of fluoride from toothpaste can be a problem, especially with children who may have poor control of the swallowing reflex. Detailed studies of fluoride ingested by children from swallowing toothpaste have led to ingestion estimates ranging from 0.1 to 0.4 mg per brushing (Ophaug et al., 1985; Levy and Zarei-M. 1991; Rojas-Sanchez et al., 1999). A USPHS report (Institute of Medicine, 2000) summarized the findings by concluding that an average of about 0.3 mg of fluoride is introduced with each episode of tooth brushing in young children. Additional, and highly variable, amounts of fluoride may be ingested by individuals who take fluoride supplements (e.g., drops) or receive topical fluoride application by dental professionals.

Many foods and beverages contain detectable amounts of fluoride. The USDA National Fluoride Database on the fluoride content of a wide range of beverages and foods (USDA, 2004) contains an extensive list. Some representative entries from the USDA database are displayed in Table 5.2.

Table 5.2. Fluoride concentrations in selected foods and beverages available in the United States. Adapted from USDA National Fluoride Database of Selected Beverages and Foods (2004) and Lalumandier and Ayers (2000).

Food or Beverage	Mean (ppm)	Standard Deviation	Range (ppm)
Dairy Products	0.25	0.38	0.02–0.82
Grain and Cereals	0.42	0.40	0.08–2.01
Potatoes	0.49	0.26	0.210–0.84
Leafy Vegetables	0.27	0.25	0.21–0.84
Fruits	0.06	0.03	0.02–0.08
Sugar and Substitutes	0.28	0.27	0.02–0.78
Tea (brewed)	3.7	0.6	2.6–5.3
Soda Pop or Cola	0.5	0.1	0.05–0.8
Bottled Water[a]	NA	NA	0.02–0.94

a. An analysis of bottled water available in Scotland found some European bottled waters to contain nearly 6 ppm (MacFayden et al., 1982).

Part of the variation in fluoride concentrations in foods reflects differences in plant metabolism (for example, tea leaves seem to sequester higher concentrations of fluoride than do the leaves of lettuce or kale). However, one notable aspect of the range of fluoride concentrations in prepared foods is what is called the "halo effect"—the result of the use of fluoridated water to prepare foods and beverages (Griffin et al., 2001). Thus, the fluoride content of processed foods and beverages reflects, in large part, the fluoride concentrations in the water used in their processing.

While the halo effect is manifested in a variety of products, perhaps the most obvious is bottled water, a product of special interest to residents of communities with fluoridated water supplies because it provides an alternative to tap water. The fluoride content of bottled water is regulated by law (see National Research Council, 2006), and it can contain up to 2.4 ppm fluoride with no requirement for a statement of fluoride content on the label, unless fluoride has been added. The large range of

allowable concentrations of fluoride and the lack of a requirement for notification of fluoride content clearly compromises the utility of bottled water (as opposed to distilled water) as an alternative to fluoridated community water.

A final source of fluoride, or at least fluorine in some form, is from the air. This is largely due to trace amounts of pesticides and other industrial chemicals in the atmosphere. For the most part the fluoridated substances in the air are organic fluorides (as are some medications such as Prozac and Ciprofloxacin) rather than the fluoride ion found in water, dental products, foods, and beverages. Although our knowledge of the fate of fluorine from organic fluorides as the result of metabolism in the human body is very limited, it seems unlikely that the "fluoride" that comes from atmospheric sources adds significantly to the fluoride ion burden in humans.

Various estimates of the total fluoride exposure of individuals in the United States have been made, but the most comprehensive effort is probably that of an NRC committee (National Research Council, 2006). Tables 5.3 through 5.5, below, were constructed by the Fairbanks Fluoride Task Force from data in that report. The NRC committee's estimates of fluoride exposure from water were based on estimates of water consumption (EPA, 2000), which had been used in many of the studies considered by the committee. Because updated estimates of water consumption are now available (EPA, 2004), the task force substituted the updated estimates of water consumption and repeated the calculations used to construct Tables 5.3 through 5.5. The results are displayed in Tables 5.6 through 5.8.

*Table 5.3. Estimated fluoride exposure (mg/kg body weight/day) of U.S. populations on water with **1.0 ppm fluoride**, based on water intakes estimated in NRC (2006)*

Population	water[a]	toothpaste[b]	background food[b]	pesticides & air[b]	total exposure[c]	% from water
Nursing infant	.0260		.0046	.0019	.033	79
Non-nursing Infant	.0860		.0114	.0019	.099	87
1–2 year old	.0314	.0115	.0210	.0020	.066	48
3–5 year old	.0292	.0114	.0181	.0012	.060	49
6–12 year old	.0202	.0075	.0123	.0007	.041	49
13–19 year old	.0152	.0033	.0097	.0007	.029	52
20–49 year old	.0196	.0014	.0114	.0006	.033	59
50+ year old	.0208	.0014	.0102	.0006	.033	63

a. Assuming all water, tap plus other, at 1.0 ppm
b. NRC (2006), Table 2-9
c. NRC (2006), Table 2-11

*Table 5.4. Estimated fluoride exposure (mg/kg body weight/day) of U.S. populations on water with **0.7 ppm fluoride**, based on water intakes estimated in NRC (2006)*

Population	water[a]	toothpaste[b]	background food[b]	pesticides & air[b]	total exposure[c]	% from water
Nursing infant	.0182		.0046	.0019	.025	73
Non-nursing Infant	.0602		.0114	.0019	.074	81
1–2 year old	.0220	.0115	.0210	.0020	.056	39
3–5 year old	.0204	.0114	.0181	.0012	.051	40
6–12 year old	.0141	.0075	.0123	.0007	.035	40
13–19 year old	.0106	.0033	.0097	.0007	.024	44
20–49 year old	.0138	.0014	.0114	.0006	.027	51
50+ year old	.0146	.0014	.0102	.0006	.027	54

a. Calculated from Table 5.3, assuming all water, tap plus other, at 0.7ppm NRC (2006)
b. NRC (2006), Table 2-9
c. NRC (2006), Table 2-11

*Table 5.5. Estimated fluoride exposure (mg/kg body weight/day) of U.S. populations on water with **0.3 ppm fluoride**, based on water intakes estimated in NRC (2006)*

Population	water[a]	toothpaste[b]	background food[b]	pesticides & air[b]	total exposure[c]	% from water
Nursing infant	.0078		.0046	.0019	.014	56
Non-nursing Infant	.0258		.0114	.0019	.039	66
1–2 year old	.0094	.0115	.0210	.0020	.044	20
3–5 year old	.0088	.0114	.0181	.0012	.040	22
6–12 year old	.0061	.0075	.0123	.0007	.027	23
13–19 year old	.0046	.0033	.0097	.0007	.018	26
20–49 year old	.0059	.0014	.0114	.0006	.019	31
50+ year old	.0062	.0014	.0102	.0006	.018	34

a. Calculated from Table 5.3, assuming all water, tap plus other, at 0.3ppm
b. NRC (2006), Table 2-9
c. NRC (2006), Table 2-11

*Table 5.6. Estimated fluoride exposure (mg/kg body weight/day) of U.S. populations on water with **1.0 ppm fluoride**, based on water intakes estimated by EPA in 2004*

Population	water[a]	toothpaste[b]	background food[b]	pesticides & air[b]	total exposure	% from water
Nursing infant	.017		.0046	.0019	.024	71
Non-nursing Infant	.055		.0114	.0019	.068	81
1–2 year old	.029	.0115	.0210	.0020	.064	45
3–5 year old	.026	.0114	.0181	.0012	.057	46
6–12 year old	.017	.0075	.0123	.0007	.038	45
13–19 year old	.014	.0033	.0097	.0007	.028	50
20–49 year old	.018	.0014	.0114	.0006	.032	56
50+ year old	.018	.0014	.0102	.0006	.030	60

a. Calculated from Table 5.3, assuming all water, tap plus other, at 1.0ppm
b. NRC (2006), Table 2-9

Table 5.7. Estimated fluoride exposure (mg/kg body weight/day) of U.S. populations on water with ***0.7 ppm fluoride,*** *based on water intakes estimated by EPA in 2004*

Population	water[a]	toothpaste[b]	background food[b]	pesticides & air[b]	total exposure	% from water
Nursing infant	.012		.0046	.0019	.019	63
Non-nursing Infant	.039		.0114	.0019	.052	75
1–2 year old	.020	.0115	.0210	.0020	.055	36
3–5 year old	.018	.0114	.0181	.0012	.049	37
6–12 year old	.012	.0075	.0123	.0007	.033	36
13–19 year old	.010	.0033	.0097	.0007	.024	42
20–49 year old	.013	.0014	.0114	.0006	.026	50
50+ year old	.013	.0014	.0102	.0006	.025	52

a. Calculated from Table 5.4, assuming all water, tap plus other, at 0.7ppm
b. NRC (2006), Table 2-9

Table 5.8. Estimated fluoride exposure (mg/kg body weight/day) of U.S. populations on water with ***0.3 ppm fluoride,*** *based on water intakes estimated by EPA in 2004*

Population	water[a]	toothpaste[b]	background food[b]	pesticides & air[b]	total exposure	% from water
Nursing infant	.0051		.0046	.0019	.012	43
Non-nursing Infant	.017		.0114	.0019	.030	57
1–2 year old	.0087	.0115	.0210	.0020	.043	20
3–5 year old	.0078	.0114	.0181	.0012	.039	20
6–12 year old	.0051	.0075	.0123	.0007	.026	20
13–19 year old	.0042	.0033	.0097	.0007	.018	23
20–49 year old	.0054	.0014	.0114	.0006	.019	28
50+ year old	.0054	.0014	.0102	.0006	.018	30

a. Calculated from Table 5.5, assuming all water, tap plus other, at 0.3 ppm
b. NRC (2006), Table 2-9

Several things must be kept in mind when interpreting the data in these tables:

- The average intakes of water are based on two different estimates of water consumption (NRC, 2006; EPA, 2004). The following pairs of tables allow direct comparison of the overall estimated exposures based on the differences in estimates of water intake: Tables 5.3 and 5.6, Tables 5.4 and 5.7, Tables 5.5 and 5.8.
- The range of water intakes among individuals is quite large.
- For simplicity of calculation, the estimated intake of fluoride from water assumes that all water has the fluoride concentration indicated in each table. This clearly is not the case for someone who uses several sources of water (for example, well, public system, and bottled) on a regular basis. This assumption, coupled with the range of fluoride concentrations in commercial bottled water, injects quite a bit of uncertainty into the results of these calculations.
- The estimated amounts of fluoride ingested by individuals from toothpaste are for individuals who regularly brush twice daily with fluoridated toothpaste and who have control over swallowing.
- Estimates of intakes from food (and beverages) are really just educated guesses because of variability in diets and in the magnitude of the halo effect.

Despite the limitations on the validity of the estimates of exposure, the data in the tables can be evaluated in light of recommendations made by relevant organizations of health professionals. There

have been a number of recommendations through the years, and the situation is complicated by the fact that some recommendations are in terms of mg per individual per day and others in terms of mg per kg per day. In the opinion of the task force, the key recommendations on fluoride are:

- Adequate daily intake (Institute of Medicine, 1997):
  0.0014 mg/kg/day for infants 0–6 months
  0.06 mg/kg/day for infants 7–12 months
  0.05 mg/kg/day for other children and all adults
- Upper limits:
  Agency for Toxic Substances and Disease Registry (ATSDR): 0.023 mg/kg/day
  Environmental Protection Agency (EPA, 2010): 0.06 mg/kg/day
  Institute of Medicine tolerable upper intake (Institute of Medicine, 1997):
  0.1 mg/kg/day for newborns through age 8
  0.15 mg/kg/day for ages 9 through adult

The ATSDR limit (MRL, minimal risk level) is an estimate of the daily human exposure to sodium fluoride that is likely to be without appreciable risk of adverse noncancer health effects (set, in the case of sodium fluoride, by the lowest level of fluoride judged to be correlated with increased bone fracture rates and then divided by a "safety factor" of ten). The ATSDR "upper limit" of 0.023 mg/kg/day for fluoride cited in this report takes into account the fluoride content of sodium fluoride for which the ATSDR has set an MRL of 0.05 mg/kg/day. The EPA limit ("reference dose") is based on a "no observed adverse effect level" for mottling of the teeth. The Institute of Medicine limits (tolerable upper intake limits, or UL's), which were also endorsed by the American Dental Association in 1994 and the American Dietetic Association in 2000, are set to minimize the risk of dental fluorosis but are at or near those that have been associated with mild (Institute of Medicine, 1997) or even crippling (National Research Council, 1993) skeletal fluorosis. While these upper limit recommendations have been used in formulation of a number of public health programs, the opponents of fluoridation have often critiqued and questioned the propriety of the recommendations and have called for lower limits for exposure to fluoride (see, for example, Connett et al., 2010). The problems associated with using these guidelines to develop public policy is perhaps best illustrated by the observation that the adequate daily intakes recommended by the Institute of Medicine for individuals greater than six months of age are equal to or greater than upper limits recommended by the ATSDR and the EPA.

The relationships between estimated fluoride exposures of several subpopulations of Fairbanks residents consuming drinking water with 0.7 or 0.3 ppm fluoride can be analyzed with the aid of Figs. 5.4 and 5.5 (derived from Tables 5.7 and 5.8, respectively). In analyzing these data, it is important to keep in mind that the numbers represent "average" individuals and that the consumption of drinking water varies widely among individuals (Fig. 5.1). In the existing scenario (0.7 ppm fluoride in drinking water, Fig. 5.4), it is apparent that nursing infants (NI) are estimated to be exposed to daily fluoride doses well below those established by ATSDR, EPA, and IOM; those over twenty years of age (20+ YR) have exposure well below EPA and IOM upper limits and about at the limit recommended by ATSDR. However, non-nursing infants (NNI) and one to five year-olds receive daily doses significantly above the ATSDR recommendation, marginally below that recommended by EPA, and significantly below that recommended by IOM. In contrast, while drinking water with 0.3 ppm fluoride does place non-nursing infants and one to five year-olds at risk of exceeding ATSDR upper limits, the exposure of other age groups remains below the ATSDR recommendation. Furthermore, no age group risks exposure greater than the recommended upper limits of the EPA or IOM (Fig. 5.5).

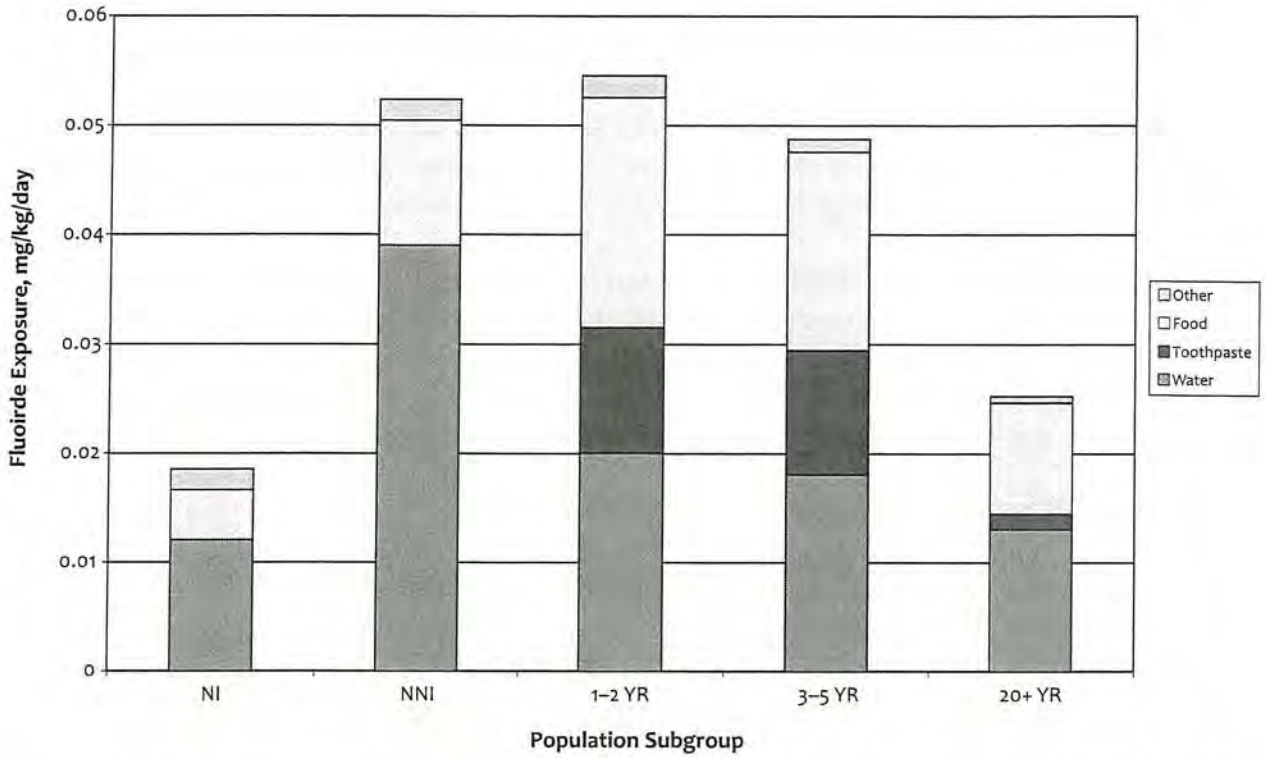

Figure 5.4. Estimates of fluoride exposure of individuals with 0.7 ppm fluoride in drinking water (data from Table 5.7)

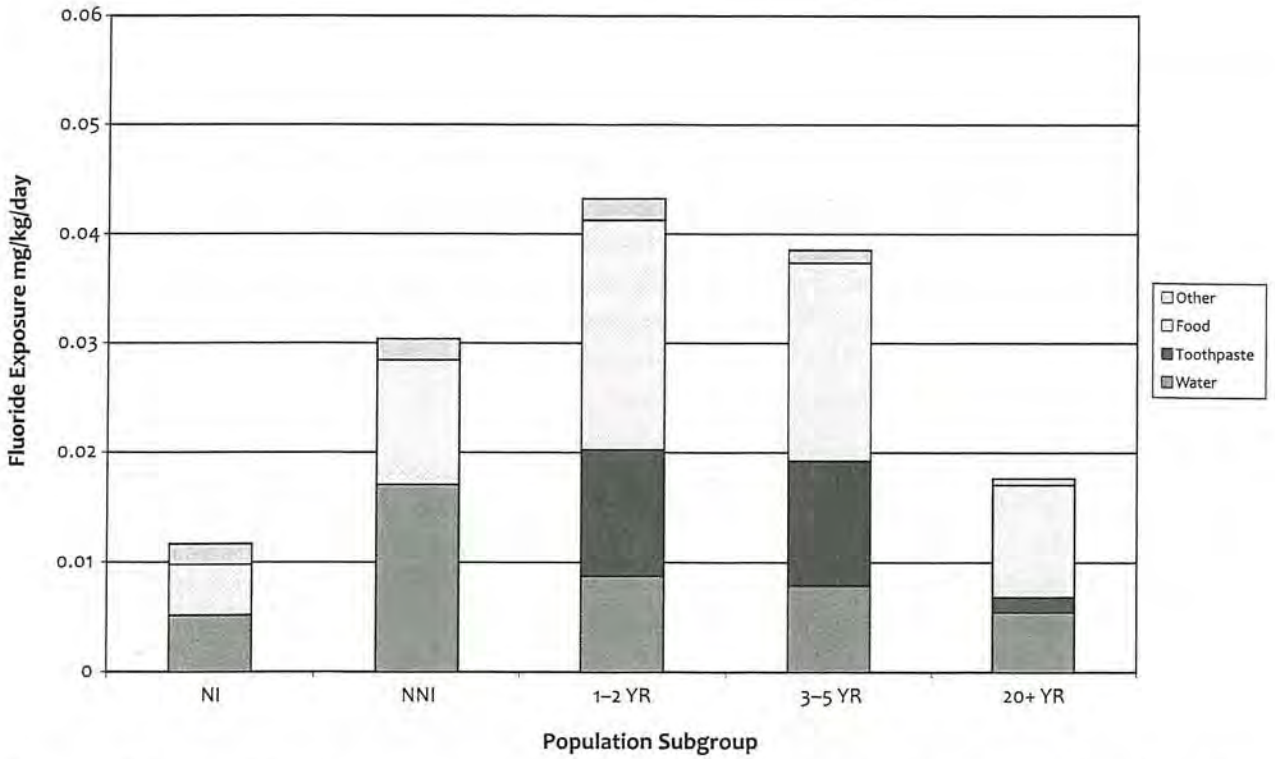

Figure 5.5. Estimates of fluoride exposure of individuals with 0.3 ppm fluoride in drinking water (data from Table 5.8). NI = nursing infant, NNI = non-nursing infant

In addition to the officially defined upper and lower limits for exposure to fluoride, there has been a widely accepted "optimal intake" of fluoride of 0.05 to 0.07 mg/kg/day. The optimal intake was thought to be a narrow range of doses that provide protection from caries but do not cause dental fluorosis. However, recently the concept of an "optimal" intake has been called into question because of (1) the overlap in fluoride intakes of groups of children who are caries-free and groups of children diagnosed with fluorosis and (2) the high variability in individual fluoride intakes (Warren et al., 2009).

Because the Fairbanks Fluoride Task Force had concerns about exposure of infants to fluoride and about the uncertainties associated with estimates of drinking water consumption, we performed some independent calculations. The results of the calculations for infants are displayed in Table 5.9. While the values in Table 5.9 are not identical with corresponding entries in Tables 5.3 through 5.5, the task force judges that they are sufficiently consistent, given the uncertainties and assumptions involved.

Table 5.9. Average fluoride intake per day by non-nursing infants (mg/kg/day)

Age	1 ppm F in water	0.7 ppm F in water	0.3 ppm F in water	upper limit
Birth	0.164	0.115	0.049	0.023,[a]0.10[b]
1 mo.	0.161	0.113	0.048	0.023,[a]0.10[b]
2 mo.	0.179	0.125	0.054	0.023,[a]0.10[b]
4 mo.	0.130	0.091	0.039	0.023,[a]0.10[b]
8 mo.	0.089	0.064	0.027	0.023,[a]0.10[b]
10 mo.	0.070	0.049	0.021	0.023,[a] 0.10[b]
12 mo.	0.065	0.045	0.019	0.023,[a] 0.10[b]

a. ATSDR

b. IOM (1997)

Findings

1. The problematic relationship between fluoride concentration in drinking water and "fluoride dose," due to varying amounts of water consumed by individuals and to other sources of ingested fluoride, severely complicates attempts to determine both health risks and benefits associated with 0.7 ppm fluoride in drinking water. In particular, commonly available foods and beverages contain from high (greater than 2 ppm) to negligible levels of fluoride, and fluoridated toothpaste is variably used and swallowed. We believe that these factors grossly complicate interpretation of drinking water studies and explain why the numerous studies conducted have come to a variety of conclusions that, in some cases, are quite different.

2. The concentration of fluoride in raw Fairbanks city water averages 0.3 ppm and is adjusted to 0.7 ppm in the treatment process. Because removing the fluoride from the raw water is impractical, the City of Fairbanks does not seem to have a realistic option for "fluoride free" city water (for a discussion of fluoride-removal processes see Fawell et al., 2006). Whatever benefits and detriments are caused by fluoride in drinking water will continue to a smaller degree if Fairbanks city water is no longer fluoridated.

3. Fluoride concentrations in Fairbanks area well water vary from 0.1 to greater than 1.0 ppm. Thus, some well water in the Fairbanks area contains more fluoride than fluoridated city water.

4. Fluoridation of Fairbanks city water has ramifications throughout the surrounding area because of the distribution of GHU water by College Utilities and several suppliers of trucked water.

5. The practice of fluoridation as carried out in Fairbanks has sufficient safeguards to protect public health beyond whatever health effects are associated with 0.7 ppm fluoride. The chemical employed is of sufficient purity and the manner in which it is added and monitored meets or exceeds standard practices.

6. An analysis of the estimates in Tables 5.3 through 5.8 and Figures 5.4 and 5.5 indicates that two segments of the Fairbanks area population must be considered separately with respect to professional recommendations on upper limits of fluoride exposure: (1) the average consumer of GHU water (fluoride concentration of 0.7 ppm) who is greater than five years of age is projected to consume less than the daily upper limits set by the EPA and IOM and just about at the upper limit set by ATSDR, and (2) children less than six years of age (with the exception of nursing infants) are projected to have total fluoride exposures that remain below the upper limits set by IOM and EPA but exceed those of ATSDR. It appears that drinking water with a fluoride concentration of 0.3 ppm would bring total fluoride exposure for those over 20 years of age well below even the most stringent of the recommendations of upper limits (ATSDR) and would significantly reduce concerns about overexposure of infants and young children. However, due to the tremendous variability in amount of drinking water consumed by individuals, the fluoride exposures of significant portions of the population are not adequately represented by the average values.

7. Nevertheless, the estimates of Table 5.9 highlight additional concerns about fluoride exposure of non-nursing infants in their first year. The use of fluoridated water to make up infant formula leads to levels of fluoride consumption that exceed recommended upper limits. While the magnitude of the problem obviously declines with a decline in fluoride concentration in the water used to make up formula, the most conservative of the upper limits of fluoride exposure would be approached or exceeded even when using GHU well water (fluoride concentration averaging 0.3 ppm) to which no fluoride has been added. While bottled water would seem to be the water of choice, the data of Table 5.2 indicate that not all bottled waters available in the United States would provide this level of protection. The use of bottled water for this purpose is further complicated by the absence of information about fluoride content on the labels of most bottled water. The only certainty for consumers seems to be that the distilled water sold in supermarkets has an undetectable concentration of fluoride.

Chapter 6

Efficacy of Community Water Fluoridation

Evaluation of Efficacy Before 2000

The addition of fluoride was effective in reducing caries in those municipalities that were the subject of reports in the primary dental literature during the mid-twentieth century. The Ft. Collins report gives the historical background that led to widespread fluoridation of public water systems:

> In 1901, a Colorado Springs dentist recognized that his patients with teeth with a brown stain or mottled dental enamel also had a very low prevalence of cavities (also called caries) (Centers for Disease Control and Prevention [CDC], 1999b). At this time in history, extensive dental caries were common, so this observation and its subsequent correlation with high amounts of fluoride ion in the water supply (2.0–12.0 milligrams per liter, mg/L) proved to be significant. Another dentist, H. T. Dean, DDS, took this information and conducted a survey of dental caries in relation to natural concentrations of fluoride in drinking water of 21 U.S. cities (Committee to Coordinate Environmental Health and Related Programs, USPHS [USPHS], 1991, pp. 18–19; CDC, 1999a, p. 934). Dean observed that at a concentration of 1 mg/L, fluoride would significantly reduce caries while causing a low incidence of mottled enamel, now called fluorosis, of the mostly very mild type. Beginning in 1945 and 1946, community trials were conducted over 13–15 years in four pairs of cities in the U.S. and Canada. These studies found a 50-70% reduction of caries in children following addition of fluoride (in the form of sodium fluoride) to community water supplies at 1 mg/L. The incidence of mild fluorosis remained low (CDC, 1999a, p. 936). Some of the early studies were criticized for lacking appropriate controls, not applying randomization, and not controlling for potential examiner bias (Sutton, 1960). However, the large effect sizes in these trials, along with replication of these findings in subsequent studies, led to the acceptance of community water fluoridation as a public health approach to caries prevention. (Fluoride Technical Study Group, 2003)

Many reviews and meta-analyses, which combine the results of several studies that address a set of related research hypotheses, support the hypothesis that water fluoridation reduces the incidence of caries. The York Report (McDonagh et al., 2000) is a systematic review made to assess the evidence of the positive and negative effects of population-wide drinking water fluoridation strategies to prevent caries. It is a summary of 254 studies published from the mid-1960s to mid-1999, which were chosen for relevance from over 3,000 studies identified in the literature. The authors of the York Report identified five objectives to make their assessment.

Their first objective was to answer the question: "What are the effects of fluoridation of drinking water supplies on the incidence of caries?" Of the 254 studies, twenty-six were relevant to this question. They are optimistic about the caries reductions caused by water fluoridation, yet cautious.

> The best available evidence suggests that fluoridation of drinking water supplies does reduce caries prevalence, both as measured by the proportion of children who are caries free and by

> the mean change in dmft/DMFT score. The studies were of moderate quality (level B), but of limited quantity. The degree to which caries is reduced, however, is not clear from the data available. The range of the mean difference in the proportion (%) of caries-free children is –5.0 to 64%, with a median of 14.6%. . . . The range of mean change in dmft/DMFT score was from 0.5 to 4.4, with a median of 2.25 teeth. . . . It is estimated that a median of six people need to receive fluoridated water for one extra person to be caries-free. . . . The best available evidence from studies following withdrawal of water fluoridation indicates that caries prevalence increases, approaching the level of the low fluoride group. Again, however, the studies were of moderate quality (level B), and limited quantity. The estimates of effect could be biased due to poor adjustment for the effects of potential confounding factors. (McDonagh et al., 2000, p. xii)

Their second objective was to answer the question: "If water fluoridation is shown to have beneficial effects, what is the effect over and above that offered by the use of alternative interventions and strategies?" Of the 254 studies, nine conducted after 1974 were relevant to this question. Again, their summary statement is positive toward the extra benefits of water fluoridation in the presence of other sources of fluoride:

> In those studies completed after 1974, a beneficial effect of water fluoridation was still evident in spite of the assumed exposure to non-water fluoride in the populations studied. The meta-regression conducted for Objective 1 confirmed this finding. (McDonagh et al., 2000, p. xii).

A summary of observed effects of fluoridation on caries in children is presented in Figs. 6.1 and 6.2 (McDonagh et al., 2000, pp. 12–13).

An examination of twenty-one studies, half of which were published between 1990 and 2000, came to a similar conclusion, although without as many caveats: "According to *Community Guide* rules of evidence, strong evidence shows that CWF (community water fluoridation) is effective in reducing the cumulative experience of dental caries within communities" (Truman et al., 2002, p. 28; see http://www.thecommunityguide.org/index.html for more about Community Guide).

A meta-analysis of twenty studies concluded that fluoride prevents caries among adults of all ages (Griffin et al., 2007). Some details are worth noting. Water fluoridation was responsible for preventing 27% of the caries. Self- and professionally applied topical fluoride was responsible for the remaining 73% reduction. For studies published after 1980, fluoride from all sources annually averted 0.29 carious coronal and 0.22 carious root surfaces per person. The authors point out the value of all types of fluoride for low-income adults and the elderly, who may not be receiving routine dental care. Note that the York Report (McDonagh et al., 2000) does not support this conclusion.

An epidemiological study in the United Kingdom addressed the question of differences in effect of water fluoridation over a range of socioeconomic groups (Riley et al., 1999). They conclude that water fluoridation reduced dental caries more in materially deprived wards than in affluent wards. In addition, the introduction of community water fluoridation substantially reduced inequalities in dental health. This conclusion is supported to an extent in the York Report (McDonagh et al., 2000, p. xii), although with considerable caution due to the low quality of the evidence and the general lack of variance

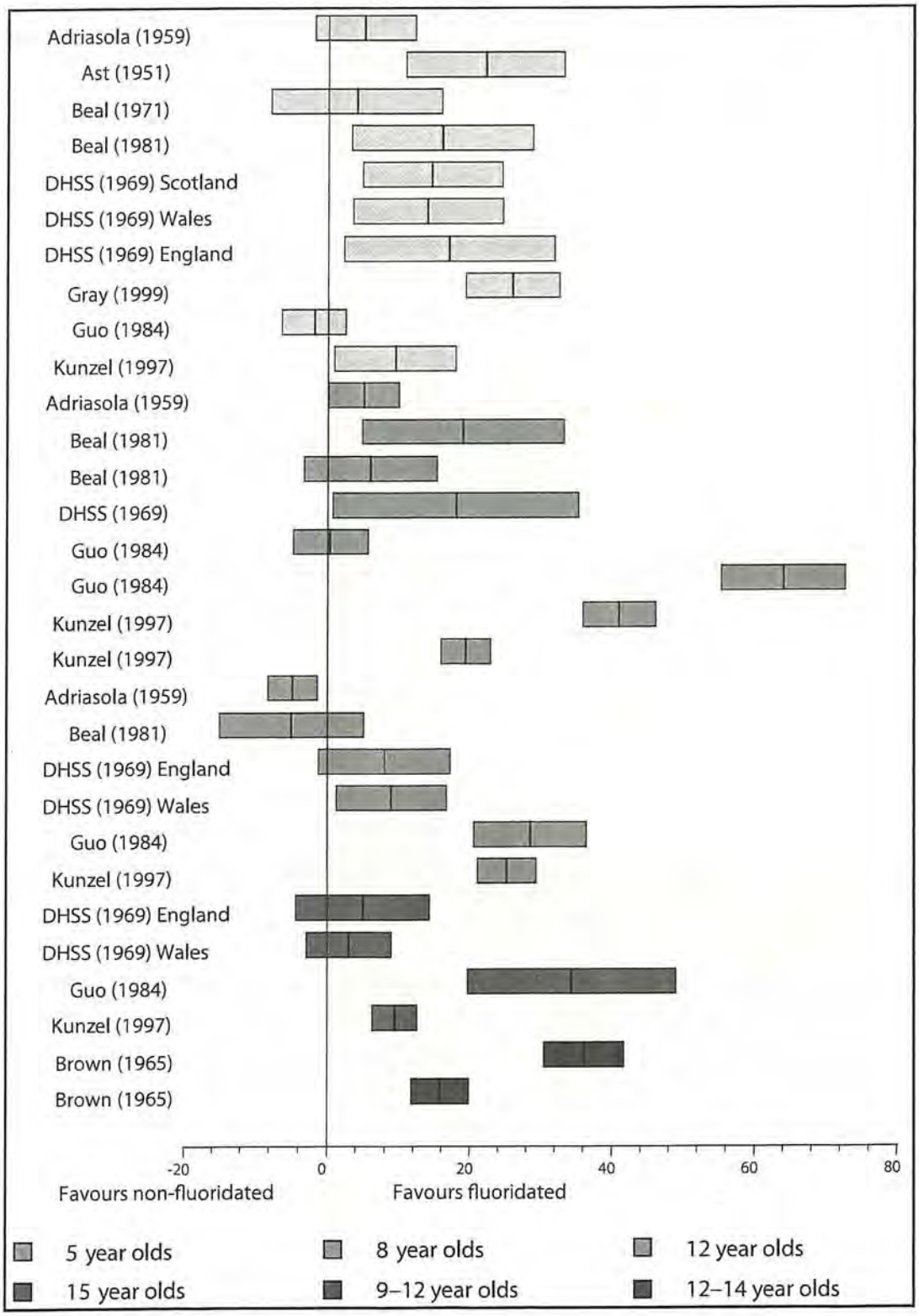

Figure 6.1. The mean difference of the change in the proportion (%) of caries-free children in the exposed (fluoride) group compared with the control group (low fluoride), for all ages extracted (color coded by age), for studies in which fluoridation was initiated after the baseline survey (McDonagh et al., 2000, p. 12)

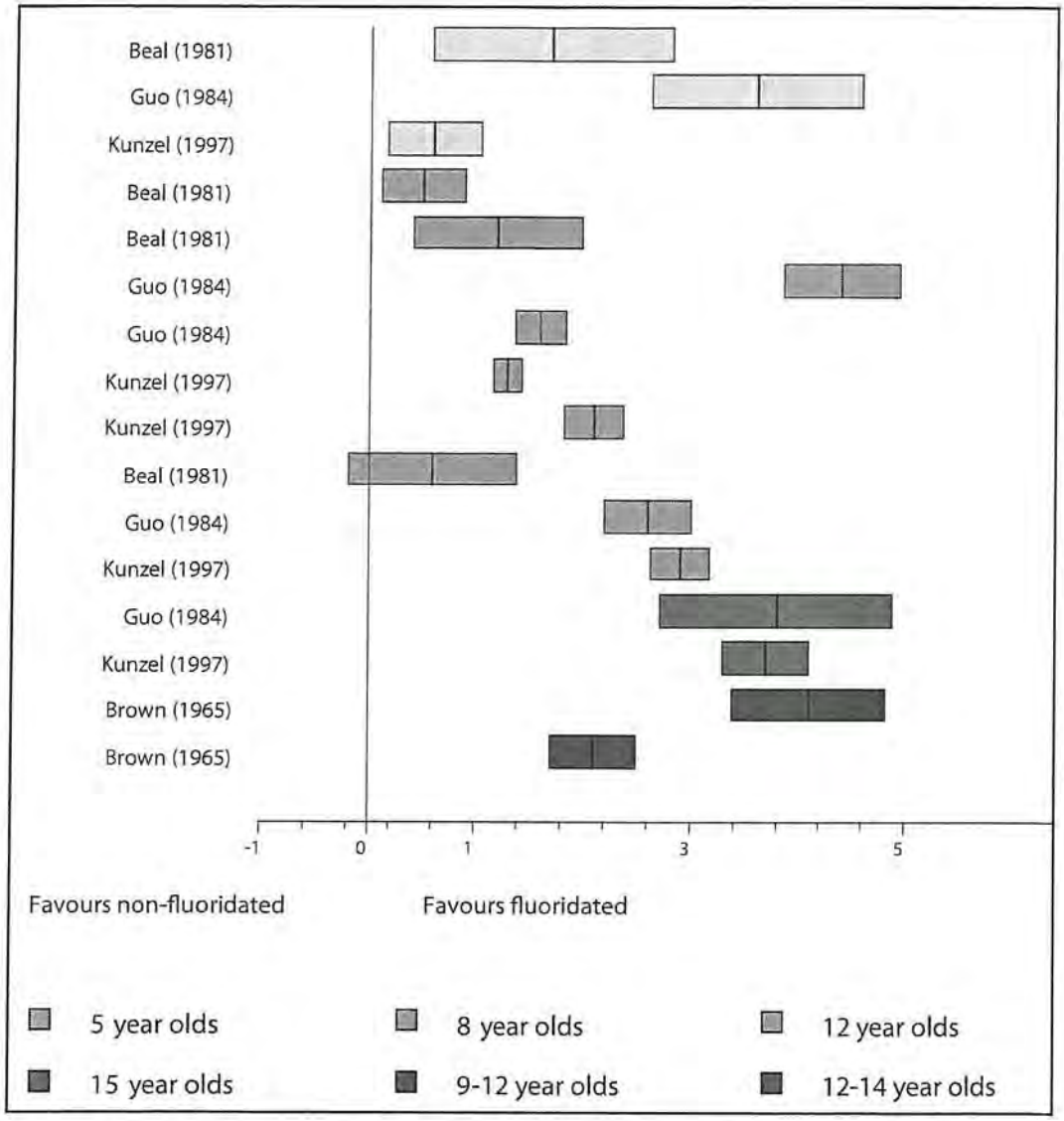

Figure 6.2. Change in dmft/DMFT Score (mean difference and 95% CI) (McDonagh et al., 2000, p. 13)

estimates in the fifteen studies. To objective 3, "Does water fluoridation result in a reduction of caries across social groups and between geographical locations, bringing equity?", their response was

> There appears to be some evidence that water fluoridation reduces the inequalities in dental health across social classes in 5 and 12 year-olds, using the dmft/DMFT measure. This effect was not seen in the proportion of caries-free children among 5 year-olds. The data for the effects in children of other ages did not show an effect. The small quantity of studies, differences between these studies, and their low quality rating, suggest caution in interpreting these results. McDonagh et al., 2000, p. xii)

It is apparently difficult to design and execute good studies to test the hypothesis that fluoridation of public water systems decreases the incidence of caries. Questions have been raised on a regular basis about the design and analysis of studies investigating the efficacy of municipal water fluoridation for the reduction of caries incidence. Concerns about experimental design and examiner bias were raised long ago (Sutton, 1960). The York Report (McDonagh et al., 2000), a meta-analysis of 214 studies published before 2000, presented relatively positive results for efficacy, with many caveats. In particular, they note the general lack of analysis, lack of control for potentially confounding factors, and the lack of any measure of variance for the estimates of decay. The difficulties of an accurate analysis and interpretation of data from a large and carefully designed longitudinal trial have been pointed out, with the observation made that "our analysis shows no convincing effect of fluoride-intake on caries development" in the permanent first molars in children between 7 and 12 years of age (Komárek et al., 2005, p. 145).

Equally important to the critical evaluation of the efficacy of water fluoridation to prevention of caries is "The Mystery of Declining Tooth Decay," which was reported in the journal *Nature* (Diesendorf, 1986). He notes in summary that "large temporal reductions in tooth decay, which cannot be attributed to fluoridation, have been observed in both unfluoridated and fluoridated areas of at least eight developed countries over the past thirty years" (p. 125). The magnitude of the reductions observed in unfluoridated areas were generally comparable with those observed in fluoridated areas over similar periods. In his discussion of the why's of the reductions, the author emphasized the literature that suggests changes in diet, immunity, and perhaps topical fluoride exposure with time are more likely candidates than fluoridated municipal water. The magnitude of the decrease in tooth decay is demonstrated in World Health Organization data, which was put into graphical form (Fig. 6.3) for the antifluoridation Fluoride Action Network (FAN) (Osmunson, 2010b).

The European experience has been one of generally decreasing DMFT scores. This is reported for fluoridated regions, nonfluoridated regions, and regions where fluoridation has been discontinued. In East Germany, the introduction of water fluoridation in Spremberg and Zittau brought about caries reduction averaging 48%. Surprisingly, caries levels for the twelve-year-olds of both towns significantly decreased following the cessation of water fluoridation (Kunzel et al., 2000). In Spremberg, DMFT fell from 2.4 to 1.4 (~40 %) and in Zittau from 2.5 to 2.0 (~20%). In Tiel (The Netherlands), where water fluoridation was discontinued in 1973, DMFS scores varied somewhat less consistently. The mean DMFS score increased between 1968/1969 and 1979/1980 from 10.8 to 12.7 (+18%) and then decreased to 9.6 (–26%) in 1987/1988. Overall the mean DMFS score decreased by 11% from 1968/1969, when water was fluoridated, to 1987/1988, when the town water had been

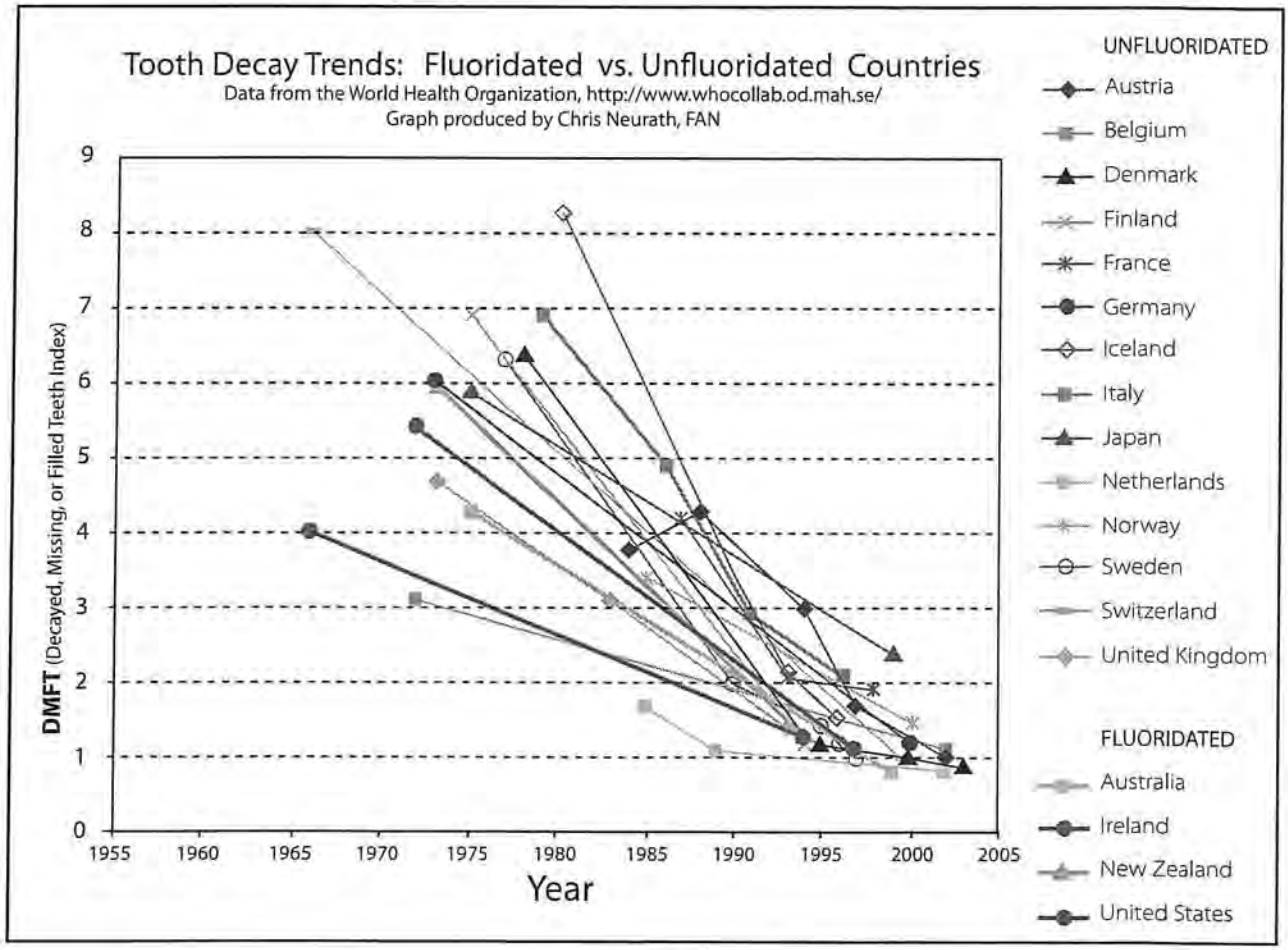

Figure 6.3. Tooth decay trends internationally in countries with fluoridated vs. unfluoridated water

unfluoridated for fourteen years. In Culemborg, where the water was never fluoridated, the mean DMFS score decreased from 27.7 in 1968/1969 to 7.7 in 1987/1988. This decrease of 72% occurred with no fluoridation of the public water supply (Kalsbeek et al., 1993). Presuming the application of existing preventive measures, the question as to whether water fluoridation would have had an additional effect if it had been continued cannot be answered, because no communities in The Netherlands now fluoridate water.

Evaluation of Efficacy After 2000

A recent review of community water fluoridation and caries prevention considers only recent data (Pizzo et al., 2007). Using MEDLINE as the primary database, the authors reviewed articles published from January 2001 to June 2006. They conclude that community water fluoridation is not necessary for caries prevention in modern, industrialized societies. Because the primary cariostatic action of fluoride occurs after tooth eruption, the use of topical fluoride is a more effective approach in communities where caries levels have become low. This line of thought is noted in a recent analysis published in the *British Medical Journal* (Cheng et al., 2007). The average number of decayed, missing, and filled teeth in twelve-year-old children in a number of European countries is near 1.5, and half of children have no cavities. There is no correlation in the downward trends with degree of

water fluoridation. Pizzo and coworkers are cautious, however, and these cautions may be germane in Fairbanks. They state that "water fluoridation may still be a relevant public health measure in populations where oral hygiene conditions are poor, lifestyle results in high caries incidence, and access to a well-functioning oral health care system is limited" (p. 192).

An evaluation of three reviews culled from fifty-nine publications published between 2000 and 2008 resulted in positive support for the effectiveness of water fluoridation in prevention of dental caries (Parnell et al., 2009). Two of the reviews have been discussed previously and they include mostly older literature (McDonagh et al., 2000; Griffin et al., 2007). The third review (National Health and Medical Research Council, 2007) identified one systematic review (Truman et al., 2002) and one cessation study (Seppa et al., 2000) published since the York Report (McDonagh et al., 2000). As noted above, the Truman study was strongly positive toward water fluoridation. In contrast, the Seppa study showed no evidence of increased caries when a previously fluoridated town reverted to nonfluoridated water. Parnell et al. concluded that the two new studies do not change the findings of the York Report that "the existing body of evidence strongly suggests that water fluoridation is beneficial at reducing dental caries" (p. 143).

A recent, somewhat indirect, study makes an association between lack of water fluoridation and inclusion of Nevada youth in the high caries prevalence group (Ditmyer et al., 2010). For adolescents in the study group (the 30% highest DMFT scores, DMFT > 4.0), 27.3% lived in a water-fluoridated community. For the control group (caries free, DMFT score = 0), 64.7% lived in a water-fluoridated community. Thus, participants living in nonfluoridated communities were almost twice as likely to be in the highest DMFT group as those living in fluoridated communities.

Discussions of efficacy may sometimes revolve around the mode of action of fluoride in optimally fluoridated water. The theoretical mechanism by which fluoride prevents caries has undergone significant revision since the introduction of community water fluoridation. The original systemic theory was that fluoride had to be ingested to incorporate into tooth mineral during its development (Dean et al., 1942). By the 1970s, doubts emerged regarding the exclusively pre-eruptive effect of fluoride. Numerous clinical studies suggested that fluoride action is predominantly post-eruptive (topical). While there are conflicting results, most recent epidemiological and laboratory studies indicate that topical application of fluoride plays the dominant role in caries prevention (CDC 2001; Hellwig and Lennon, 2004).

Fluoride's effect depends on its being in the right amount in the right place at the right time. It works primarily after teeth have erupted, especially when small amounts are maintained constantly in the mouth, specifically in dental plaque and saliva. The fluoride in saliva aids in enamel remineralization in enamel lesions by inducing apatite formation from calcium and phosphate ions present in saliva (Fejerskov et al., 1981). The effectiveness of toothpaste in decreasing the prevalence of caries is particularly clear. When introduced into the mouth, fluoride in toothpaste is taken up directly by dental plaque and demineralized enamel. Brushing with fluoride toothpaste increases the fluoride concentration in saliva 100- to 1,000-fold for one to two hours. Some of this salivary fluoride is taken up by dental plaque. The ambient fluoride concentration in saliva and plaque can increase during regular use of fluoride toothpaste (CDC, 2001).

In its recommendations, the CDC (2001) makes a strong argument supporting the topical mode of action in caries prevention. That said, they report that people living in communities with optimally fluoridated water who also use topical fluoride on a regular basis have a lower incidence of caries than people who use only optimally fluoridated drinking water or who only use topical fluoride. Thus the mode of action has been established in the modern literature as predominantly topical. Yet the epidemiological evidence, at least as reported a decade ago by CDC, still shows an empirical effect for fluoride in drinking water. Drinking fluoridated water prevents caries.

> When fluoridated water is the main source of drinking water, a low concentration of fluoride is routinely introduced into the mouth. Some of this fluoride is taken up by dental plaque; some is transiently present in saliva, which serves as a reservoir for plaque fluoride; and some is loosely held on the enamel surfaces. Frequent consumption of fluoridated drinking water and beverages and food processed in fluoridated areas maintains the concentration of fluoride in the mouth. (CDC 2001)

Thus, although the mode of action for fluoride in drinking water was initially thought to be systemic, its true action is predominantly topical in caries prevention, as is the action of the fluoride present in toothpaste, supplements, mouth rinse, and professionally applied gels and varnishes.

Publications and a federal proposal made even in the past year show that the jury is very much 'out' with respect to questions about the efficacy of community water fluoridation at 1 ppm fluoride and about the benefit-to-risk assessment.

- A proponent of community water fluoridation has recently written of the existing uncertainties associated with the efficacy of community water fluoridation (Newbrun, 2010). These include the effect of reducing the concentration of fluoride below 1 ppm, the expected result of discontinuing community water fluoridation in a community, and the role of socioeconomic factors in the importance of continuing water fluoridation.
- On January 7, 2011, the U.S. Department of Health and Human Services (HHS) announced a proposal recommending that water systems practicing fluoridation adjust their fluoride content to 0.7 ppm, as opposed to the previous temperature-dependent optimal levels ranging from 0.7 ppm to 1.2 ppm (http://www.hhs.gov/news/press/2011pres/01/20110107a.html, accessed January 27, 2011).
- An opponent of community water fluoridation has noted the 15% difference in the proportion of caries-free children reported in the York Report and the 20% to 40% reduction in tooth decay reported by the American Dental Association (Thiessen, 2009a). She has no apparent objection to the numerical accuracy. However, she does put these values in context: "which would translate to < 1 decayed, missing, or filled permanent tooth (DMFT) in older children and adolescents (based on U.S. data from CDC 2005). Is this adequate justification for imposing inadequately characterized risks?" (Thiessen, 2009a, p. 3).

Findings

1. There has never been a double blind, randomized, long-term study of the effectiveness of community water fluoridation on decreasing the incidence of caries. Nor has there been a comparable study on the effect of discontinuing water fluoridation on the incidence of caries.

2. The degree of caries reduction due to community water fluoridation was large and significant in the first decades that it was done. In recent decades, the degree of caries reduction attributed to community water fluoridation has decreased as other sources of fluoride have come into common use and as effective dental health measures have become more prevalent. The relative importance of water fluoridation is currently much smaller, more variable among populations, and perhaps unknowable.
3. The problematic relationship between fluoride concentration in drinking water and "fluoride dose" (due to varying amounts of water consumed by individuals and to other sources of ingested fluoride) severely complicates attempts to determine both health risks and benefits associated with 1 ppm fluoride in drinking water. In particular, at this time commonly available foods and beverages range from high (greater than 2 ppm) to negligible fluoride content, and fluoridated toothpaste is variably swallowed. We believe that these factors grossly complicate interpretation of drinking water studies and explain why the numerous studies conducted have come to a variety of different conclusions.
4. Studies of the relative effectiveness of community water fluoridation among socioeconomic groups give contradictory results. Dietary habits, dental hygiene, and intervention by health/dental providers are independent factors that confound the investigation of the efficacy of fluoridation of water on caries prevalence.

Chapter 7

Adverse Effects

Introduction

Fluoride can clearly lead to adverse health effects in humans. However, as for most chemicals, the dose that one is exposed to is a critical factor in determining the effect(s). For example, many drugs with therapeutic benefit are toxic at higher-than-recommended doses. Further, some drugs may have a very narrow window of therapeutic benefit. That is, the dose at which the drug provides benefit may be only slightly lower than the dose leading to ill effects. We focused primarily on studies that examined the effects on humans of drinking water with fluoride concentrations of less than 2 ppm (or 2 mg/L).

In Fairbanks (Golden Heart Utilities), the water is fluoridated to a concentration of 0.7 ppm. One challenge in understanding possible adverse effects is that, depending on water consumption and other possible sources of fluoride exposure (such as toothpaste or heavy tea consumption), individuals may be exposed to widely different doses of fluoride. Another challenge is that the average expected dose may also vary by age (an infant receiving most nutrition from formula reconstituted with fluoridated water vs. an infant who is breast fed), health (for example, patients with kidney problems vs. people with normal kidney function), or other confounding factors.

In this section we rely heavily on several comprehensive review studies. Notably, we frequently cite the 2006 National Research Council (NRC) report by the Committee on Fluoride in Drinking Water, *Fluoride in Drinking Water: A Scientific Review of EPA's Standards*. Although the purpose of this well-researched report was to determine if the Environmental Protection Agency's drinking water standard of 4 ppm maximum allowable concentration for fluoride protects the public from harmful effects of fluoride, the report also provides valuable information about possible effects of drinking water containing lower concentrations of fluoride, such as those found in Golden Heart Utilities water. We supplemented information from this report with other comprehensive reviews and with refereed literature, particularly those papers published since the NRC report came out in 2006.

Dental Fluorosis

Dental fluorosis, a mottling and/or pitting of the tooth surface due to fluoride exposure, develops in children during tooth formation when exposure to excess fluoride leads to disruption of the crystalline-enamel structure. Fluoride has a strong affinity for developing pre-eruptive enamel, leading to integration of fluoride into the crystal lattice. Teeth appear to be most susceptible to fluorosis at early maturation stages, which vary for different tooth types. For example, central incisors of the upper jaw are most susceptible at age 15 to 24 months for boys and age 21 to 30 months for girls (Fluoride Recommendations Work Group, 2001).

Infants primarily ingesting formula reconstituted with fluoridated water, even at concentrations recommended for municipal systems, may receive doses of fluoride that could lead to more than mild fluorosis or possibly other adverse health effects from fluoride. For example, a recent study (Levy et

al., 2010) found that participants with fluorosis of permanent incisors (generally rated as mild) had significantly greater intake of fluoride from reconstituted powdered infant formula or other beverages with added water than those without fluorosis. The clinical implication suggested by the authors is that avoiding ingestion of formula or other drinks mixed with fluoridated water can reduce the likelihood of fluorosis.

Due to the increased risk of fluorosis for non-nursing infants, in 2007 the American Dental Association (ADA) made an interim recommendation that infant formula be reconstituted with water that is fluoride-free or containing low levels of fluoride (ADA, http://www.ada.org/1767.aspx). In January 2011, the ADA rescinded the interim recommendation and issued a new recommendation based on research by the ADA's Council on Scientific Affairs (Berg et al., 2011). The new recommendations "for infants who consume reconstituted infant formula as the main source of nutrition" are (1) "Continue use of liquid or powdered concentrate infant formulas reconstituted with optimally fluoridated drinking water while being cognizant of the potential risk for enamel fluorosis" and (2) "Use ready-to-feed formula or liquid or powdered concentrate formula reconstituted with water that is either fluoride-free or has low concentrations of fluoride when the potential risk for enamel fluorosis is a concern." These "evidence-based" recommendations were ranked by the ADA as being "based on lower levels of evidence" (ADA, http://ebd.ada.org/contentdocs/ADA_Evidence-based_Infant_Formula_Chairside_Guide.pdf).

The results of fluoride exposure on developing teeth range from mild discoloration to highly stained and pitted teeth, depending on the concentration of fluoride and to a certain degree the susceptibility of the individual (NRC, 2006; Fagin, 2008). Severe enamel fluorosis characterized by pitting results in teeth that are very susceptible to dental caries. Severe fluorosis is estimated to occur at a rate of about 10% among children drinking water at the current EPA maximum allowable fluoride concentration (4 ppm) (NRC, 2006). The incidence of severe dental fluorosis is near zero where fluoride in water is below 2 ppm (NRC, 2006). But fluoride ingestion at levels commonly used to fluoridate water (1 ppm) can lead to mild to moderate levels of fluorosis. In its mildest form, fluorosis leads to opaque areas on the teeth. Estimates in the literature on the incidence of fluorosis vary, but it can be expected that at least 30% of school-aged children who consume water with between 0.7 and 1.2 ppm fluoride will have very mild or more severe dental fluorosis (Heller et al., 1997). A more recent study reported that the incidence of fluorosis has increased since the 1980s, and an analysis of data from 1999 to 2004 found that the prevalence of dental fluorosis in adolescents aged 12 to 15 is 41% (Centers for Disease Control and Prevention, 2010b). This condition has not been linked to other adverse health effects (Fagin, 2008). However, even mild fluorosis is considered by some to be of cosmetic concern. Since fluorosis cannot be reversed, treatment requires costly cosmetic dentistry where teeth are coated to hide the effects.

For slightly older children (16 to 36 months), fluorosis risk increases with higher fluoridated toothpaste ingestion. To avoid fluorosis, it is recommended that ingestion of toothpaste should be reduced through parental supervision and using only a small smear of toothpaste when brushing (Levy et al., 2010).

There are challenges to determining the relationship between fluorosis and dental caries. One challenge is consistent diagnosis of mild dental fluorosis, which is subjectively rated using various rating scales. Another challenge is that there is some evidence that fluoride delays the eruption of permanent teeth, thus affecting studies comparing caries rates in children of different age groups

exposed to varying fluoride concentrations (NRC, 2006). A final challenge that affects all studies linking water fluoridation to both positive and negative health effects is that the concentration in water can lead to widely different individual doses, depending on water consumption and exposure to other sources of fluoride.

Bone Effects and Skeletal Fluorosis

Since about 50% of ingested fluoride not excreted is deposited in bone, and 99% of the fluoride in a human body is contained in the skeleton (cited in Bassin et al., 2006), a number of studies have examined the effects of fluoride on bone. Ingestion of fluoride at very high concentrations results in thickened bone and can lead to bone deformities (skeletal fluorosis). Debilitating skeletal fluorosis is rare in the U.S. (NRC, 2006), and there is no evidence that ingestion of fluoride at levels used to treat drinking water leads to significant skeletal fluorosis. However, exposure to fluoride at relatively high concentrations has been linked to an increased risk of bone fractures because fluoride incorporation, while increasing bone density, also leads to a decrease in bone strength. The Committee on Fluoride in Drinking Water (NRC, 2006) found that people consuming drinking water containing 4 ppm or greater fluoride over their lifetime had an increased risk of bone fractures. However, they could not reach a conclusion about the relationship between consumption of water containing lower concentrations of fluoride and risk of bone fractures.

There are a number of studies on the relationship between fluoride consumption and bone fractures. Interestingly, since fluoride is known to increase bone density, treating patients at risk of osteoporosis with fluoride was once a clinically accepted strategy. However, studies suggesting, at best, no protection against fractures and a high level of side effects have led to a decline in fluoride treatment (Vestergaard et al., 2008). Studies are confounded by factors that include the possibility that fluoride may affect different bones differently (NRC, 2006). Two comprehensive reviews of the literature have concluded that there is no clear association between hip fractures (either positive or negative) or osteoporosis and water fluoridation (McDonagh et al., 2000; Yeung, 2008). Overall, the data suggesting an increased risk of bone fractures in populations drinking fluoridated water in the concentration range recommended for drinking water are not conclusive.

Cancer

The potential link between fluoride and cancer, most specifically osteosarcoma, is an area of recent controversy. Since fluoride incorporates readily into developing bone and increases the proliferation of osteoblasts, it has been hypothesized that there could be a link between fluoride and osteosarcoma. Published studies have drawn different conclusions about whether or not there is a relationship, in part complicated by the relative rarity of this type of cancer. But several studies have indicated a potential link, including a 1990 study conducted by the U.S. National Toxicology Program (Bucher et al., 1991). In this study, where rats were exposed to high levels of fluoride, there appeared to be a relationship between osteosarcoma frequency in male rats and the level of exposure to fluoride.

A more recent paper by Bassin et al. (2006) on humans used a case-control approach to assess the patient history of 103 patients with osteosarcoma matched with 215 controls. The authors concluded "our exploratory analysis found an association between fluoride exposure in drinking water during

childhood and the incidence of osteosarcoma among males but not consistently among females." Interestingly, Dr. Bassin's PhD supervisor, Chester Douglass, challenged the data in a rebuttal published in the same issue of the journal that the Bassin et al. paper appeared (Douglass and Joshipura, 2006). In that rebuttal he suggested that a paper was forthcoming with more extensive data that would show no link. To date, no such paper has been published. Our task force committee chair contacted Dr. Douglass by e-mail to try to get more information. Dr. Douglass was not forthcoming with information, only stating that: "A paper has been submitted to a scientific journal for publication. Thank you for your interest." A literature search in late November 2010 did not find a publication on this topic by Dr. Douglass.

While the Bassin paper is intriguing, the authors admit that the results are in contrast to several other case control studies (see Bassin et al., 2006) that found no link between fluoride consumption and osteosarcoma. They were careful to outline limitations to their preliminary study, including lack of data on actual consumption of fluoride by their subjects, lack of data on other potential unidentified factors, and selection bias. The authors cautiously referred to their study as "exploratory" and urged that "further research is required to confirm or refute this observation." Unfortunately, as of 2010 it appears that no more comprehensive studies have been published that might shed light on a possible link between fluoride consumption and osteosarcoma. We find that although there may be such a link, the data published to date suggesting a link are limited and published studies are conflicting in their conclusions. This conclusion is supported by comprehensive reviews of the literature (Yeung, 2008; McDonagh et al., 2000), which both concluded that there is no clear association between water fluoridation and overall cancer incidence and mortality.

Other Effects

Endocrine Effects: Fluoride exposure has been shown to affect some endocrine glands and may function as an endocrine disruptor. Although fluoride is generally not thought to accumulate in soft tissues, there is evidence that it may accumulate in the thyroid where exposure can lead to decreased thyroid function. According to the NRC's *Fluoride in Drinking Water* report (2006), many effects of low-dose fluoride exposure may be "subclinical effects, meaning there are no adverse health effects." However, they also point out that "borderline hormonal imbalances" might lead to an increased risk of adverse health effects. Their report concluded that studies to date on the effects of fluoride on endocrine function have limitations and that further research is needed to explore the possible connections between fluoride, particularly at low doses, and endocrine function. Additional research is important since there is some indication that concentrations of fluoride in drinking water of 4 ppm or less may affect endocrine function in "young children" or in "individuals with high water intake."

Neurotoxicity and Neurobehavioral Effects: A number of studies have reported changes to the nervous system following fluoride exposure that could lead to functional effects. Of the neurobehavioral studies, epidemiological studies suggesting a link between fluoride exposure and cognitive abilities are of particular interest. For example, several Chinese studies have consistently reported lower IQs in children drinking water containing 2.5 to 4 ppm fluoride (e.g., see NRC, 2006). The mechanism of the action of fluoride on IQ is not clear (Tang et al., 2008) but could be related to changes in membrane lipids in brain cells or to effects of fluoride on thyroid activity. It is unclear how the Chinese studies relate to U.S. populations, since U.S. populations are generally

exposed to drinking water with less than 2.5 ppm and there may be other confounding factors affecting the Chinese communities studied. Although the NRC's Fluoride in Drinking Water committee (2006) did not include neurological effects on their list of adverse effects not protected by the current EPA maximum allowable concentration for fluoride in drinking water, they did strongly advise that because of the "consistency of the results" in studies, such as those conducted on Chinese populations, additional research on the effects of fluoride on intelligence and on other neurological processes is warranted. A literature search conducted in December 2010 did not find published results that provide new information. It appears that there is reasonably good evidence that fluoride in drinking water at concentrations above 4 ppm may have neurological effects, including an effect on cognitive abilities. But the effects, if any, at lower concentrations of fluoride are not clear.

Effects on Other Organ Systems: Other systems that may be affected by fluoride exposure include the gastrointestinal system, kidneys, liver, and immune system. The NRC committee (2006) found a lack of well-documented studies on humans exposed to drinking water at 4 ppm or less for all of these systems. They concluded that the risk of adverse effects was likely to be low for most individuals drinking water with fluoride at 4 ppm but that there is a possibility of adverse effects in particular subpopulations such as those with renal impairment. In an apparent response to the possibility of an increased risk of adverse health effects for renal-impaired patients, the National Kidney Foundation recently changed its position on fluoridated water from "safe" to "takes no position" and "further research is needed" (www.kidney.org/atoz/pdf/Fluoride_Intake_in_CKD.pdf).

Findings

1. The problematic relationship between fluoride concentration in drinking water and "fluoride dose" (due to varying amounts of water consumed by individuals and to other sources of ingested fluoride) severely complicates attempts to determine both health risks and benefits associated with 1 ppm fluoride in drinking water. In particular, at this time commonly available foods and beverages range from high (greater than 2 ppm) to negligible fluoride content, and fluoridated toothpaste is variably swallowed. We believe that these factors grossly complicate interpretation of drinking water studies and explain why the numerous studies conducted have come to a variety of different conclusions.
2. The only commonly agreed-upon adverse effect related to drinking water with 1 ppm fluoride is mild dental fluorosis. Although debate continues concerning the quality of the studies, there are a large number that report deleterious effects from elevated fluoride in drinking water. On the other hand, numerous communities around the world use drinking water with natural fluoride concentrations of 1 ppm with no obvious ill effects, aside from mild dental fluorosis.
3. A fluoride concentration in water of 4 ppm is not protective for several adverse effects, including bone effects. That means that at best there is only a safety factor of about six for persons drinking Fairbanks water fluoridated to 0.7 ppm.
4. Although there may be a link between fluoride and osteosarcoma, the data published to date suggesting a link are limited and published studies are conflicting in their conclusions.
5. Fluoridated water is not recommended for all consumers. Recently several organizations have expressed concern about using fluoridated water to reconstitute infant formula. Consequently, the American Dental Association has recommended that parents of infants who primarily consume

reconstituted formula consult with their health care providers about the potential risks of using fluoridated water to make up infant formula. Despite those recommendations and cautions, pediatricians in the Fairbanks area (polled by committee member Dr. Medford) were not aware of these recommendations. The National Kidney Foundation has also changed its position on fluoridated water from "safe" to "takes no position" and "further research is needed."

6. Research on possible adverse effects of drinking fluoridated water (at concentrations less than 2 ppm) on the endrocrine glands, nervous system, or other organ systems has showed mixed results, with many studies showing no effects. However, studies involving extensive review of the literature (e.g., McDonagh et al., 2000; NRC, 2006) recommend that more high-quality research is warranted.

Chapter 8

Socioeconomic Issues

One of the public policy arguments put forward for fluoridation of public water supplies has been that it reduces disparities in dental health among populations. The argument goes that, if fluoridated water reduces the incidence of caries, it seems reasonable that the availability of fluoridated water for an entire community should provide particular benefit to those with the greatest risk of developing caries. This argument has been strongly put forward by professional organizations and government officials, including former U.S. Surgeon General David Satcher who "noted that water fluoridation is a powerful strategy in efforts to eliminate health disparities among populations" (ADA, 2005, p. 46).

For decades it has been noted that members of lower socioeconomic categories have significantly higher rates of caries than those who are more fortunate (Kozol, 1992; CDC, 2010a), so fluoridation should provide particularly valuable benefits to these groups. The refereed literature contains numerous reports that support (for example, Riley et al., 1999; Jones and Worthington, 2000) and refute this proposal (for example, Bradnock et al., 1984; Carmichael et al., 1989). McDonough et al. (2000) could reach no clear consensus on whether this public policy argument is valid, and shortly thereafter Cohen and Locker (2001) concluded that there is "little evidence that water fluoridation has reduced social inequalities in dental health" (p. 579). However, the most recent reviews of the matter tend to be guardedly positive (Cheng et al., 2007; Pizzo et al., 2007; Parnell et al., 2009; Newbrun, 2010). Newbrun's review provides a good example of the dilemma. It cites evidence in support of the proposition but concludes by stating, "whether fluoridation reduces disparities in caries is a continuing research question."

Arguments that members of lower socioeconomic groups disproportionately benefit from fluoridation of public water supplies raise questions about the existence of evidence that these groups also bear elevated risk of adverse effects from consuming fluoridated water. While the task force could find no good evidence on this topic, it does note that there is documentation that breast-feeding rates among mothers from lower socioeconomic groups are lower than those of their more affluent counterparts (Scanlon et al., 2010). Thus the task force's concerns about the exposure of formula-fed infants to fluoride (see Chapter 5) are particularly directed toward those from lower socioeconomic groups.

Finding

Although claims are made both that the detriments and the benefits of fluoridated water are greater for those in lower socioeconomic status, documentation of this is not conclusive.

Chapter 9
Cost

The proponents of water fluoridation continue to tout its cost effectiveness. For example, both the Centers for Disease Control and Prevention (CDC, 2010a) and the American Dental Association (ADA, 2005) claim that the fluoridation of public water supplies in the United States costs between approximately $0.50 and $3.00 per person per year and provides something on the order of $40 per person in annual benefits (decreased costs of dental care) for every dollar invested. However, both costs and benefits are very difficult to identify and quantify in any generally agreed upon and reliable way, so there is widespread disagreement about the legitimacy of any of these estimates.

In Fairbanks, the only clearly quantifiable cost of the water fluoridation program is the annual GHU expenditure for sodium fluorosilicate, which is $10,000 to $12,000 per year. The additional indirect costs to GHU for handling the material, adding it to the water, and monitoring the concentration of fluoride in the distributed water are difficult to estimate but are probably negligible in that these duties are incorporated into the work schedules of employees who dedicate the majority of their time and effort to other responsibilities. Similarly, while there are real costs associated with the purchase, operation, and maintenance of equipment used in the fluoridation process, those costs have never been documented but are probably modest.

If GHU discontinues its fluoridation process, it will have to adjust its protocol for conditioning the distributed water. While the task force did not investigate the projected costs of the required changes (mostly focused on maintenance of an appropriate pH), it seems likely that they will not be significant.

No attempts have been made to quantify indirect medical and dental costs or benefits resulting from the fluoridation of Fairbanks water.

Finding

There is little in the way of reliable data that can be used to estimate the cost of fluoridating Fairbanks' water or the net savings or costs associated with discontinuing the existing fluoridation process.

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Appendix A
Resolution

Introduced By: Council Member Cleworth
Introduced: February 8, 2010

RESOLUTION NO. 4398

A RESOLUTION ESTABLISHING A TASK FORCE TO RESEARCH CURRENT POLICY REGARDING FLUORIDATION OF THE MUNICIPAL WATER SUPPLY.

WHEREAS, the health and security of Fairbanks citizens are a primary concern of the City Council; and

WHEREAS, the use of fluoride in the City's water supply was established in 1960 (FGC Sec. 82-1) as a way to enhance dental care; and

WHEREAS, this practice has raised questions regarding potential long-term effects caused by the use of fluoride; and

WHEREAS, it is advisable to periodically reanalyze this policy to make sure the potential benefits outweigh any potential side effects associated with fluoridation; and

WHEREAS, the amount of research available on this subject is voluminous and often extremely technical.

NOW, THEREFORE, BE IT RESOLVED, that a committee is formed consisting of the six individuals listed below to research documentation provided by both proponents and opponents of fluoridation through public hearings and to supplement this information with any other sources deemed appropriate. A final report along with analysis and recommendations will be presented to the City Council no later than early July. Legal notifications and assistance will be given by the City Clerk's office. The committee consists of individuals having extensive backgrounds in chemistry, biology, dentistry, and medicine, who have expressed a strong interest in objectively analyzing research regarding fluoridation.

Committee Chair: Dr. Paul Reichardt, former Provost, Dean, and Professor at UAF, with a Ph.D. in Organic Chemistry;

Dr. Dick Stolzberg: Professor Emeritus of Chemistry at UAF, with a Ph.D. in Chemistry, who has done extensive research in the field of analytical chemistry;

Dr. Rainer Newberry: Professor in Geochemistry, Mineralogy, and Economic Geology, with a Ph.D. in Economic Geology;

Dr. Bryce Taylor: Doctorate of Dental Surgery, formerly serving in public health with the TCC, now in private practice;

Dr. Joan Braddock: Most recently Dean of the College of Natural Science and Mathematics, with a Master's Degree in Microbiology and a Ph.D. in Oceanography;

Dr. Beth Medford: Board Certified Pediatrician with a background in biochemistry; formerly at Eielson AFB before entering private practice.

Terry Strle, City Mayor

AYES: Roberts, Eberhart, Gatewood, Bratcher, Cleworth, Stiver
NAYS: None
ABSTAIN:
ABSENT:
ADOPTED: February 08, 2010

ATTEST:

Janey Hovenden, CMC, City Clerk

APPROVED AS TO FORM:

Paul J. Ewers, City Attorney

INFANT MORTALITY AND EXPOSURE

Effective interventional approach to control anaemia in pregnant women

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Anaemia in pregnancy and low birth weight babies, a serious public health problem, troubles India and several other nations. This article reports the results of a approach to address the issue. Women up to 20 week pregnancy with haemoglobin (Hb) 9.0 g/dl or less, those with urinary fluoride beyond 1.0 mg/l and not suffering from any other ailments, were selected. Out of the 205 pregnant women attending antenatal clinics (ANCs) during 1st and 2nd trimesters, the sample and control groups were selected through computerized random sampling procedure. Ninety pregnant women formed the sample group and 115 formed the control group. The sample group was introduced to two interventions, viz.: (1) removal of fluoride from ingestion through drinking water, food and other sources, (2) counselling based intake of essential nutrients, viz. calcium, iron, folic acid, vitamins C, E and other antioxidants through dairy products, vegetables and fruits. No intervention was introduced for the control group. Sample and control groups were monitored for urinary fluoride and Hb until delivery during their visits to ANC. Birth weight of the babies were recorded from the labour room register. Results reveal that (1) the urine fluoride levels decreased in 67% and 53% of the pregnant women respectively, who attended ANCs during 1st and 2nd trimester of pregnancy. (2) An increase in Hb upon withdrawal of fluoride followed by nutritional intervention in 73% and 83% respectively has also been recorded. (3) Body mass index (BMI) also enhanced. (4) The percentage of pre-term deliveries was decreased in sample group compared to control. (5) Birth weight of babies enhanced in 80% and 77% in sample group women who attended ANC in 1st and 2nd trimester respectively as opposed to 49% and 47% respectively in the control group. (6) The number of low birth weight babies was reduced to 20% and 23% respectively in sample as opposed to 51% and 53% in control groups.

Keywords: Anaemia, haemoglobin, low birth weight, pregnancy, urine fluoride.

INDIA and many other nations face a serious problem of anaemia in pregnancy, resulting in low birth weight babies. The Government took cognizance of the issue in 1970 (ref. 1). Considering that the diet is deficient in iron requirement for haemoglobin (Hb) biosynthesis, the decision to supplement iron along with folic acid to pregnant women visiting antenatal clinics (ANCs) was implemented throughout the country. Iron (60 mg) and folic acid (500 µg) was administered orally as a tablet for 90 days during the 1st and 2nd trimesters. Based on a review conducted during 1985–86 in 11 states in India, by the Indian Council of Medical Research (ICMR), it was observed that the intervention had not made any difference in the haemoglobin level/anaemia in pregnant women of more than 37 weeks of gestation[2]. This led to changing the strength of the tablet to 100 mg iron and 500 µg folic acid and this has been in vogue since 1992 (ref. 3).

The problem continues to plague the country even as late as 2009. According to UNICEF 2008 Report, the highest percentage of low birth weight babies below 5 years of age, namely 43%, is in India. The prevalence in South Asia is 42%; it is 35% in the least developed countries; 26% in developing countries and 25% in the world[4]. The situation is alarming and requires to be addressed with a relook at the risk factors, other than dietary deficiencies, parasitic infestation, urinary tract infection and malaria. The literature is voluminous. Consequences of iron deficiency anaemia in pregnant women could result in (i) increased maternal mortality and morbidity, (ii) increased foetal morbidity and mortality, (iii) increased risk factor of low birth weight babies resulting in brain and thyroid gland damage which may be irreparable[5–8].

While dealing with iron deficiency anaemia, if rectification is approached through iron and folic acid supplementation besides diet counselling, it is important to address factors enhancing non-haeme iron absorption, viz. vitamin C and low pH achieved through lactic acid production. The inhibitors of non-haeme absorption such as phytates, polyphenols, tannins besides soya protein are to be avoided[9]. While counselling, if the women are expected to practise the interventions, the information should be packaged in a manner that can be put into practice with ease. It has been our observation that any number of IEC (information, education and communication) materials printed and distributed is unlikely to achieve the same results as those that can be achieved by discuss-

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ing and explaining the requirements of dietary changes to the pregnant women with concern and compassion.

Iron cum folic acid supplementation and diet counselling offered by ANCs ought to have corrected iron deficiency anaemia in India; but it has not happened as desired in spite of massive efforts and investments. Breymann[10] is of the view that haemoglobin alone is insufficient to guide management of pregnant women with iron deficiency and anaemia. In Nepal, iron and folic acid supplementation reduced the incidence of low birth weight by 16%. Supplementation of 14 micronutrients including iron, folic acid and zinc reduced low birth weight by 14%, thus confirming no added advantage of multiple micronutrients over iron and folic acid[11]. Other factors need to be investigated, and one such factor is fluoride intake. Fluoride causes serious damage to the gastrointestinal (GI) mucosa by destroying microvilli resulting in non-absorption of nutrients from the diet[12–15]. Fluoride is also known to destroy erythrocytes, thereby contributing to loss of haemoglobin which results in anaemia[16,17].

Fluoride is a toxic chemical and it is a risk factor for thyroid hormone production in children when the exposure to fluoride occurs during intrauterine growth period[18]. Pagin's report in *Scientific American* 'on second thoughts about fluoride' during 2008 is a warning to all concerned as he has revealed the risk of fluoride causing disorders affecting teeth, bone, brain and thyroid gland[19]. As early as 1979, US dairy scientists have reported that thyroxine and triiodothyronine in serum decreased with increasing urinary fluoride in cattle. Cattle affected with fluorosis developed hypothyroidism and anaemia[17]. Thyroid hormone status in married women prior to conception may therefore be required to be assessed.

Keeping in view the information available on fluoride and fluoride toxicity, a protocol was designed with prime objective of controlling anaemia in pregnant women with high fluoride intake who are on routine iron and folic acid supplementation. ANC approach was preferred, as the study could be executed and monitored until delivery.

Material and methods

A general hospital located in the National Capital Territory of Delhi (NCTD), India, where women from the lower strata of society attend ANCs, was identified and necessary clearance to work in the hospital was obtained from the administration and the head of the obstetrics and gynaecology (OBGY) department. The project was launched in 2005. A total of 2055 pregnant women were screened over a period of 2 years and 6 months. Pregnant women who were more than 20 weeks into gestation and those suffering from diabetes, tuberculosis, bleeding during pregnancy, high blood pressure, HIV AIDS, malaria and other medical problems were excluded. Only those women who were anaemic (Hb ≤ 9.0 g/dl) were considered for investigations. These exclusion criteria reduced the target population size to 249.

Computerized random sampling procedure

The study population was grouped into sample and control using computerized random sampling procedure. The sample and control groups were subjected to the following three laboratory tests: (i) Hb test, (ii) Fluoride content in drinking water, and (iii) Fluoride content in urine sample.

Hb content was measured using HemoCue 201+, a digital, portable unit, so that the women can see the results instantaneously. HemoCue is used extensively for field-based studies[20,21].

The fluoride test was conducted in both drinking water and urine samples based on the method of Hall *et al.*[22]. The equipment used is Ion meter model, ION 85 Ion Analyser, Radiometer, Copenhagen.

Information on the following confounding factors was gathered using a specially designed proforma for the dietary regime, economic status, literacy status of the women, employment status, first pregnancy or the women had earlier issues, any miscarriage/intra-uterine death, history of previous or present ailment; and consumption of iron and folic acid tablets provided by the hospital.

Height and weight measurements were made. Blood pressure measurements from hospital records were transferred to the study proforma. Wherever any of these were missing, measurements were done by the investigating team. The information thus collected was used for analysis.

Sample population: All pregnant women in the sample population were anaemic (Hb ≤ 9.0 g/dl, though anaemia is denoted when Hb is <11.0 g/dl, anaemic women with Hb from 9.0–5.0 g/dl were chosen for the study), with urinary fluoride more than 1.0 mg/l during the first visit to the ANC. Only those with Hb level up to 5.0 g/dl were selected. The sample population was subjected to two intervention procedures.

(i) *Intervention procedure 1: to avoid consumption of fluoride containing water and food items.* Counselling was provided to avoid consumption of fluoride containing food, water and other substances for arrest of injury to cells/tissues and for enabling regeneration of the damaged GI mucosa.

Upon reviewing the data on fluoride content in drinking water and if fluoride was beyond normal limits (more than 1.1 mg/l, the national guideline for fluoride in drinking water is 1.0 mg/l, as the upper limit, less the better), the subjects were shifted to an existing safe source of water in their neighbourhood for collecting water for drinking and cooking purposes. In the event that drinking water fluoride was below 1.0 mg/l and therefore safe but urinary fluoride was high (reference range 0.1–1.0 mg/l), the source(s) of fluoride was traced through retrieving

information on diet and dietary habits to find the food items consumed and the sources known to contain high fluoride.

Consumption of all items including food enriched with fluoride was withdrawn. Items leading to high fluoride intake are (i) use of black rock salt (CaF_2) with 157 ppm fluoride (analytical data 2007 – unpublished) in cooking – black rock salt is toxic and harmful to health; (ii) all items including Indian street food (junk food) enriched with black rock salt to enhance the aroma and tangy taste; (iii) black tea without milk, *churans*, and toothpaste with high fluoride and (4) salted snacks and spices smeared with rock salt.

Counselling to avoid drinking water and food containing fluoride is an intervention that the pregnant women in sample group were introduced to for rectifying the damage caused to the GI mucosa[23]. The mucosa is known to regenerate within a short interval of 10–15 days upon withdrawal of fluoride and absorption of nutrients including orally administered iron and folic acid tablets would commence thereafter.

(ii) *Intervention procedure 2: promotion of intake of essential nutrients through diet.* Diet counselling was given for promoting adequate intake of essential nutrients, viz. calcium, iron, folic acid, vitamins C, E and other antioxidants for repair and maintenance of the damaged cells and tissues as well as for enhancing rise in Hb.

The focus of counselling was on the importance of consuming a nutritive diet and the need to acquire essential nutrients through dairy products, vegetables and fruits. Antioxidant intake is essential since fluorine, being a powerful oxidizing agent, produces oxygen free radicals and these need to be eliminated from the system. Vegetables and fruits rich in antioxidants act as scavengers for eliminating free radicals and restoring the system to normalcy. In addition to counselling, a little pictorial booklet in local language on dietary details was also given to pregnant women.

The pregnant women followed the advice as it involved simple recipes, affordable by the poor, rural and urban population. Besides, they found the information easy to practice. It was also indicated that the information imparted was not only for the benefit of one member, and that other members of the family, could consume the same food. The counselling was for improving the health of all members of the household and not 'pregnancy specific'. Such advice is found more acceptable to the women as food is cooked for the entire family and not particularly for one individual.

Distribution of IEC materials followed by discussion with concern and compassion had an impact on the sample group.

Control population

The control population is the same in all respects as the sample population with Hb ≤ 9.0 g/dl and urinary fluoride > 1.0 mg/l. The only difference is that they were not counselled for intervention procedures 1 and 2.

Monitoring the subjects: The sample and control population were monitored until delivery by testing for urinary fluoride and haemoglobin during every visit to the ANC. The pregnant women were informed about their Hb levels. A target number of Hb to be attained, prior to the next visit to the ANC was also indicated. It turned out that a few women came only once or twice due to personal reasons while the majority visited the ANC 3–7 times prior to delivery. Those who came only once or twice, were not included for reporting the data.

Some women did not come to the same hospital for delivery; but went to different hospitals closer to their homes. The investigating team was alert to this fact, since every woman was followed up through telephone calls and information about the mother and baby was being monitored. The information collected and recorded in the labour room register was checked and all relevant information transferred to the project proforma.

Results and discussion

Various sources of fluoride starting from drinking water (naturally contaminated with fluoride), food, food products and beverages like black tea (without milk) were identified and withdrawn. The impact of such an approach along with promotion of nutritive diet, and the outcome in 90 sample group women who attended ANC during 1st and 2nd trimester of pregnancy are reported in Tables 1 and 2. As there are reports to suggest that iron deficiency during 1st trimester results in significant reduction in foetal growth[24,25] and not beyond, the focus was to evaluate the results trimester-wise.

Withdrawal of fluoride source(s) possibly resulted in the expected regeneration of GI mucosa and microrvilli, and this in turn enhanced the absorption of nutrients as evidenced by the reduction in urinary fluoride followed by rise in Hb levels.

Those women who either withdrew or reduced fluoride intake irrespective of whether they attended the ANC during the 1st or 2nd trimester of pregnancy not only showed reduced levels of urinary fluoride but also benefitted in terms of raising their Hb levels. This is amply evident from the data reported in Tables 1 and 2. In sample group, the urinary fluoride was reduced from 2.082 ± 1.058 to 1.628 ± 1.631 in women who attended ANC during 1st trimester of pregnancy, showing a concomitant rise in Hb from 8.2 ± 0.9 to 10.8 ± 2.0. In a similar manner, when urinary fluoride is reduced from 1.939 ± 1.122 to 1.441 ± 0.894, the Hb enhanced from 8.4 ± 0.8 to 10.1 ± 1.8 in the women who attended ANC first time during 2nd trimester. Reduction in urinary fluoride is recorded in 20 out of the 30 women of the sample

Table 1. Urine fluoride, haemoglobin of pregnant women of sample and control groups during the initial and prior to delivery in those who first attended ANC during the 1st trimester of pregnancy (10–15 week) and birth weight of babies

	Urinary fluoride level (UFL) (mg/l)			Haemoglobin (Hb) (g/dl)			Birth weight of babies (kg)		
		UFL initial	UFL prior to delivery		Hb initial	Hb prior to delivery		Low birth wt (< 2.5 kg)	Normal birth wt (≥ 2.5 kg)
Sample $n = 30$	Mean ± SD	2.082 ± 1.058	*1.628 ± 1.631	Mean ± SD	8.2 ± 0.9	**10.8 ± 2.0	Mean ± SD	2.21 ± 0.17	***2.94 ± 0.27
	Range	1.082–6.256	0.254–7.749	Range	5.7–9.0	5.4–13.3	Range	1.95–2.45	2.5–3.5
	Reduction in UFL in 20/30 = 67%			Rise in Hb in 22/30 = 73%			Normal birth weight born in 24/30 = 80%		
							Low birth weight born in 6/30 = 20%		
Control $n = 37$	Mean ± SD	1.617 ± 1.158	*1.702 ± 1.709	Mean ± SD	8.3 ± 0.9	**9.3 ± 1.5	Mean ± SD	2.00 ± 0.58	***2.76 ± 0.27
	Range	0.403–4.094	0.263–8.768	Range	5.6–9.0	6.3–12.0	Range	1.3–2.43	2.5–3.3
	Reduction in UFL in 18/37 = 49%			Rise in Hb in 22/37 = 59%			Normal baby weight 18/37 = 49%		
							Low birth weight 19/37 = 51%		

*$P > 0.01$ (nonsignificant); **$P < 0.001$; ***$P < 0.1$; SD, Standard deviation.

Table 2. Urine fluoride, haemoglobin of pregnant women of sample and control groups during the initial and prior to delivery in those who first attended ANC during the 2nd trimester (16–20 week) and birth weight of babies

	Urinary fluoride level (UFL) (mg/l)			Haemoglobin (Hb) (g/dl)			Birth weight of babies (kg)		
		UFL initial	UFL prior to delivery		Hb initial	Hb prior to delivery		Low birth wt (< 2.5 kg)	Normal birth wt (≥ 2.5 kg)
Sample $n = 60$	Mean ± SD	1.939 ± 1.122	*1.441 ± 0.894	Mean ± SD	8.4 ± 0.8	**10.1 ± 1.8	Mean ± SD	2.24 ± 0.19	***2.94 ± 0.32
	Range	0.130–5.464	0.549–5.446	Range	6.3–9.0	5.6–13.2	Range	1.87–2.48	2.5–3.89
	Reduction in UFL in 32/60 = 53%			Rise in Hb in 50/60 = 83%			Normal birth weight 46/60 = 77%		
							Low birth weight 14/60 = 23%		
Control $n = 78$	Mean ± SD	1.364 ± 1.038	*1.723 ± 0.986	Mean ± SD	8.0 ± 1.2	**9.1 ± 1.6	Mean ± SD	2.01 ± 0.64	***2.72 ± 0.21
	Range	0.231–6.134	0.387–4.778	Range	5.1–9.0	5.1–12.2	Range	1.25–2.48	2.5–3.3
	Reduction in UFL in 29/78 = 37%			Rise in Hb in 42/78 = 54%			Normal birth weight in 37/78 = 47%		
							Low birth weight in 41/78 = 53%		

*$P > 0.01$; **$P < 0.001$; ***$P < 0.0001$; SD, Standard deviation.

group of trimester 1, and 32 out of 60 women of trimester 2, which is about 67% and 53% respectively. The rise in Hb is recorded in 22 out of the 30 women which is about 73% and 50 out of the 60 women which is about 83%. This is a significant change arising as a result of withdrawal or reduction in fluoride ingestion followed by a dietary improvement by the pregnant women.

The women in the control group (1st trimester of pregnancy) on the contrary reveal a rise in urinary fluoride instead of reduction from 1.617 ± 1.158 to 1.702 ± 1.709. The Hb has been minimally increased, i.e. 8.3 ± 0.9 to 9.3 ± 1.5. In a similar manner, for women in 2nd trimester of pregnancy, the urinary fluoride was enhanced from 1.364 ± 1.038 to 1.723 ± 0.986; the Hb level had also minimally increased from 8.0 ± 1.2 to 9.1 ± 1.6.

In the control, urinary fluoride reduction is recorded in 49%, i.e. 18 out of the 37 women of 1st trimester. This shows 51% women showed rise or no change in urinary fluoride. Similarly in women of 2nd trimester, 37% showed reduction in urinary fluoride level (UFL), i.e. reduction is recorded in 29 out of the 78 women investigated during 2nd trimester. This again showed 63% women revealed rise/no change in urinary fluoride.

In the control group, rise in Hb is observed in 22 out of the 37 women of the 1st trimester, i.e. 59%; and Hb rise is in 42 out of the 78 women of 2nd trimester which is only 54% of women who participated in the study.

The next obvious issue probed into is whether the rise in Hb in pregnant women has in any manner affected the birth weight of the babies. In the 30 pregnant women of trimester 1, the initial Hb range being 5.7–9.0 g/dl, the practice of interventions led to rise in Hb in the range 5.4–13.3 g/dl prior to delivery. The rise in Hb is recorded in 22 out of the 30 women investigated, which works out to about 73% of the study group. How many among the 30, gave birth to normal birth weight babies and how many delivered low birth weight babies?

Table 1 shows that 24 out of the 30 women delivered normal birth weight babies (i.e. 80%), whose birth weight ranged from 2.5 to 3.5 kg (mean 2.94 ± 0.27). Among the sample group, 6 women out of the 30 (i.e. 20%) delivered low birth weight babies; birth weight range was from 1.95 to 2.45 kg (mean 2.21 ± 0.17).

Reviewing the outcome of the interventions in sample group ($n = 60$) who attended ANC during 2nd trimester, the Hb range was initially 6.3–9.0 g/dl (mean 8.4 ± 0.8);

after practice of interventions, it was in the range of 5.6–13.2 g/dl (mean 10.1 ± 1.8). The rise in Hb is recorded in 50 out of the 60 women investigated which is to the tune of 83%. The rise in Hb in 83% pregnant women and its impact on the birth weight of the babies is reported in Table 2. It is evident that 46 out of the 60 women delivered normal birth weight babies, i.e. 77%. The normal birth weight of babies ranged from 2.5 to 3.89 kg (mean 2.94 ± 0.32). Among the 60 women, 14 delivered low birth weight babies (i.e. 23%). The weight of the babies ranged from 1.87 to 2.48 kg (mean 2.24 ± 0.19).

The impact of interventions among the pregnant women who attended the ANC first either during 1st or 2nd trimester has been manifested in terms of rise in Hb, resulting in birth of normal birth weight babies to the extent of 80% and 77% respectively. The number of low birth weight babies (< 2.5 kg) has been reduced to 20% and 23% respectively. This is considered highly significant (P value < 0.01 in 1st trimester; P value < 0.0001 in 2nd trimester, Tables 1 and 2).

In the women of the control group of 1st trimester, the initial Hb range recorded is 5.6–9.0 g/dl (mean 8.3 ± 0.9) and prior to delivery the changes recorded in Hb is 6.3–12.0 g/dl (mean 9.3 ± 1.5). The rise in Hb was observed in 22 out of the 37 which is about 59%. Similarly, among the control group women who attended the ANC during the 2nd trimester, the Hb range during the initial phase was 5.1–9.0 g/dl (mean 8.0 ± 1.2) and prior to delivery, the range was 5.1–12.2 (mean 9.1 ± 1.6). Among the 37 women of the 1st trimester, 18 delivered normal birth weight babies (i.e. 49%), whereas among the 78 control group women of 2nd trimester, 37 delivered normal birth weight babies (i.e. 47%). In the control group of trimesters 1 and 2, 51% and 53% babies born respectively are of low birth weight; birth weights ranging from 1.3 to 2.43 kg (mean 2.00 ± 0.58) and 1.25 to 2.48 (mean 2.01 ± 0.64) (Tables 1 and 2).

Tables 1 and 2 provide evidence that fluoride ingestion arrests the absorption of nutrients including orally administered iron and folic acid. Therefore, withdrawal of fluoride from ingestion does provide beneficial results in controlling anaemia and improving the birth weight of babies.

The interventions have also been of advantage to the women in terms of increasing body mass index (BMI)[26] and reducing pre-term deliveries[25]. These results are reported in Tables 3 and 4.

The data reveals that among the 90 women in the sample group, 22 had BMI at the initial stages < 18.5 (range 14.4–18.4), suggesting prevalence of underweight/undernutrition. However, prior to delivery the number in this category was reduced to 2. This is an impact of interventions. Initially there were 63 women, with BMI 18.5–24.9 (i.e. normal range) but prior to delivery improvement was observed in 67 women, yet again showing impact of interventions. In the BMI category, 25.0–29.9 is normally considered overweight but in pregnancy the rise in weight is desirable. Initially there were 5 women in the category; this number was increased to 21 prior to delivery. In the category of BMI ≥ 30.0 which is obese, there were none, which is also a healthy sign.

The impact of interventions on gestation period at delivery is an added advantage to the sample group women. Table 4 shows that pre-term delivery, i.e. < 34 weeks of gestation, was recorded in 2 out of 90 (i.e. 2%); whereas it is 9 out of 115 in the control (i.e. 8%); four times more in the control group who were not introduced to interventions. Delivery between 34 and 37 weeks of gestation was 30% and 42% respectively, in sample and control groups. Delivery at gestation period > 37 weeks was 68% in sample and 50% in control groups. There were two stillbirths in control and none in sample group. The overall benefits accrued through the intervention procedures have undoubtedly been extraordinary.

Statistical analysis

The overall interventional impact was assessed through different statistical parameters. Very significant reduction in urinary fluoride level (UFL) along with substantial gains in the Hb level and BMI are observed in the sample group subjected to interventions. The quantified gains are

Table 3. Impact of intervention(s) on body mass index (BMI)

	Body mass index							
	Initial				Prior to delivery			
BMI categories	< 18.5	18.5–24.9	25.0–29.9	≥ 30.0	<18.5	18.5–24.9	25.0–29.9	≥ 30.0
Sample group n = 90	n = 22	n = 63	n = 5	n = nil	n = 2	n = 67	n = 21	n = nil
BMI range	14.4–18.4	18.5–24.7	25.2–26.0	–	18.2–18.4	18.8–24.8	25.3–28.9	–
Mean ± SD	17.2 ± 1.3	20.8 ± 1.8	25.7 ± 0.4	–	18.3 ± 0.1	22.3 ± 1.6	27.1 ± 1.2	–
Control group n = 115	n = 36	n = 73	n = 6	n = nil	n = 4	n = 88	n = 20	n = 3
BMI range	15.6–18.3	18.5–24.5	25.2–29.0	–	17.3–18.2	18.5–24.9	25.0–28.2	30.1
Mean ± SD	17.2 ± 0.9	20.9 ± 1.6	27.3 ± 1.7	–	17.8 ± 0.4	22.1 ± 1.7	26.2 ± 0.9	30.1 ± 0.0

SD = Standard deviation.

Table 4. Gestation period at delivery

	Gestation period					
	< 34 Weeks (pre-term delivery)		34–37 Weeks (term delivery)		> 37 Weeks (full-term delivery)	
Sample $n = 90$		$n = 2$		$n = 27$		$n = 61$
	Mean ± SD	33.0 ± 0.0	Mean ± SD	36 ± 1.1	Mean ± SD	39.3 ± 1.1
	Range (weeks-days)	33.0–33.0	Range (weeks-days)	34.0–37.0	Range (weeks-days)	38.0–42.0
	< 34 weeks (pre-term delivery) in 2/90 = 2%		34–37 weeks (term delivery) in 27/90 = 30%		> 37 weeks (full-term delivery) in 61/90 = 68%	
Control $n = 115$		$n = 9$*		$n = 48$**		$n = 58$***
	Mean ± SD	31.2 ± 4.6	Mean ± SD	36 ± 1.1	Mean ± SD	39.2 ± 1.0
	Range (weeks-days)	31.0–33.0	Range (weeks-days)	34.0–37.0	Range (weeks-days)	38.0–41.0
	< 34 weeks (pre-term delivery) in 9/115 = 8%		34–37 weeks (term delivery) in 48/115 = 42%		> 37 weeks (full-term delivery) in 58/115 = 50%	

*One spontaneous abortion at 19 weeks. ***Two pre-term stillbirth at 30th and 31st weeks. SD, Standard deviation.

far greater in this group as compared to the control group that was not subjected to interventions. Additionally, the sample groups had higher average baby weight as compared to the control group. The proportion of babies born with normal weight was also higher in the sample group.

Quantifications of all these parameters suggest that interventions for control of anaemia in pregnant women have been highly impactful. The benefit accrued to the families is highly significant and this was possible due to elimination of fluoride and promotion of a nutritive diet rich in essential nutrients.

The results have been as critically evaluated as possible, with a view to identify the major detrimental and confounding factors statistically. The goal is to understand the merits of the results better so that such a process can be replicated/scaled-up within the country and elsewhere in the world.

We conjectured the following two hypotheses and validated them statistically: (1) Pregnant women with high urinary fluoride have low Hb levels. (2) Reduction in urinary fluoride of pregnant women results in increase in Hb.

The analysis indicated a positive impact of the withdrawal of urinary fluoride on Hb levels in pregnant women. The analysis also reveals that our hypothesis (2) is statistically valid. It signifies that interventions have played a key role in increasing Hb levels with lowering of urinary fluoride in the pregnant women, an approach hitherto unknown.

Evaluation of data based on hypotheses 1 and 2

At the overall level, it is observed that urinary fluorides were reduced to normal levels in 35% sample group cases as against 26% in the control group (Table 5). It should be pointed out that in statistical evaluation, the data analysis is based on the total number in sample and control groups; there is no bifurcation into trimesters 1 and 2.

This observation alone is misleading if conclusions are made regarding an impact of the intervention programme on account of the following facts.

- UFLs were observed to have fluctuated drastically in the intervening periods due to non-adherence to dietary guidelines.
- In table, we observe only the difference between UFLs during the first and the last patient visits.
- Changes in Hb and BMI levels are equally critical for any conclusions to be made about the interventions.
- It will also be important to find the factors detrimental to the reduction of UFL and Hb through interventions.

It is, therefore, important to assess the impact of changes in UFLs in conjunction with changes in Hb and BMI levels. We have made an attempt to set up a few relevant hypotheses and justify them using the observed data.

Hypothesis 1

Pregnant women with high urinary fluoride have low Hb levels. It is observed (Table 6) that Hb levels improved to normalcy in 71% of the sample cases as against 37% in the control group. Clearly some strong impact of interventions to control urinary fluoride and thereby Hb levels is visible. Further, studying UFLs in those cases where Hb levels were low would provide a better understanding of the impact of interventions.

Percentage of pregnant women with high urinary fluoride and low Hb levels has decreased from 99% at their first visit to 85% prior to delivery in the sample group (Table 7). It means a substantial 14% of these women have improved their Hb levels. On the other hand, comparable women in the control group indicated an equivalent rise in their percentage (15%; from 54% at first visit to 69% prior to delivery). This clearly indicates a very

Table 5. Percentage distribution of pregnant women by urinary fluoride levels (UFLs)*

	Sample group (90)			Control group (115)		
	High UFL (%)	Normal UFL (%)	Total (%)	High UFL (%)	Normal UFL (%)	Total (%)
On first visit	99	1	100	54	46	100
Prior to delivery	64	36	100	28	72	100
Percentage change	–35	+35		–26	+26	

UFL (normal) means urinary fluoride less than 1.0 mg/l on the 1st visit. In Materials and methods, all pregnant women have UFL more than 1.0 mg/l.

Table 6. Pregnant women by Hb levels (% distribution)

	Sample group (90)			Control group (115)		
	Low Hb (%)	Normal Hb (%)	Total (%)	Low Hb (%)	Normal Hb (%)	Total (%)
On first visit	100	0	100	100	0	100
Prior to delivery	29	71	100	63	37	100
Percentage change	–71	+71		–37	+37	

Table 7. Pregnant women by UFLs with low Hb (% distribution)

	Sample group (26/90)			Control group (72/115)		
	High UFL (%)	Normal UFL (%)	Total (%)	High UFL (%)	Normal UFL (%)	Total (%)
On first visit	99	1	100	54	46	100
Prior to delivery	85	15	100	69	31	100
Percentage change	–14	+14		+15	–15	

positive correlation between withdrawal of urinary fluorides and the rise in Hb levels of pregnant women.

Hypothesis 2

Interventions do not result in significant increases in Hb levels with reduction in UFLs of pregnant women. Interventions, either direct (withdrawal of drinking water source and food contaminated with fluorides) or indirect (advice on proper dietary methods) are intended to be preventive in nature. A simple correlation analysis (Table 8) indicates that interventions have acted as huge stimuli for improved Hb levels in sample group of pregnant women as compared to the control group. Correlation between UFLs and Hb levels increases (in absolute terms) from –0.14 to –0.39 for the sample group between first and last visits of the pregnant women. Starting from a negative relationship (corr = –0.14) between the two factors at the first visits, it strengthens by a margin of 25 points in the negative direction. A similar differential for the control group is only 17 points. This differential is highly significant and nullifies the hypothesis of 'no impact of intervention'. It signifies that interventions have played a key role in increasing Hb levels with lowering of UFLs (Table 9; Figures 1 and 2) in the pregnant women.

Confounding factors detrimental to improvement in Hb levels

It is observed that despite subjecting the pregnant women to interventions, in 29% (26 cases) of the sample group, Hb levels remained below normal (Table 6). Therefore, it is pertinent to study the factors that caused this adversity. Amongst the prominent factors that we could consider and for which data was collected were – family income, subject's age, subject's literacy, subject's occupation, husband's occupation, subject's locality type, subject's family size, etc. For our analysis, as required and appropriate, data had to be either regrouped (e.g. create age groups instead of age in years) or transformed (e.g. compute per capita income instead of absolute family income).

Data transformation

Understanding data transformations and any assumptions is equally important to appreciate the analytical results. While the important ones are listed here, others are non-critical.

HB_LowLow: A dummy variable for low Hb level at the first contact remaining low at the last contact. Vari-

Table 8. Correlation analysis

	Sample group	Control group
Correlation between UFL and Hb levels – first visit	–0.14	0.00
Correlation between UFL and Hb levels – prior to delivery	–0.39	–0.17
Average number of visits	4.13	3.55
Number of valid observations	90	115

Table 9. Summary of interventional impact

	With intervention			Without intervention		
	At 1st visit	Prior to delivery	Percentage change (%)	At 1st visit	Prior to delivery	Percentage change (%)
Median* Hb level	8.65	10.60	22.54	8.60	9.40	9.30
Median UFL	1.72	1.13	–34.22	1.14	1.34	17.65
Median BMI level	19.80	23.28	17.58	20.15	22.65	12.41
Average intervention period (days)		120.0			111.0	
Average baby weight at birth (kg)		2.78			2.44	
Percentage babies born with ≥ 2.5 kg		78%			48%	
Percentage patients with decline in UFLs		73%			40%	
Number of valid observations		90			115	

*Median is a point above/below which 50% of the cases lie.

able HB_LowLow is equal to 1 if low Hb remains low; and is equal to 0 otherwise.

Occupation: Subject's occupation is grouped as housewife = 1, unskilled worker = 2, other workers (all cases unclassified as 1 or 2) = 3. This grouping incorporates the level of occupation, 1 being low and 3 being high. Wherever subject's occupation was not mentioned, it was assumed to be the same as that of husband's occupation. It is expected that occupation (thus defined) would have a negative relationship with HB_LowLow.

Age: Subject's age is grouped as 1 if age is less than 20 years, 2 if age ranges between 20 and 24 years, 3 if age ranges between 25 and 29 years and 4 if age is 30 years or more. We expect Hb to remain low in lower age groups of subjects.

Locality: Subject's locality is grouped as 1 = government colony, 2 = village/developing colony and 3 = slum. It would be expected that subjects from slum areas would be more prone to non-improvements in their Hb levels.

Literacy: Subject's and husband's literacy is grouped as illiterate = 1, literate = 2, matric = 3, graduate and above = 4. It is expected that low Hb levels could remain low in literacy groups 1 and 2.

Identifying factors

Data analysis is aimed at finding the major factors that are detrimental to rise in subject's Hb level. We use logistic regression approach for this purpose. It may be noted that at this stage we are not really keen to know the exact magnitudes of impact of various factors. Therefore, any inferences from analytical results need to be made in light of the objective.

Table 10 presents a snapshot of logistic regression analyses carried out to identify such key factors that are detrimental to growth in subject's Hb level. Prominently, per capita income (which is defined as total family income divided by family size – that takes into account both absolute incomes as well as the family size) and number of times the subject visits the ANC in the hospital for medical and interventional advice are the most significant factors that determine the improvements in Hb levels. On the other hand, most of the demographic characteristics – subject's age, education level (either of the subject or that of the husband), their occupations and the type of locality they lived in – are observed to be nonsignificant factors. Income in absolute terms is also a nonsignificant factor towards lower Hb levels. Very interestingly, all the factors show correct direction of impact (signs of coefficients, see Table 10, Logit 1).

It is logical to ask if application of intervention itself is a factor for improvements in Hb levels. This is tested by introduction of a dummy variable for application of intervention (Int_applied = 1 if subject belongs to the sample group, Int_applied = 0 if subject belongs to the control group). It is observed that application of intervention is a critical factor for improvements in Hb levels. This also strongly supports our results in earlier sections.

Evaluating the background information retrieved from the pregnant women belonging to the sample and control groups is informative in the sense that in the sample group, members of a household are from a minimum of 2 to a maximum of 12. In the control group the maximum

Table 10. Summary of logistic regression results – dependent variable Y = HB_LowLow = 1

Factor	Logit 1		Logit 2	
	Direction of impact	Significance level	Direction of impact	Significance level
Per capita income (PCI)	Negative	5%	Negative	7%
Subject's age (age_gr)	Negative	Nonsignificant	Negative	Nonsignificant
Subject's occupation (Occ_gr)	Negative	Nonsignificant	Negative	Nonsignificant
Number of contacts (No_visits)	Negative	1%	Negative	1%
Subject's locality type (loc_type_gr)	Negative	Nonsignificant	Negative	Nonsignificant
Intervention applied or not (Int_applied = 1 if yes, = 0 if no)	–	–	Negative	1%

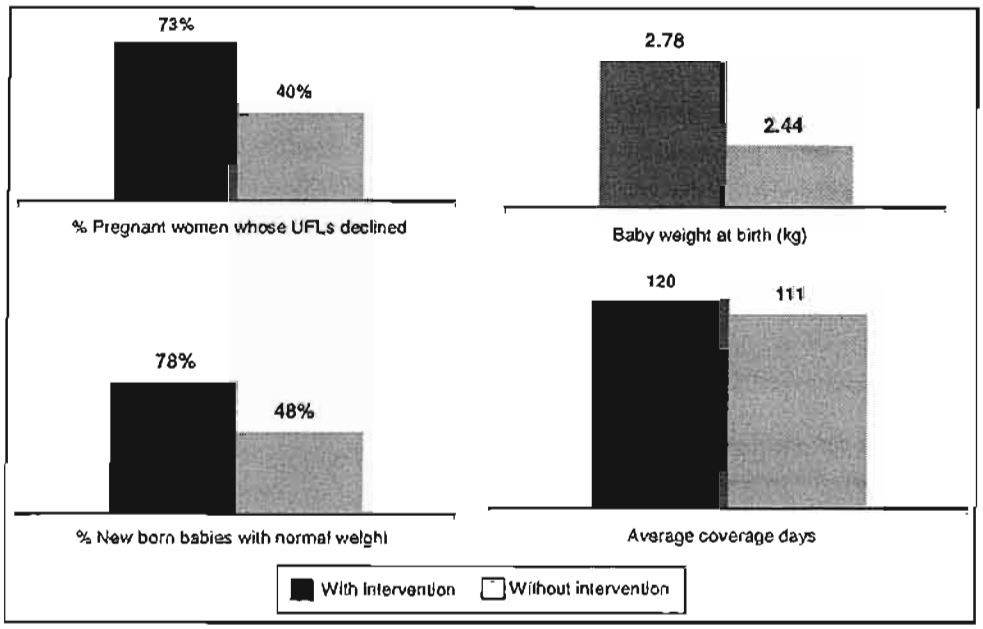

Figure 1. Impact of intervention.

number was 7 and not beyond. The income per day varies from a minimum of Rs 83 (US$ 2.0) to a maximum of Rs 500/day (US$ 12.5) in the sample group. In control group, the income was a minimum of Rs 66 (US$ 11.5) to a maximum of Rs 300/day (US$ 7.5). The per capita income in the sample and control groups is a meagre amount. It ought to be noted that the pregnant women never came alone but were accompanied by family members. To and fro transport charges for two at frequent intervals may be beyond the means of the family. It would appear therefore that the cash availability was a deterrent for frequent hospital visits. This would also suggest that there may be constraints in the purchasing power for food as well and is likely to have an adverse impact.

This analysis also brings out the fact that application of intervention and number of visits by the subject for regular antenatal follow-up are interdependent. This means that, even if a particular subject is put under an intervention programme, the subject may or may not visit for regular antenatal follow-up. However, for success/effectiveness of interventions, subject's number of visits (contacts) for antenatal follow-up should be highly associated with application of intervention. And, we should be expecting this to be so. This clearly indicates that there is a missing link here.

This brings up a question – 'Are there any specific factors that govern how many times the subject is likely to be present for regular antenatal follow-ups?' This is tested through a regression analysis. Table 11 depicts a snapshot of factors influencing number of contacts/visits.

It is observed that there is a significant influence of application of intervention on the subject and the locality type where the subject comes from on the number of subject's visits.

Subject's locality type is observed to be a highly significant factor determining number of contacts by the subject. The negative sign indicates that the number of

Table 11. Summary of regression analysis (dependent variable Y = No_visits)

Factor	Direction of impact	Significance level
Intervention applied or not (Int_applied = 1 if yes, = 0 if no)	Positive	5%
Subject's age (age_gr)	Positive	Nonsignificant
Subject's occupation (Occ_gr)	Positive	Nonsignificant
Per capita income (PCI)	Positive	Nonsignificant
Subject's locality type (loc_type_gr)	Negative	5%

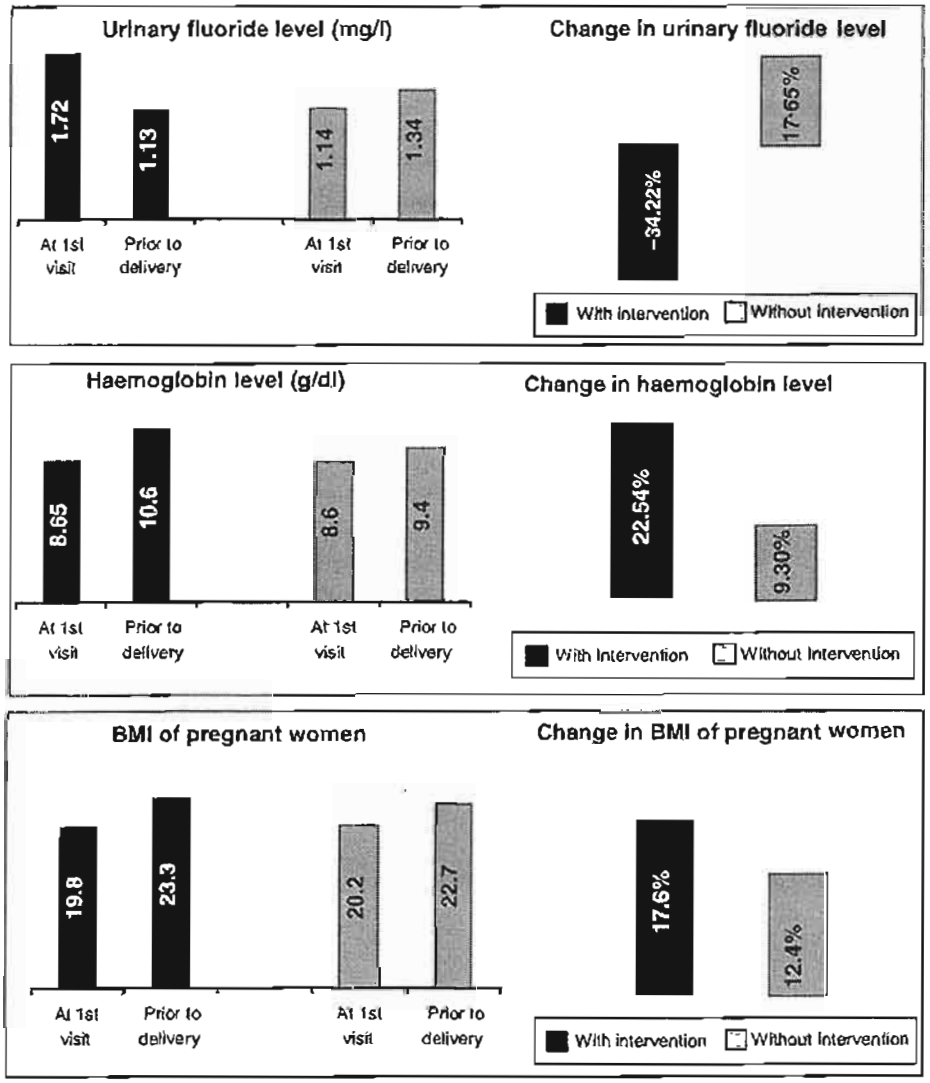

Figure 2. Impact of intervention on UF, Hb and BMI.

contacts declines from government locality to a slum area. We further performed a similar analysis only for subjects from the slum areas and, apparently, none of the factors explains any specific influence on the slum dweller's behaviour. There appears to be more for further investigation.

The first factor (application of intervention) throws up a bit of a confusing result to which we do not have a concrete answer at the moment. We could dig deeper into this relationship in a further analysis of our subsequent intervention programmes.

Though fluoridation of a variety of products is still in vogue in many nations, it is difficult to overlook the high percentage of pregnant women exposed to fluoride ingestion and being anaemic in developing countries[24]. A

simple procedure of assessing fluoride in urine and Hb levels in women is adequate to introduce interventions for controlling anaemia in such high percentage of pregnant women. It is evident from the data reported in this communication that maternal and child under-nutrition and anaemia is not necessarily due to insufficient food intake but because of the derangement of nutrient absorption due to damage caused to GI, mucosa by ingestion of undesirable chemical substance, viz. fluoride through food, water and other sources. These aspects have so far been unexplored, and this is the first time such a possibility is investigated and results reported. The findings of this approach in the context of anaemia in pregnancy provide a new path for reducing the burden of disabled and mentally challenged children[25,27] by reducing percentage of low birth weight babies.

In a small percentage in sample group, though the urine fluoride was reduced, Hb did not rise. Low per capita income and not consuming adequate nutritive diet and possibly other reasons such as low thyroid hormone for non-production of adequate RBCs need to be explored.

In conclusion, a novel and effective intervention approach therefore has scope for reducing anaemia in pregnancy and improve birth weight of babies. Fluoride toxicity, as a risk factor was never considered even in the highly endemic regions for fluoride and fluorosis in India and around the globe. This is the first report dealing with fluoride, pregnancy, anaemia, low birth weight babies and the linkages to act upon for the benefit of maternal and reproductive child health programmes.

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Received 17 March 2009; revised accepted 22 Ma

study linking Fluoride consumption by pregnant mothers to low birth weight babies. See summary to right

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NOVEMBER 20, 2009 11:00AM

Study Links Fluoride to Premature Births

New York -- November 2009 – State University of New York (SUNY) researchers found more premature births in fluoridated than non-fluoridated upstate New York communities, according to a presentation made at the American Public Health Association's annual meeting on November 9, 2009 in Philadelphia. (1)

Fluoridation is the addition of fluoride chemicals into public water supplies ostensibly to prevent tooth decay. Many groups oppose fluoridation because of its scientifically-documented health risks. (2)

Human pregnancy lasts about 40 weeks or just more than 9 months. A baby born before 37 weeks of pregnancy is considered a preterm (or premature) birth. About 12 percent of US pregnancies are preterm and this is one of the top causes of infant death in the US, according to the US National Institutes of Health. (3)

The SUNY researchers used 1993-2002 data from the NY Statewide Planning and Research Cooperative System (SPARCS), which collects patient characteristics, diagnoses, treatments, services and charges for every hospital discharge, ambulatory surgery patient and emergency department admission in New York State. They recorded fluoridation residence status (under or over 1 milligram fluoride per Liter of water) and adjusted for age, race/ethnicity, neighborhood poverty level, hypertension and diabetes.

"Domestic water fluoridation was associated with an increased risk of PTB [preterm birth]. This relationship was most pronounced among women in the lowest SES [socio-economic-status] groups (>10% poverty) and those of non-white racial origin," write Rachel Hart, et al. Department of Epidemiology & Biostatistics, SUNY School of Public Health.

Previous published research by others has shown that fluoride can interfere with the reproductive system. (4)

"It would be wise to follow the lead of the 7,000 Environmental Protection Agency scientists and public health professionals (5) who asked Congress to place a moratorium on fluoridation until definitive studies are conducted to prove fluoridation is safe for every human consuming it," says attorney Paul Beeber, President, New York State Coalition Opposed to Fluoridation, Inc. "Clearly fluoridation is not safe for everyone," says Beeber.

At the request of the US Environmental Protection Agency (EPA), a National Research Council (NRC) panel of experts reviewed current fluoride toxicology. In 2006 they concluded that the maximum amount of fluoride allowed in drinking water is too high to be protective of health. At least three NRC panel members believe water fluoride levels should be as close to zero as possible. The EPA has yet to perform a fluoride risk assessment based on the NRC's findings leaving millions of Americans at risk of fluoride's adverse health effects.

According to Dr. Bill Hirzy, Chair of American University's Chemistry Department and former EPA scientist from 1981 to 2008, the EPA fears "setting a maximum contaminant level goal of zero because that would mean the EPA is going to be responsible for the end of the water fluoridation program. EPA knows that there will be enormous political flak for doing that." (6)

References:

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http://apha.confex.com/apha/137am/webprogram/Paper197468.html

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Kerry Lauerman

Joan Walsh
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UPDATES

UPDATED: Better, faster, stronger
posted by: Kerry Lauerman


Me on Open Salon
posted by: Joan Walsh

By 2012, Water Fluoridation Will Be Banned
posted by: uprightnetizen

(2) Fluoride Action Network – Health Effects Database
http://www.fluoridealert.org/health/sitemap.html

(3) National Institutes of Health, "Preterm Labor and Birth,"
http://www.nichd.nih.gov/health/topics/Preterm_Labor_and_Birth.cfm

(4) Fluoride Action Network - HEALTH EFFECTS: Fluoride & the Reproductive System http://www.fluoridealert.org/health/repro/index.html

(5) Why EPA Headquarters Union of Scientists Opposes Fluoridation
http://nteu280.org/Issues/Fluoride/NTEU280-Fluoride.htm

(6) The Eagle, "Hirzy: EPA drags feet on fluoride." by Howie Perlman, October 28, 2009
http://www.theeagleonline.com/news/story/hirzy-epa-drags-feet-on-flouride

AUTHOR TAGS: tooth decay, teeth, babies, infants, birth, preterm, fluoridation, premature, fluoride

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Study Links Fluoride To Pre-term Birth And Anemia In Pregnancy

03 Sep 2010 Click to Print

Fluoride avoidance reduced anemia in pregnant women, decreased pre-term births and enhanced babies' birth-weight, concludes leading fluoride expert, AK Susheela and colleagues, in a study published in *Current Science* (May 2010).

Susheela's team explains that anemia in pregnancy, which can lead to maternal and infant mortality, continues to plague many countries despite nutritional counseling and maternal iron and folic acid supplementation. This is the first examination of fluoride as an additional risk factor for anemia and low-birth-weight babies.

Anemic pregnant women living in India, whose urine contained 1 mg/L fluoride or more, were separated into two groups. The experimental group avoided fluoride in water, food and other sources and ate a nutritious diet per instruction. The control group received no instructions. Both groups supplemented with iron and folic acid.

Results reveal that anemia was reduced and pre-term and low-birth-weight babies were considerably fewer in the fluoride-avoidance group as compared to the control. Two stillbirths occurred in the control group, none in the experimental group.

Susheela et al. writes, "Maternal and child under-nutrition and anemia is not necessarily due to insufficient food intake but because of the derangement of nutrient absorption due to damage caused to GI (gastrointestinal) mucosa by ingestion of undesired chemical substances, viz. fluoride through food, water and other sources."

Fluoride avoidance regenerated the intestinal lining which enhanced the absorption of nutrients as evidenced by the reduction in urinary fluoride followed by rise in hemoglobin levels, they report.

Could the same thing be happening in the United States? State University of New York researchers found more premature births in fluoridated than non-fluoridated upstate New York communities, according to a presentation made at the 2009 American Public Health Association's annual meeting.

Current Science reports that adverse reactions of fluoride consumption are known to occur including reducing red blood cells, reducing blood folic acid activity, inhibiting vitamin B12 production and the nonabsorption of nutrients for hemoglobin biosynthesis.

"Citizens must demand that water fluoridation be stopped," says attorney Paul Beeber, President, New York State Coalition Opposed to Fluoridation, Inc. "It's disturbing that public-health officials and organized dentistry continue to ignore the overwhelming evidence revealing fluoride to be non-nutritive, unnecessary and unsafe," says Beeber.

Source: NYS Coalition Opposed to Fluoridation, Inc.

Article URL: http://www.medicalnewstoday.com/releases/199829.php

Overview: Infant Formula and Fluorosis

The proper amount of fluoride from infancy through old age helps prevent and control tooth decay. **Community water fluoridation** is a widely accepted practice for preventing and controlling tooth decay by adjusting the concentration of fluoride in the public water supply.

Fluoride intake from water and other fluoride sources, such as toothpaste and mouthrinses, during the ages when teeth are forming (from birth through age 8) also can result in changes in the appearance of the tooth's surface called dental fluorosis. In the United States, the majority of **dental fluorosis** is mild and appears as white spots that are barely noticeable and difficult for anyone except a dental health care professional to see.

Recent evidence suggests that mixing powdered or liquid infant formula concentrate with fluoridated water on a regular basis may increase the chance of a child developing the faint, white markings of very mild or mild enamel fluorosis.

You can use fluoridated water for preparing infant formula. However, if your child is exclusively consuming infant formula reconstituted with fluoridated water, there may be an increased chance for mild dental fluorosis. To lessen this chance, parents can use low-fluoride bottled water some of the time to mix infant formula; these bottled waters are labeled as de-ionized, purified, demineralized, or distilled.

What is the best source of nutrition for infants?

Breastfeeding is ideal for infants. CDC is committed to increasing breastfeeding throughout the United States and promoting optimal breastfeeding practices. Both babies and mothers gain many benefits from breastfeeding. Breast milk is easy to digest and contains antibodies that can protect infants from bacterial and viral infections. More can be learned about this subject at **http://www.cdc.gov/breastfeeding/**.

If breastfeeding is not possible, several types of formula are available for infant feeding. Parents and caregivers are encouraged to speak with their pediatrician about what type of infant formula is best suited for their child.

Why is there a focus on infant formula as a source of fluoride?

Infant formula manufacturers take steps to assure that infant formula contains low fluoride levels—the products themselves are not the issue. Although formula itself has low amounts of fluoride, if your child is exclusively consuming infant formula reconstituted with fluoridated water, there may be an increased chance for mild dental fluorosis.

Infants consume little other than breast milk or formula during the first 4 to 6 months of life, and continue to have a high intake of liquids during the entire first year. Therefore, proportional to body weight, fluoride intake may be higher for younger or smaller children than for older children, adolescents, or adults.

What types of infant formula may increase the chance of dental fluorosis?

There are three types of formula available in the United States for infant feeding. These are powdered formula, which comes in bulk or single-serve packets, concentrated liquid, and ready-to-feed formula. Ready-to-feed formula contains little fluoride and does not contribute to development of dental fluorosis. Those types of formula that require mixing with water—powdered or liquid concentrates—can be a child's main source of fluoride intake (depending upon the fluoride content of the water source used) and may increase the chance of dental fluorosis.

Can I use optimally fluoridated tap water to mix infant formula?

Yes, you can use fluoridated water for preparing infant formula. However, if your child is exclusively consuming infant formula reconstituted with fluoridated water, there may be an increased chance for mild dental fluorosis. To lessen this chance, parents can use low-fluoride bottled water some of the time to mix infant formula; these bottled waters are labeled as de-ionized, purified, demineralized, or distilled.

How can I find out the level (concentration) of fluoride in my tap water?

The best source of information on fluoride levels in your water system is your local water utility. Other knowledgeable sources may be a local public health authority, dentist, dental hygienist, or physician. CDC's Web site **My Water's Fluoride** allows consumers in some states to learn the fluoridation status of their water system. Nearly all tap water contains some natural fluoride, but, depending on the water system, the concentration can range from very low (0.2 mg/L fluoride or less) to very high (2.0 mg/L fluoride or higher). Approximately 72% of all public water systems serving about 195 million people adjust the fluoride in their water to the level recommended to prevent tooth decay.

Will using only low fluoride water to mix formula eliminate my child's risk for dental fluorosis?

Using only water with low fluoride levels to mix formula will reduce, but will not eliminate, the risk for dental fluorosis. Children can take in fluoride from other sources during the time that teeth are developing (birth through age 8). These sources include drinking water, foods and beverages processed with fluoridated water, and dental products, such as fluoride toothpaste, that can be swallowed by young children whose swallowing reflex is not fully developed.

Additional Resource

Dental Fluorosis – Learn more about simple steps to reduce your child's risk for dental fluorosis.

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Page Located on the Web at http://www.cdc.gov/fluoridation/safety/infant_formula.htm

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Affidavit of Gerard F. Judd, Ph.D.

Dr. Gerard Judd's book "Good Teeth, Birth to Death " is available at Amazon.

In Support of Motion for Summary Judgment

State of Wisconsin Circuit Court Fond Du Lac County

SAFE WATER ASSOCIATION, INC.,

Plaintiff,
vs.
Case No. 92 CV 579
CITY OF FOND DU LAC,

Defendant.

AFFIDAVIT OF GERARD F. JUDD, Ph.D.

IN SUPPORT OF MOTION FOR SUMMARY JUDGMENT

State of Arizona

County of Maricopa

Gerard F. Judd, Ph.D., being first duly sworn on oath and with personal knowledge of the information contained herein, respectfully states to the Court as follows:

BACKGROUND

1. I have been a professor of Chemistry at Phoenix College, Phoenix Arizona, since 1965.

2. I received my B.A. in chemistry from the University of Utah in 1943. I received my M.S. from the University of Portland in 1948. I received my Ph.D. in physical and organic chemistry from Purdue University in 1953. I did Postdoctoral research at Purdue University, on fluorinated organometallic reactions in 1954.

3. A few of my more recent research, academic and service honors include:

- a. Elected to Emeritus Member of American Chemical Society, 11/92.
- b. "20 Year Outstanding Teaching Award and Pin" from Maricopa County Community College District, Dr.

Paul A. Elsner, Chancellor, 2/92.
- c. Reviewed two fundamental chemistry textbooks for publishers, 5/91.
- d. Received "Speaker's Gold Plated Champion's Award for Research, Communication and Education," regarding better teeth, health and government. Arizona Breakfast Club, Harry E. Everingham, President, 11/24/90.
- e. "Faculty Appreciation Gift for Outstanding Contributions to Teaching," Maricopa County Community College District, Phoenix, Arizona, Dr. Paul A. Elsner, Chancellor, 3/4/85.

4. I have devoted thousands of hours during my career to studying the chemistry of fluoride. In addition, in preparing this affidavit, I have specifically researched and summarized the following professional and technical literature on the epidemiological effects of fluoride:

- a. Journal of Fluoride, August 1992 - January 1983.
- b. Chemical Abstracts, August 1992 - January 1989.
- c. Index Medicus, May, June and July 1992.
- d. Fluoridation The Great Dilemma, a 421-page book by
- George L. Waldbott, M.D. in collaboration with Albert W. Burgstahler, Ph.D. and H. Lewis McKinney, Ph.D.
- e. A Struggle With Titans, Forces Behind Fluoridation, a 383-page book by George L. Waldbott, M.D. (a scientist's look at fluoridation).
- f. Fluoridation, the Aging Factor, a 203-page book by John Yiamouyannis, Ph.D. in Biochemistry, and world-leading authority on the biological effects of fluoride.
- g. Fluoride, The Freedom Fight, a 207-page book by Dr. Hans Moolenburgh, M.D. (The Netherlands).
- h. Fluoridation, a 264-page book by Isabel Jansen, R.N.
- i. The Fluoride Question, Panacea or Poison?, a 176-page book by Anne-Lise Gotzsche, medical journalist (England).
- j. Hello, Test Animals . . . Chinchillas or You and Your Grandchildren, a 180-page book by W. R. Cox, chinchilla breeder and researcher.
- k. The Grim Truth About Fluoridation, a 128-page book by Robert M. Buck, journalist.
- l. Fluoridation, Poison on Tap, a 460-page book by Glen S. R. Walker, consultant in strategic metals, munitions, and chemical industry, registered by the National Association of Testing Authorities in Australia.
- m. Fluoride in Australia, a Case to Answer, a 159-page book by Wendy Varney, journalist.

5. During the past two years I have personally discussed the effects of artificial fluoridation of drinking water with many individuals possessing outstanding background on the subject of fluoridation, including the following:

- a. Dr. Albert Burgstahler, Ph.D., University of Kansas;
- b. Dr. Mel Ruber, Ph.D., Columbia, Maryland;
- c. Dr. Robert Carton, Ph.D., former head of EPA Employees Union, Environmental Protection Agency, Washington, D.C.;
- d. Dr. William Marcus, Ph.D., epidemiologist, Environmental Protection Agency, Washington, D.C.;
- e. Dr. William Foulkes, M.D., Vancouver, Washington, former head of Ministry of Health in British Columbia;
- f. Dr. John Colquhoun, Ph.D., Titi Rangi, New Zealand;
- g. Dr. Albert Schatz, Ph.D., chemistry researcher, retired, Temple University, Philadelphia, Pennsylvania;
- h. Dr. Cornelius Steelink, Ph.D., Chemistry Professor, University of Arizona;
- i. Dr. John Yiamouyiannis, Ph.D., Delaware, Ohio;
- j. Dr. John R. Lee, M.D., Sebastopol, California.

EVIDENCE AGAINST THE SAFETY OF FLUORIDATION

6. My research, communication and discovery concerning the epidemiological effects of fluoridation has provided me with solid scientific evidence on which to base the following conclusions.

7. Fluoride has never been established as, and is not, essential in nutrition for soft tissues, bones or teeth.

8. There are no experiments or data which establish that fluoride in any form or in any concentration is harmless when put into drinking water for human consumption or usage.

9. Fluoride at low levels has been shown to unsnap hydrogen bonds in the enzyme cytochrome oxidase, and thus

ruin its ability to handle oxygen in humans, animals or plants. (Exhibit____).

10. It is well-established in academic and industrial chemical industry that the hydrogen-fluoride hydrogen bond is stronger than the hydrogen-nitrogen or hydrogen-oxygen hydrogen bonds characteristic of human enzymes. Therefore, human enzyme systems (thousands of enzymes) are subject to destruction when water containing fluoride is consumed. After a few weeks for some, and a lifetime for others, a large enough reservoir of fluoride is built up to cause serious ailments.

11. At least 63 human, animal and plant enzymes are for the most part destroyed or modified by fluoride.

12. A summary of important epidemiological effects of fluoride from Chemical Abstracts, 1992-1989 (53 pages); Journal of Fluoride, August 1992-January 1993 (42 pages); and Index Medicus, May, June and July 1992 (3 pages) failed to produce even one article proving fluoride to be harmless.

13. Contrariwise, hundreds of experiments on test animals, humans, plants, and their cells, have invariably demonstrated harmful effects.

14. A large number of epidemiological effects in the way of ailments and allergies caused by fluoride have been clinically established by competent authorities, including those below.

15. Forty-nine or more serious allergenic conditions were established by George Waldbott, M.D. These were proven by removing patients from drinking water with fluoride in it, in which case they were cured. This was followed up with single- or double-blind tests with fluoride tablets.

16. Eight of Dr. Waldbott's side effects were confirmed through double-blind tests organized by Dr. H. Moolenburgh, with 12 other physicians, one pharmacist, and one attorney. (Exhibit____). Only one of these side effects presented in court was sufficient to cause the Holland Ministry of Health to discontinue fluoridation of water in that country. These side effects are listed in Waldbott's book, pp. 123-125.

17. Genetic changes in bone cells and sperm cells of mice were thoroughly studied, re-studied and established by Dr. Albert Taylor. This work has been confirmed by numerous other researchers.

18. Fluoride as a factor in cataracts has been established by statistical studies of Dr. Ionel F. Rapaport and confirmed by the research of Dr. Burgstahler. This has also been confirmed by analysis of cataracted and un-cataracted eye lenses. The older the person, the more the fluoride in the lens. (Exhibit____).

19. SIDS (crib or cot death) has been related to fluoride poisoning by Dr. J. Colquhoun (exhibit____), Dr. Bruce Spittle, and others.

20. Chronic fatigue syndrome (CFS), and chronic fluoride toxicity (CFT) have been found to be very closely related in their symptoms (Exhibit____).

21. RSI (repetitive stress injury, or carpal tunnel syndrome) has been linked to the accumulation of fluoride in the bone by Dr. Geoffrey E. Smith. Additional work supporting this link was found by Dr. Sutton. (Exhibit____).

22. Dental fluorosis has been shown recently to occur at fluoride levels as low as .3 ppm, as opposed to earlier studies of Dr. H. Trendly Dean, who set 1.0 as a tolerable limit, allowing 24 percent fluorosis. The degree of fluorosis depends on the nutritional status of the person.

23. Dr. Waldbott had over 400 cases of pre-skeletal bone fluorosis in patients, which he established was caused by their drinking fluoridated water. (Exhibit____) This has been further confirmed by many other studies. The degree of bone fluorosis is strictly related to bone fluoride content.

24. Embrittled bones are caused by drinking fluoridated water, as well as by administration of tablets to "harden bones." (Riggs study, Exhibit____; Utah study, Exhibit____; Jacobson's study, Exhibit____; Cooper's study, Exhibit____; and Sower's study, Exhibit____.

25. Increased infant mortality and birth defects (two to three times increase) was established by Dr. Albert Schatz

to be present in Chilean children administered fluoridated water in an experimental study in Curico, Chili, with San Fernando and La Serena as a control towns. (Exhibit____). Dr. Schatz found fluoridation did no good for teeth, and caused enormous increase in miscarriages. The malformations and infant mortality dropped dramatically upon cessation of the fluoridation. Similar malformations and infant mortalities are now occurring in U.S.

26. C. R. Cox, working with the University of Oregon, found that 17 ppm fluoride in feed caused constipation, great mature and baby chinchilla death, small litters and over four generations a smaller, inferior rabbit.

27. Down's Syndrome was established to be linked to consumption of fluoride through statistical studies and re-studies by Dr. Ionel F. Rapaport, M.D. and Waldbott, Fluoridation the Great Dilemma, pp. 212-219. Dr. Rapaport also found that 70% of Down's Syndrome babies were born with cataracted eyes.

28. Genu valgum (knock knees) has been reported as having been caused by fluoride in drinking water..

29. Gilbert's Disease (hemorrhagic yellow jaundice) has been cured by taking the patient off fluoridated drinking water. (Exhibit____).

30. Collagen synthesis has been shown to be impeded by fluoride by the work of B. Uslu, Andola School of Medicine, Eskisehir, Turkey.

31. Immunosuppression, according to Sutton and Gibson, may be caused by consumption of fluoride. (See Exhibits_____).

32. Decreased immunodiffusion has been established as due to fluoride ion, making it a negative chemitaxic agent (this means it impedes the "taxiing" or motion effect). (Exhibit____).

33. Between 1953 and 1968, there were approximately 572,810 (44,062 per year average) more deaths due to all types of cancer in 10 major fluoridated cities compared to non-fluoridated cities. Sex, race and age changes in these populations were insignificant during this period, so that nothing else could be established as causal. (Exhibit____).

34. In Antigo, Wisconsin, heart attacks were shown to dramatically increase both in the general population and the people under 65 and over 65 when fluoridation was instituted and continued over 35 years.

35. A tremendous increase in caiman (alligator) deaths was experienced once Kansas City, Kansas water was fluoridated at the Parrot Hill farms under the care of Patricia Jacobs, naturalist.

EVIDENCE AGAINST THE EFFECTIVENESS OF FLUORIDATION

36. In contrast to the claims of the Human Health Services and the American Dental Association that fluoride reduces DMF (decayed, missing, filled teeth) 65 percent, it has now been established through a very large number of reliable studies that fluoride may actually cause a slight amount of DMF. (A large amount of DMF is actually related to nutrition.)

37. Dr. Yiamouyiannis found that of 39,200 students, ages 5-19, from 89 fluoridated and non-fluoridated areas, the teeth of those living in non-fluoridated areas had slightly less DMF. (Exhibit___/___).

38. A survey of 1,500 fifth grade students in Missouri gave slightly lower DMF for those who lived in a non-fluoridated area. This was also true in a survey of 1500 6th graders.(Exhibit____).

39. A study of school children in Tucson, Arizona by Dr. Cornelius Steelink (Chemistry Department, University of Arizona), established that there was an increase in DMF with an increase in fluoride in the water. (Exhibit____).

40. A thorough study of the entire population of Japan (included 20,000 school children, 1972) established that when the fluoride in the drinking water was above .4 ppm there was more decay. (Exhibit____).

41. A study of Auckland, New Zealand, found that DMF decreases depended heavily on dental education in the schools and the salary of people from various areas, and insignificantly on the amount of fluoride in the water.

(Exhibit____).

42. In Garis, Africa a high proportion of 14 to 15-year-olds had first permanent molars which were extensively carious or missing despite 1.06 ppm fluoride in drinking water. High sugar intake was a possible factor.

43. Earlier "studies" justifying fluoridation of drinking water have been unmasked and debunked by competent authorities (Dr. Waldbott, Dr. Colquhoun, Dr. Foulkes, Dr. Mark Diesendorf, Dr. Sutton, Dr. Exner and Dr. Rudolf Ziegelbecker) on the basis of neglecting variables, cheating and group selection, not completing the studies, etc. (Exhibits_____).

44. As one example, phosphate, calcium and strontium were not accounted for in the Newburgh-Kingston study, or any other study, to the best of my knowledge. Dr. Waldbott established that the Kingston water had deficiencies of these elements.

CONCLUSION

45. My research has made it clear that the American Dental Association and U. S. Human Health Services have made a wrong turn in their attempt to improve the teeth of the American public.

46. Fluoride in drinking water should be limited to .1 ppm where possible, since reverse osmosis can easily reduce fluoride below this value.

47. It is my best judgment, reached with a high degree of scientific certainty, that fluoridation is invalid in theory and ineffective in practice as a preventive of dental caries. It is also dangerous to the health of consumers.

48. I make this Affidavit in support of the Plaintiff's Motion for Summary Judgment.

Dated this _____ day of _____, 1993.

_____
Gerard F. Judd, Ph.D.

Subscribed and sworn to before me

this ___ day of _____, 1993.

_____

Notary Public, State of _____

My Commission:_____

Dr. Gerard Judd's book "Good Teeth, Birth to Death " is available at Amazon.

Back

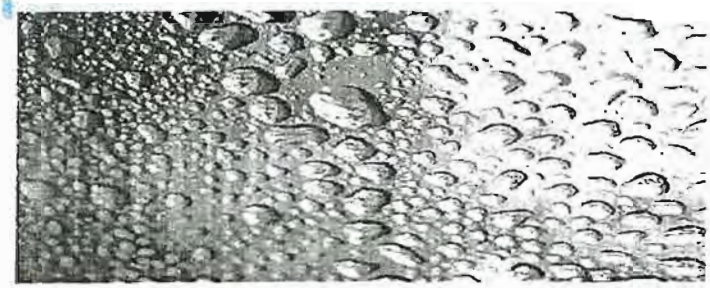
LEAD – FLUORIDE LINK

Chloramine + Lead Pipes + Fluoride = Contaminated tap water

JULY 13, 2009

By Olga Naidenko, EWG Senior Scientist

The lead pollution crisis of the Washington, D.C. water supply - and the culprit that caused it, the water disinfection chemical chloramine - is a powerful example of how things can go terribly wrong when water quality problems are considered and tackled in isolation.



Earlier this year, Virginia Polytechnic Institute and State University (Virginia Tech) scientists reported the shockingly high lead levels in the blood of young Washington, D.C. children tested between 2001 and 2004, when the District of Columbia's drinking water was being contaminated with lead from aging pipes.

Unfortunately, this situation is not unique: similar results have been reported in Greenville, North Carolina, according to studies by the Duke University researchers.

Chloramines and lead pipes: Not so good together

American water utilities are increasingly switching to chloramines, a mixture of chlorine and ammonia, for final disinfection of drinking water. Chloramine was supposed to be a "safer" water disinfectant than chlorine because it reduces formation of toxic chlorination byproducts. A 2005 survey by the American Water Works Association found that approximately a third of all utilities now use chloramines.

Water disinfection byproducts are associated with increased risk of cancer and possibly adverse effects on the development of the fetus, so minimizing their levels in drinking water is a good thing. Yet, chloramines drastically increase the leaching of lead from pipes. And here is a real challenge: there are tens of thousands of lead service lines in the water system administered by the DC Water and Sewer Authority. Add to these lines the lead based solder used to join copper pipe, brass and chrome plated faucets, and water fixtures, and the opportunities for lead to leach into the drinking water multiply.

We all accept that water disinfection is a public health necessity. However, we need to thoroughly consider the full impact of any chemical added to drinking water *given the current water distribution infrastructure in place*, not in some theoretical vacuum. As described by Duke researchers, chloramine-induced lead leaching might be lessened by the addition of anticorrosivity agents during the water treatment process. Is that sufficient for protection of public health? We really don't know! Chloramine itself has been associated with severe respiratory toxicity and skin sensitivity. Overall, despite ongoing research, water treatment chemistry is still insufficiently understood by scientists and specific water quality outcomes depend on the particular chemical interactions found in each water treatment and distribution system.

And now add fluoride

In addition to disinfection chemicals, other additives are commonly mixed with the finished drinking water before it leaves the water treatment plant. Of them, fluoride is possibly the most known. Two thirds of the U.S. municipal water supply is artificially fluoridated *in an effort to prevent* tooth decay. But fluoridation additives in tap water are not the same form of fluoride as found in toothpaste. Typically, water is fluoridated with fluorosilicic acid (FSA) or its salt, sodium fluosilicate, collectively referred to as fluorosilicates. In contrast, fluoride in toothpaste is usually in form of simple sodium fluoride salt, NaF.

Here comes a second unpleasant "surprise" for those in lead-piped locations: fluorosilicates have a unique affinity for lead. In fact, lead fluorosilicate is one of the most water-soluble forms of lead. In fact, fluorosilicic acid has been used *as a solvent* for lead and other heavy metals in metallurgy. In industrial applications, chemical engineers rely on this acid to remove surface lead from leaded-brass machine parts.

Research shows what happens when we mix it all up

What happens when fluorosilicates in water pass through lead-containing pipes and metal fixtures? Not surprisingly, the

fluorosilicates extract high levels of soluble lead from leaded-brass metal parts (researchers from the Environmental Quality Institute of the University of North Carolina-Asheville performed this actual experiment).

In research published in the scientific journal *Neurotoxicology*, researchers found that the mixture of the two chemicals: disinfectant (whether chlorine or chloramine) with fluorosilicic acid has a drastically increased potency, leaching amazingly high quantities of lead.

Where does this lead go? Into our drinking water and right on into our bodies, where they wreak havoc by poisoning our heart, kidneys and blood, causing irreversible neurological damage and impairing reproductive function.

North Carolina researchers concluded that the supposedly innocuous - and purportedly beneficial - quantities of fluoride added to drinking water may, in fact, precipitate a cascade of serious health problems, especially when chloramines and lead pipes are added into the mix.

Do we even need fluoride in tap water?
The mixture of chloramine and fluorosilicates in drinking water causes extensive leaching of lead. We cannot dispense with water disinfection - everybody acknowledges this. Thus, chlorine and chloramine are probably here to stay for some time. On the other hand, fluoride, or, specifically, water fluoridation with fluorosilicates, is quite dispensable.

But wait - isn't fluoride the miracle chemical that improves dental health?

Well, yes and no. Much of what is publicized today in caries prevention programs worldwide is derived from the theories generated in the 1950s and '60s, when water fluoridation was actively promoted. As we now know, the main benefits of fluoride for dental health are derived from *surface* application on the teeth, not from ingestion.

In fact, ingestion of fluoride causes dental fluorosis, a range of adverse health effects that includes mottling, pitting, and weakening of the teeth. These risks are especially significant for infants and young children. In the U.S. and worldwide, about 30 percent of children who drink fluoridated water experience dental fluorosis. In 2006, the American Dental Association (ADA) issued an "Interim Guidance on Fluoride Intake for Infants and Young Children." ADA recommended that in areas where fluoride is added to tap water, parents should consider using fluoride-free bottled water to reconstitute concentrated or powdered infant formula to avoid excess fluoride.

According to the latest research, the anti-caries activity of fluoride is due to topical effects, which supports the value of fluoride-containing toothpaste to dental health. There is clear evidence that fluoride dental products significantly reduce the incidence of cavities. In contrast, a substantial and growing body of peer-reviewed science suggests that ingesting fluoride in tap water does not provide any additional dental benefits other than those offered by fluoride toothpaste and may present serious health risks.

To learn more about fluoride health effects, read the recent report by EWG.

The message: Don't assess chemicals in isolation
The lesson here is straightforward: it is completely unscientific to simply toss any chemical into the drinking water on the premises that this chemical might provide some benefits. **The real question is:** what would be the effect of this chemical given what else is going on with the water system? In case of fluoridation and chloramines, what emerges at the end of the pipe (our faucets!) is a potentially highly hazardous mixture of fluorosilicates, lead, and residual levels of disinfectants.

To protect the health of my family today, I can buy a water filter to remove heavy metals and disinfection byproducts from my drinking water with a simple pitcher filter. But **to protect the health of the entire nation,** we really need to consider if our current methods of water treatment can withstand scientific scrutiny, or whether they should be re-assessed so as to provide safe, healthy tap water to all Americans.

« EWG heads to Congress: Thanks for your help |

International Journal of Environmental Studies

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Water treatment with silicofluorides and lead toxicity

DOI:
10.1080/00207239908711215

Roger D. Masters[a] & Myron J. Coplan[b]

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Toxic metals like lead, manganese, copper and cadmium damage neurons and deregulate neurotransmitters like serotonin and dopamine (which are essential to normal impulse control and learning). Earlier studies show that — controlling for socio-economic and demographic factors — environmental pollution with lead is a highly significant risk factor in predicting higher rates of crime, attention deficit disorder or hyperactivity, and learning disabilities. Exposure and uptake of lead has been associated with industrial pollution, leaded paint and plumbing systems in old housing, lead residues in soil, dietary habits (such as shortages of calcium and iron), and demographic factors (such as poverty, stress, and minority ethnicity). We report here on an additional "risk co-factor" making lead and other toxic metals in the environment more dangerous to local residents: the use of silicofluorides as agents in water treatment. The two chemicals in question — fluosilicic acid and sodium silicofluoride — are toxins that, despite claims to the contrary, do not dissociate completely and change water chemistry when used under normal water treatment practices. As a result, water treatment with siliconfluorides apparently functions to increase the cellular uptake of lead. Data from lead screening of over 280,000 children in Massachusetts indicates that silicofluoride usage is associated with significant increases in average lead in children's blood as well as percentage of children with blood lead in excess of 10μg/dL. Consistent with the hypothesized role of silicofluorides as enhancing uptake of lead whatever the source of exposure, children are especially at risk for higher blood lead in those communities with more old housing or lead in excess of 15 ppb in first draw water samples where silicofluorides are also in use. Preliminary findings from county-level data in Georgia confirm that silicofluoride usage is associated with higher levels of lead in children's blood. In both Massachusetts and Georgia, moreover, behaviors associated with lead nurotoxicity are more frequent in communities using silicofluorides than in comparable localities that do not use these chemicals. Because there has been insufficient animal or human testing of silicofluoride treated water, further study of the effect of silicofluorides is needed to clarify the extent to which these chemicals are risk co-factors for lead uptake and the hazardous effects it produces.

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Keywords

- lead,
- toxicity,
- pollution,
- children's health,
- public water supplies

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ELSEVIER FULL-TEXT ARTICLE

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Fluoride increases lead concentrations in whole blood and in calcified tissues from lead-exposed rats.

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Abstract

Higher blood lead (BPb) levels have been reported in children living in communities that receive fluoride-treated water. Here, we examined whether fluoride co-administered with lead increases BPb and lead concentrations in calcified tissues in Wistar rats exposed to this metal from the beginning of gestation. We exposed female rats and their offspring to control water (Control Group), 100mg/L of fluoride (F Group), 30mg/L of lead (Pb Group), or 100mg/L of fluoride and 30mg/L of lead (F+Pb Group) from 1 week prior to mating until offspring was 81 days old. Blood and calcified tissues (enamel, dentine, and bone) were harvested at day 81 for lead and fluoride analyses. Higher BPb concentrations were found in the F+Pb Group compared with the Pb Group (76.7+/-11.0microg/dL vs. 22.6+/-8.5microg/dL, respectively; $p<0.001$). Two- to threefold higher lead concentrations were found in the calcified tissues in the F+Pb Group compared with the Pb Group (all $p<0.001$). Fluoride concentrations were similar in the F and in the F+Pb Groups. These findings show that fluoride consistently increases BPb and calcified tissues Pb concentrations in animals exposed to low levels of lead and suggest that a biological effect not yet recognized may underlie the epidemiological association between increased BPb lead levels in children living in water-fluoridated communities.

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NeuroToxicology

Effects of fluoridation and disinfection agent combinations on lead leaching from leaded-brass parts

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Abstract

This study concerns effects on water-borne lead from combinations of chlorine (CL) or chloramines (CA) with fluosilicic acid (FSA) or sodium fluoride (NaF). CL is known to corrode brass, releasing lead from plumbing devices. It is known that CA and CL in different ratios with ammonia (NH_3) mobilize copper from brass, which we have found also enhances elution of lead from leaded brass alloys. Phase I involved leaded-brass 1/4 in. elbows pre-conditioned in DI water and soaked in static solutions containing various combinations of CL, CA, FSA, NaF, and ammonium fluosilicate. In Phase II 20 leaded-brass alloy water meters were installed in pipe loops. After pre-conditioning the meters with 200 flushings with 1.0 ppm CL water, seven different solutions were pumped for a period of 6 weeks. Water samples were taken for lead analysis three times per week after a 16-h stagnation period. In the static testing with brass elbows, exposure to the waters with CA + 50% excess NH_3 + FSA, with CA and ammonium fluosilicate, and with CA + FSA resulted in the highest estimated lead concentrations. In the flow-through brass meter tests, waters with CL + FSA, with CL + NaF, and with CL alone produced the highest average lead concentration for the first 3-week period. Over the last 3 weeks the highest lead concentrations were produced by CL + NaF, followed by CL alone and CA + NH_3 + FSA. Over the first test week (after CL flushing concentrations were increased from 1.0 to 2.0 ppm) lead concentrations nearly doubled (from about 100 to nearly 200 ppb), but when FSA was also included, lead concentrations spiked to over 900 ppb. Lead concentrations from the CL-based waters appeared to be decreasing over the study period, while for the CA + NH_3 + FSA combination, lead concentrations seemed to be increasing with time.

Keywords: Water disinfection; Chlorine; Chloramines; Fluoridation; Lead leaching

1. Introduction

1.1. Motivation for this study

The continuing problem of ingested lead from lead-bearing water was highlighted at a US House of Representatives sub-committee hearing convened in March 2004 to investigate issues concerning "First Draw" water lead levels as high as 1000 ppb in Washington DC water circa 2001–2004. An expert witness (Edwards, 2004) testified that this was found in homes without lead service lines or lead soldered copper piping. The only possible lead source had to be leaded-brass plumbing and/or brass faucets. The expert also suggested that a recent switch in disinfectant from chlorine to chloramine caused the problem. The study reported here, conducted by the Environmental Quality Institute of the University of North Carolina (EQI), focused on brass corrosion by combinations of disinfectant and fluoridating agents in two laboratory phases. In the first, small leaded-brass plumbing elbows (2% lead) were exposed under static conditions to DI water with chlorine and chloramines, either alone or in combination with municipal water fluoridating agents. Stagnant water lead data from that phase guided selection of combinations of disinfectant and fluoridating agent for a second phase in which brass water meters (8% lead) were exposed to seven water formulations under flow-through conditions. It was expected that Phase II results would be used to guide field tests under "real world" conditions in cooperating water plants, but the untimely demise of EQI Director Richard P. Maas prevented that follow-on step. Nevertheless, Phase I and Phase II results presented here provide at least heuristic insight

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into the "DC experience" and, more generally, shed new light on lead elution from brass by combinations of chlorine-based disinfectants and fluoridation chemicals.

1.2. Applicable terminology

Herein "CL" means a chlorine species used for potable water disinfection that may be injected as chlorine gas (Cl_2), or hypochlorite solutions carrying chloride ion, hypochlorous acid (HOCl), and/or hypochlorite ion (OCl^-) that may exist together in equilibrium. CL concentration may be expressed in parts per million (ppm) of "free chlorine" where 1 ppm is the stoichiometric equivalent of 29×10^{-6} mol of free chloride ion.

"NH" means ammonia added to CL treated water to produce mono-chloramine. NH may be injected as ammonia gas (NH_3), ammonium hydroxide solution, or as an ammonium salt solution. The desired proportion of CL/NH is 1:1 on a molar basis which is little less than 5:1 in ppm units.

"CA" means "chloramine" produced by adding NH to CL treated water; the desired mono-chloramine is actually part of a mixture with small amounts of di- and tri-chloramine. Actual amounts of NH and CL vary from time to time, yielding either undesired di- or tri-chloramine or excess NH. Chlorine in CA is also known as "combined chlorine," a term also applied to products created when CL reacts with water contaminants (USEPA, 2004).

"SiF" applies to the silicon/fluorine complex (fluosilicate), a class of water fluoridating agents (aka fluorosilicates, silicofluoride, silicofluoric, hexafluorosilicate, and other names) from which fluoride ion (F^-) is released upon dissociation of $[SiF_6]^{2-}$ when diluted in water. The principal SiF agents are fluosilicic acid (H_2SiF_6), herein "FSA", and its sodium salt (Na_2SiF_6). Concentrated (20–30%) FSA is injected as such into water plant water. Sodium fluosilicate (NaFSA) is added as a saturated solution. The term SiF covers $[SiF_6]^{2-}$ and its dissociation derivatives.

2. Relevant background

2.1. Continuing problem of drinking water lead

Lead contaminated drinking water remains a significant public health issue in the United States, even though water-borne lead has steadily declined along with other lead sources such as lead-based paint, roadside soils, food, and other products. In 1991, the EPA estimated that drinking water was responsible for 14–20% of total lead uptake of all ages in the U.S. (USEPA, 1991). EPA's Lead and Copper Rule (LCR) for potable water was expected to reduce drinking water lead levels by 50%. That did occur (Maas et al., 2005) after leaded-solders were banned under the Safe Drinking Water Act Amendments of 1986 (USEPA, 1986); water suppliers were required to reduce corrosivity of their finished waters (Maas et al., 1994; Ramaley, 1993; USEPA, 1991); with better control of alkalinity, pH, and additives (Cardew, 2003; Edwards et al., 1996; Lytle and Schock, 2000). On its own initiative, the California legislature set limits on lead content of leaded-brass plumbing devices and faucets (Patch et al., 1998; State of California, 1995). Much research focused on preventing lead extraction from installed lead service lines by treating water with phosphatic agents (phosphoric acid, combinations of orthophosphoric acid and zinc orthophosphate, polyphosphates, or blends of orthophosphoric acid with polyphosphate) that produce inert barrier coatings inside lead pipes. Along with successes in this area there have also been conflicting results. Orthophosphate treatments can reduce soluble lead levels by 70%, but polyphosphate can actually increase lead and copper in drinking water (Edwards and McNeill, 2002), often manifested as particulates (McNeill and Edwards, 2004).

2.2. Complicating factors

2.2.1. Switch to chloramines for disinfection

Water lead problems have been exacerbated by EPA's Stage I Disinfection By-products Rule (USEPA, 2002) requiring reduction of disinfection by-products (DBPs) such as trihalomethanes (THM's) and haloacetic acids (HAA's) created by CL disinfection (ChemScan, 1997). A switch from CL to CA was recommended and adopted in some systems since it was less expensive than other disinfection methods and easy to add NH to already-chlorinated water. One explanation for the DC experience was that the switch from CL lowered the oxidizing potential of DC water, destroying the normally protective lead dioxide (PbO_2) scale inside lead pipes (Renner, 2004).

Although CA corrosivity has received a lot of attention, no studies have included fluoridating agents (Edwards and Dudi, 2004; Eisnor and Gagnon, 2004; Lin et al., 1997; Reiber, 1993; Sung et al., 2005). A microscopy study revealed how CA alone is a good solvent for lead (Switzer et al., 2006). Whatever these studies may have found under laboratory conditions, it should be noted that CA in the water plant is not added as a commercial product with consistent properties. It is formed by adding NH to CL treated water. Ideally, mono-chloramine is the principal product formed at pH 8 and the proper 1:1 NH/CL molar ratio. Maintaining exactly the ideal NH/CL proportion at all times is not very likely.

2.2.2. The role of fluoridating agents

Fluoridating agents can only complicate matters. Sodium fluoride (NaF), used to treat less than 10% of US fluoridated water, raises pH a little with negligible effect, but the same cannot be said for possible interference by the fluoride ion in the reaction of NH with CL. The effect of SiFs is another matter. The fluosilicate anion $[SiF_6]^{2-}$ of FSA and NaFSA provides the fluoride ion (F^-) in over 90% of fluoridated water. $[SiF_6]^{2-}$ releases F^- in a complicated, poorly understood, sequence of time-, temperature- and pH-dependent steps. Under water plant operating conditions, incompletely dissociated $[SiF_6]^{2-}$ residues may survive and react with other chemicals in the water. Under some conditions, NH and FSA, as such, react to produce silica and ammonium fluoride (Mollere, 1990). How that affects corrosion is not known, but whatever its reaction with NH may be, FSA does not leach lead simply because it is an acid.

The fluosilicate anion $[SiF_6]^{2-}$ and/or partially dissociated derivatives have a unique affinity for lead. Lead fluosilicate is

one of the most water soluble lead species known, a property recognized and exploited for many years (Stauter, 1976). FSA has been used as a solvent for lead and other heavy metals in extractive metallurgy (Cole et al., 1981; Kerby, 1979) and to remove surface lead from leaded-brass brass machined parts (Bonomi et al., 2001; Giusti, 2001, 2002). With or without CA, FSA would extract lead from brass. Besides, in the water plant situation it is reasonable to expect FSA to combine with NH as ammonium fluosilicate, an excellent solvent for copper alloys (Hara et al., 2002) and other metals (Silva et al., 1995).

It has been argued that FSA dissociates almost completely at the levels typically added to drinking water, and therefore cannot be more corrosive than sodium fluoride (NaF) (Urbansky and Schock, 2000). However, in a comprehensive follow-up review of the literature, Urbansky states that FSA may not dissociate completely in drinking water (Urbansky, 2002). Evidence for that is not new (Colton, 1958; Kolthoff and Stenger, 1947; Lenfesty et al., 1952; Munter et al., 1947; Thomsen, 1951). Titration of FSA to a pH 7 end-point only neutralizes the two hydronium ions produced by ordinary hydrolysis of H_2SiF_6, leaving the fluosilicate anion $[SiF_6]^{2-}$ intact. In addition to that, $[SiF_6]^{2-}$ dissociation in cold water could take 20 min to reach 90% completion (Hudleston and Bassett, 1921; Rees and Hudleston, 1936) and may never get to that condition below pH 9 (AWWA, 1994).

Consequently, incompletely dissociated $[SiF_6]^{2-}$ residues may remain in water plant water that is not above pH 8 or some commonly occurring low temperature. Apart from problems with incompletely dissociated $[SiF_6]^{2-}$ residues, injection of concentrated of FSA simultaneously and in close proximity with NH almost guarantees unanticipated side reactions.

2.2.3. *Distribution of lead in brass*

The varied, occasionally conflicting, reports on elution of lead from brass may have a common explanation. Lead alloyed with copper is not molecularly distributed, as in a solid solution. Discrete lead nodules are embedded in a copper matrix. Agents that attack copper are likely to foster lead mobility, adding significantly to lead (probably particulate) in drinking water. CL, CA, or excess NH are all capable of doing that, either by copper stress cracking (Flom, 2002) or mobilization in an ammonia/copper complex (Clark, 2003), thereby exposing lead nodules in brass for easier transport into water.

This may help to explain the DC experience that homes with only brass as a possible source of lead, not only had high water lead, but were also experiencing serious pitting of copper pipe. In many cases, particulate lead may predominate over soluble lead eluted from brass, as well as other lead sources (McNeill and Edwards, 2004).

3. Materials/methods and statistical analyses

3.1. *Phase I (static tests of 2% leaded-brass elbows)*

Sixty 2% leaded-brass 1/2 in. barb 90° elbows were purchased locally. Three elbows were assayed for lead in a small piece sliced from one end. Measured lead concentrations ranged from 1.70 to 1.82%. Elbows were labeled, thoroughly rinsed, and placed in a tray of deionized (DI) water for conditioning. The water was changed twice and agitated three times a day for 18 days. After conditioning, two sets of static bottle tests were conducted as follows: individual elbows were removed from the trays of DI water, rinsed with DI water and placed in their own labeled bottle. Exactly 100 mL of the appropriate test water was added to each bottle which was capped and set aside to sit undisturbed overnight. After 16-h stagnation exposure, each elbow was removed from its bottle of test water with plastic tongs, rinsed with DI water, and placed back into its tray of DI water. Test waters were analyzed for lead using the EPA 200.8 method for graphite furnace atomic absorption spectrophotometry.

In the first set of bottle tests, elbows were exposed to waters at pH 7 and pH 8, comprising 2 ppm each of: (1) CL only; (2) FSA only; (3) CA only; (4) CA + FSA. CL was adjusted by adding the appropriate amount of dilute sodium hypochlorite (NaOCl) solution. For the FSA waters, enough FSA was added to produce 2.0 ppm F^- which represents above average, but not unusual conditions within the highest permissible level (MCL) for drinking water fluoride. FSA was added as pre-diluted 26% FSA.

Although the CDC nominal "optimum" adjusted F^- concentration is 1.0 ppm, it is only a mid-range figure (CDC, 2001). The CDC recommends adjusting F^- according to mean annual local temperature. In colder areas (50–54 °F annual mean) such as Great Lakes States, the optimum is 1.1–1.7 ppm and in warmer areas (71–79 °F) it is 0.7–1.3 ppm. An allowance is also made for deviation from these boundaries by 0.1 ppm on the low side and 0.5 ppm on the high side (CDC, 1999). Hence, a water plant taking water in December through April from Lake Ontario or the Northern reaches of the Mississippi River could comply with CDC's optimum F^- at 2.0 ppm. Also, the recommended optimum F^- for school water systems is 4.1–5.0 ppm for the middle temperature range (CDC, 1999). Therefore, the 2.0 ppm F^- concentrations in this study were in the range experienced by much of the U.S. public.

CA was prepared as a stock solution comprising ammonium hydroxide and sodium hypochlorite in stoichiometric equivalent concentrations. For test water exposure, appropriate amounts of stock solution were pH adjusted upward by adding sodium bicarbonate ($NaHCO_3$) or downward using hydrochloric acid (HCl). Elbows were randomly assigned to test waters so that each water composition had five elbows assigned to it.

The conditionings using pH 8 were dropped in the second set of bottle tests to allow testing of more types of water additives. The same procedures used in the first set of bottle tests were carried out in the second set at pH 7 and 2.0 ppm of each constituent: (1) DI water only; (2) CL only; (3) CA only; (4) CA + 50% excess NH; (5) CA + FSA; (6) CA + 50% excess NH + FSA; (7) CA solution into which 26% FSA was added to produce 2 ppm of fluoride without pre-dilution; (8) FSA + NH; (9) CA + ammonium fluosilicate.

CL, CA, FSA, and pH were adjusted the same way as the first bottle tests. NH was adjusted by adding ammonium hydroxide

in appropriate 1:1 molarity with CL alone as well as with 50% excess NH to represent water plant control deviations described above regarding excess ammonia. The 50% excess NH is consistent with 50% greater than nominal 1 ppm F^- optimum, therefore a reasonable condition to occur in a water plant with the risk of side reaction between SiF and NH. The difference between conditions 5 and 7 was based on that premise. In condition 5, FSA was diluted before adding it to the mixture, as was done for other treatments using FSA. In condition 7, it was added at 26% concentration and the resulting mixture diluted to 2 ppm F^-.

For reasons described earlier, mixing concentrated FSA with NH with ample time to react should approximate water plant conditions, producing a species with corrosion potential differing from that when pre-diluted dissociated FSA meets ammonia. It should be noted here that concentrated FSA and NH are frequently injected into water plant water in close proximity to each other (District of Columbia Water and Sewer Authority, 2002). The rationale for including conditions 8 and 9 was that NH is known to react with copper, forming the soluble copper/ammonia complex, thus possibly exposing additional lead surface in the brass.

3.2. Phase I statistical analyses

For both sets of bottle tests, ANOVA analyses conducted on the natural-log-transformed lead concentrations found no evidence of significant non-normality (Kolmogorov–Smirnov p-values = 0.124 and 0.100) or heterogeneity of variances (Levene p-values = 0.191 and 0.979). Tukey's least significant difference (LSD) procedure was used to perform a multiple comparison of log lead concentrations between each combination of water and pH. Confidence intervals for the median lead concentrations of all elbows that might be exposed to those conditions were calculated for each water-pH combination under the assumptions that the log lead concentrations were approximately normally distributed with common variance. Least square means and corresponding individual confidence intervals were calculated from the ANOVA analysis for these log lead concentrations for each combination, and then the inverse transform was conducted on the least square means of the logged data to obtain the estimated medians and 95% confidence intervals for the median lead concentrations.

3.3. Phase II (flow-through tests 8% leaded-brass meters)

Twenty leaded-brass Hersey Model 430 water meters were purchased locally in Asheville, NC. Three meters were selected randomly and assayed for lead on a small slice from the meter exterior with results ranging from 7.59 to 8.44% lead. Meters were randomly assigned to one of seven types of water and hooked up to a plumbing manifold consisting of three meters for each of six waters and two meters for one water. Connected plumbing included a Flojet Model 2100-953-115 plastic vacuum pump and a 100 L Nalgene laboratory carboy (see Fig. 1).

Each manifold system was conditioned by flushing 350 mL of a 1.0 ppm CL/DI water solution through all the meters about

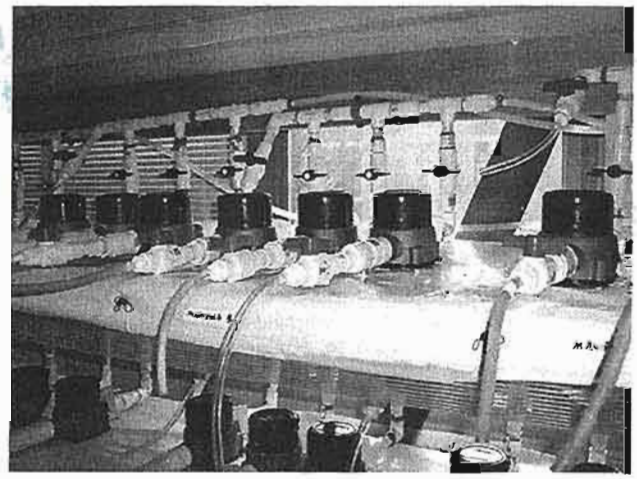

Fig. 1. Picture of Phase II setup.

15 times each weekday for 2.5 weeks for a total of approximately 190 times. One additional week of conditioning was completed by flushing 350 mL of the CL/DI water through the meters another 63 times. For this final week, samples were taken on Wednesday, Thursday, and Friday mornings at approximately 8:30 a.m. The late afternoon before each sample was taken a 2-L flush was completed at 4:30 p.m. and the meters sat undisturbed overnight 16 h until the morning sample at 8:30 a.m. Conditioning provided stagnation water lead data for 1 ppm CL prior to switching to test waters. After conditioning, the following waters with 2.0 ppm of each constituent were studied: (1) CA + FSA; (2) CA + 100% excess NH + FSA; (3) CA + 100% excess NH; (4) CA + 100% excess NH + NaF; (5) CL + FSA; (6) CL + NaF; and (7) CL alone. The target pH for all waters was 7.5 with an acceptable range of 7.3–7.7. The 100% excess NH was used as a potential worst-case scenario simulating a situation that might reasonably occur from time to time in a water plant. The CL, CA, FSA, NH, and pH were adjusted using the same methodology as that of Phase I.

Three meters were tested for each water composition, except the CA + FSA combination which was tested with two meters. For 6 weeks the plumbing manifold systems were flushed four times each weekday. Each meter had 1 L of its respective test water flushed through it three times, and the final flush of each day was 2 L. Sampling occurred each week for the 6-week sampling period on Tuesday, Wednesday, and Thursday mornings following a 16-h stagnation period.

3.4. Phase II statistical analyses

To assure that no meter was used that might be particularly susceptible to corrosion, an ANOVA analysis was performed on the log-transformed lead concentrations for the samples taken during conditioning. Median values of stagnation water lead concentration were found for each set of meters and combination of day and water composition. As with the elbow data, the natural logarithm was taken for each lead concentration. Lead concentrations for each meter were averaged over

the first 3 and last 3 weeks of the study. These arithmetic means were log transformed to give a single value to each meter for each of the two periods.

ANOVA analyses were performed on these log-transformed means and individual 95% confidence intervals for the log-transformed means calculated. An inverse transform was applied to these values to estimate the typical mean stagnation water lead concentration produced by the given water over the given time periods. ANOVA data for each of the pre-treatment period, first 3 treatment weeks, and last 3 weeks provided no evidence of significant non-normality (Kolmogorov–Smirnov p-values = 0.079, >0.15, >0.15, respectively) or heterogeneity of variances (Levene p-values = 0.962, 0.475, 0.218, respectively).

4. Results

4.1. Phase 1 results (2% lead elbows)

For the first set of bottle tests pH, water treatment, and their interactive effect were all significantly related to log lead concentration (p-values = <0.001, <0.001, 0.006, respectively). Fig. 2 displays the 95% confidence intervals for medians of the first set of static bottle tests. As seen from the results of the Tukey's LSD (Table 1) and individual confidence intervals (Fig. 2), lead concentrations are significantly higher at pH 7 for the CL and the CA waters, but not for the other two. The highest lead concentration was produced by CA + FSA under both pHs. CA at pH 7 produced the next highest lead concentration, but not significantly less than the highest. Absent a factorial design it was not possible to test formally for an interactive effect of CA + FSA. FSA alone gave results very similar to CL. Although CA + FSA produced the highest lead concentrations at both pHs, the combination was significantly higher than CA alone only at pH 8. Thus, bottle test 1 provides evidence, albeit not compelling, for a positive interactive effect of CA + FSA on leaded-brass corrosion.

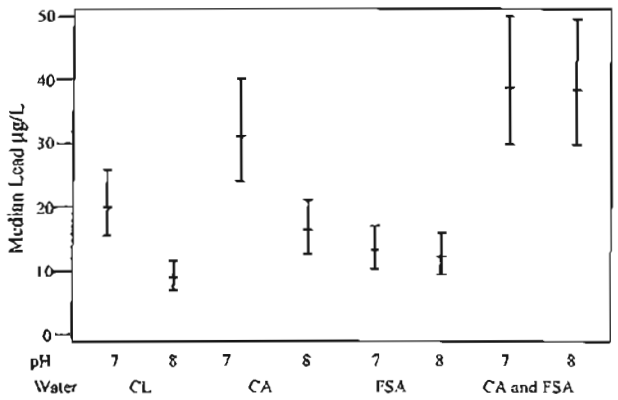

Fig. 2. Median 95% confidence intervals for bottle test 1 at pH 7 and pH 8.

Table 1
Estimated median lead concentrations for bottle test 1

	Water							
	Cl	FSA	FSA	CA	Cl	CA	CA + FSA	CA + FSA
PH	8	8	7	8	7	7	8	7
Estimated median	9.0	12.3	13.3	16.3	20.0	31.1	38.4	38.7

Combinations covered by the same line are not significantly different using Tukey's LSD statistic with a significance level of $\alpha = 0.05$.

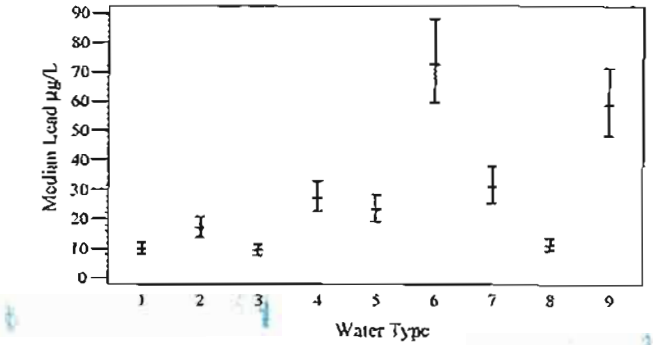

Fig. 3. Median 95% confidence intervals for bottle test 2. 1, Deionized Water; 2, CL Only; 3, CA Only; 4, CA and 50% excess NH; 5, CA and FSA 6, CA,50% excess NH, and FSA; 7, CA with 26% FSA Added; 8, FSA NH; 9, CA with concentrated ammonium fluorosilicate.

For the second set of bottle tests, water composition was the only factor having a significant effect on log lead concentrations ($p = 0.000$). Estimated median and confidence intervals for the median lead concentration of each water is displayed in Fig. 3. From the individual confidence intervals (Fig. 3) and Tukey's LSD results (Table 2) it can be seen that CA + 50% excess NH + FSA (#6) and CA + ammonium fluosilicate (#9) produced the two highest lead concentrations. CA + 50% excess NH; CA + FSA; CA + concentrated FSA produced intermediate concentrations. CL alone, CA alone, DI water alone, and the combination of NH + FSA produced the lowest concentrations. Unlike the first set of bottle tests at pH 7, CA alone produced significantly lower lead concentration than CL alone. CA + concentrated FSA produced a higher concentration than CA + pre-diluted FSA but the difference was not statistically significant.

Table 2
Estimated median lead concentrations for bottle test 2

Water code	Estimated median
3	9.4
1	9.9
8	11.1
2	17.0
5	23.3
4	27.2
7	30.8
9	58.8
6	72.3

Combinations covered by the same line are not significantly different using Tukey's LSD statistic with a significance level of $\alpha = 0.05$.
1, deionized water; 2, CL only; 3, CA only; 4, CA and 50% excess NH; 5, CA and FSA; 6, CA + 50% excess NH + FSA; 7, CA with 26% FSA added; 8, FSA + NH; 9, CA + concentrated ammonium fluosilicate.

The interactive effects of CA and FSA on lead leaching cannot be directly examined from the set of waters in the second set of bottle tests. However, effects of excess NH, FSA and their interactive effect can be evaluated for the CA waters. The four conditions: (3) CA only; (4) CA + 50% excess NH; (5) CA + FSA; and (6) CA + 50% excess NH + FSA make up a two-way full factorial experimental design. ANOVA analysis on just those four conditions found that in the presence of CA, both 50% excess NH ($p = 0.000$) and FSA ($p = 0.000$) were positively related to log lead concentration, but their interactive effect ($p = 0.662$) was not significant. Thus, in the presence of CA, 50% excess NH and FSA had positive additive effects on log lead concentrations in the second bottle study.

Median lead concentration for CA + 50% excess NH + FSA was greater than for CA + ammonium fluosilicate, but not significantly so. Thus, it seems reasonable to believe that CA + 50% excess NH + FSA leaches lead through a mechanism similar to that for CA with ammonium fluosilicate as such.

4.2. Phase II results (8% leaded-brass meters)

ANOVA analysis of the log-transformed lead concentrations during exposure to conditioning water (1.0 ppm CL/DI water solution) found no significant differences in the median lead concentrations between the groups of meters selected for the seven different water treatments (p-value = 0.771). The estimated median lead concentration for meters exposed only to the conditioning regime was 84.0 μg/L.

On Day 13 after treatment began, a meter receiving CL + FSA was reported to have a stagnation water lead concentration of 2.9 ppb, while the other two meters had values of 49.5 and 62.4. The 2.9 outlier was not included in any analyses. The median ($n = 3$, except for CA + FSA where $n = 2$) lead concentrations for each day were obtained for each water chemistry. Fig. 4 displays the median lead concentrations for the CA-type waters over the 28 days of testing. Fig. 5 displays the median lead concentration for the CL-type waters over the 28 days.

The median range in lead concentrations for the three (or, in one case two) meters subjected to the same waters for the entire study period (not displayed on the figure) was 9.0 μg/L over all days and waters. Some day-water sets had much higher meter-to-meter ranges. For example, days 1–3 for CL + FSA water had ranges of 115.5, 217.3, and 136.4 μg/L, respectively, but these were measured for water lead data in the 1000 ppb regime. As expected for data that is approximately log-normally distributed, day-water combinations with larger medians tended to also have larger ranges.

Figs. 6 and 7 illustrate the variability of the stagnation water lead concentrations produced over the entire test period by meters receiving, respectively, CA + 100% extra NH and meters

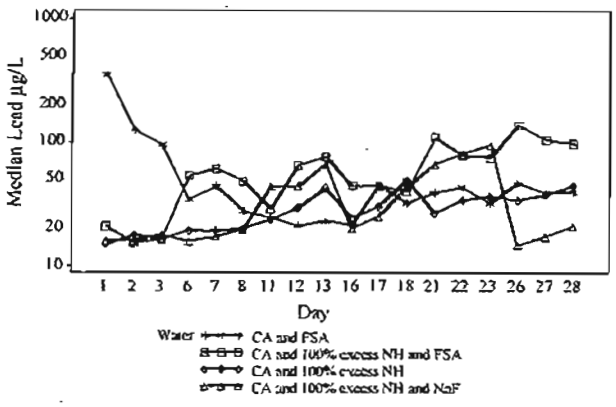

Fig. 4. Mean lead concentrations for the CA-based waters.

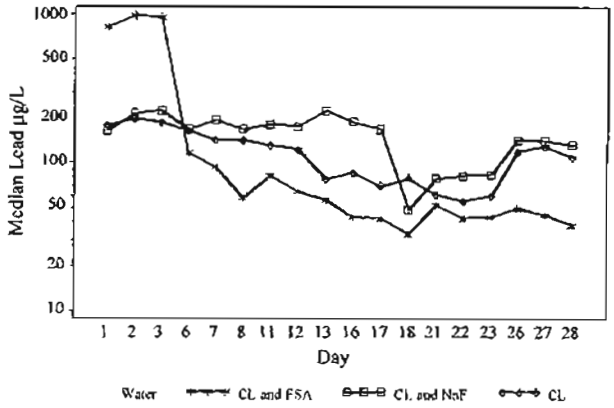

Fig. 5. Mean lead concentrations for CL waters by day.

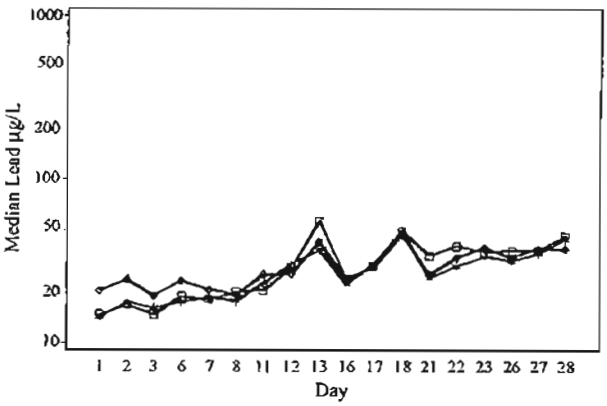

Fig. 6. Lead concentrations for meters using CA + 100% excess NH.

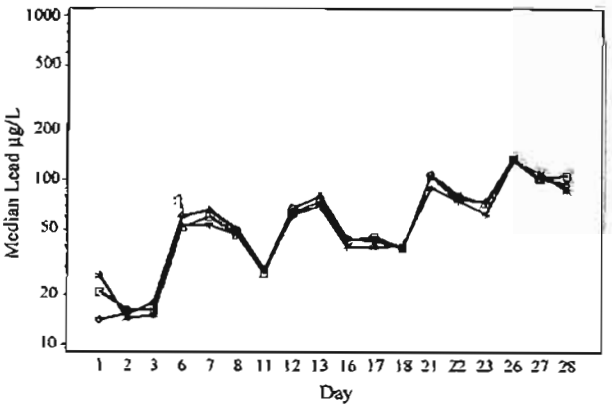

Fig. 7. Lead concentrations for meters using CA + 100% excess NH + FSA.

Table 3
Estimated median lead concentrations of meters by type of water averaged over day for the first 3 weeks of the study

Water	N meters	Estimated median (95% confidence interval)
CA and 100% extra NH	3	23.3 (21.0–25.9)
CA, 100% extra NH and NaF	3	28.1 (25.3–31.2)
CA, 100% extra NH and FSA	3	42.6 (38.4–47.3)
CA and FSA	2	83.1 (73.1–94.5)
CL	3	145.9 (131.4–162.0)
CL and NaF	3	185.3 (166.6–205.7)
CL and FSA	3	362.8 (326.7–402.9)

Water types that are covered by the same line are not significantly different from each other using Tukey's LSD with a significance level of $\alpha = 0.05$.

receiving CA + 100% extra NH + FSA. Clearly, for both treatment waters there is very little meter-to-meter variation in eluted lead on any one day. Such consistency is reasonable assurance that all the data reflect real effects, not merely random "chemical noise." Based on the premise that observed differences in stagnation water lead are not statistical aberrations, but due to explicable causes, day-to-day and week-to-week variability requires comment, which will be provided in the following section.

In the analyses shown in Figs. 4–7 a "week" is 5 days, since flushing occurred only on weekdays, and thus essentially no aging was considered to be occurring over weekends. Thus, Days 1–28 as shown in these figures represent 6 weeks of sampling on 3 successive days. Table 3 shows the median lead concentrations for all seven water chemistries averaged over the first 3 (5-day) weeks and Table 4 shows the corresponding results for the last 3 weeks of the study.

Fig. 4 and Tables 3 and 4 provide insights into corrosivity of CA-based test waters. The most corrosive of the CA-based test waters for the first 3 weeks was CA + FSA, while CA + excess NH + FSA was the most corrosive for the last 3 weeks and the only water showing a strong trend of increasing corrosivity over the whole experiment. Water with CA + excess NH and water with CA + excess NH + NaF were similar to one another in corrosivity which was significantly lower than the other waters. CA + excess NH + NaF displayed more variability over time than the other CA-based waters.

Fig. 4 and Tables 3 and 4 indicate that the water with CL + FSA was most corrosive for the first 3 weeks and CL + NaF was most corrosive for the last 3 weeks and second most corrosive for the first three weeks. Water with CL only was third most corrosive for the first 3 weeks and second most corrosive for the last 3. Water with CL + FSA showed a decreasing trend over the course of the experiment, while the other two CL-based waters displayed irregular, slightly decreasing trends.

Table 4
Estimated median lead concentrations of meters by type of water averaged over day for the last 3 weeks of the study

Water	N meters	Estimated median (95% confidence interval)
CA and 100% extra NH	3	34.8 (31.9–37.9)
CA and FSA	2	36.8 (33.1–40.9)
CA, 100% extra NH and NaF	3	43.2 (39.6–47.1)
CL and FSA	3	43.3 (39.7–47.2)
CA, 100% extra NH and FSA	3	79.2 (72.6–86.3)
CL	3	84.0 (77.0–91.6)
CL and NaF	3	115.2 (106.1–126.1)

Water types that are covered by the same line are not significantly different from each other using Tukey's LSD with a significance level of $\alpha = 0.05$.

Comparing the CL-based waters to CA-based waters, CL-based waters were most corrosive over the first 3 weeks of the study and three of the four most corrosive over the last three. This is in contrast to the results of Phase I in which the CA-based waters tended to be associated with greater stagnation water concentrations. However, it should be borne in mind that Phase I elbows were only 2% lead while Phase II meters were 8% lead. The possible effect of this difference on water lead will be discussed below along with comments about why in Phase II the highest lead level was a spike to over 1000 ppb extracted by FSA + 2 ppm CL water after initial meter conditioning with 1 ppm CL water.

5. Discussion of findings

5.1. Consistency of test results

Meter-to-meter difference in stagnation water lead values was very low on any one day and for virtually all test waters. The few instances where above median meter-to-meter variation was found, the median lead values were also on the high side. In other words, meter-to-meter differences in water lead values were about the same percent of median water lead values for most days and water formulas.

Day-to-day lead values within any week were often consistent or with a trend up or down. Such trends might be explained by loss of volatiles from a given batch of water without make-up under laboratory conditions that would not occur in a water plant where composition is in constant make-up mode. For example, excess NH alone might gradually decline, with one effect on lead extraction and excess CL alone might decline with the same or another effect. Given the fact that CL and NH combine to form chloramine, neither NH nor CL would be lost, which would have its own effect.

On the other hand, notable shifts in lead extraction occurred when a fresh batch of treatment water was prepared, typically between weeks. The most extreme shift in the entire experiment occurred when meters were first exposed to CL + FSA after having been conditioned to 1% CL/DI water. Along with the 17 other meters, the lead released by this group of 3 during the conditioning process had reached around 100 ppb. The first day these meters were exposed to CL + FSA following meter conditioning, lead concentration leaped to 800 ppb and increased to over 1000 ppb by the third day.

The second week batch of FSA + CL water was made up with the same FSA composition as the first batch, but the CL charge was different, in that sodium hypochlorite solution had been adjusted to compensate for change in the CL stock solution over time. The first day of the second week, water lead was down to 100 ppb, the same level as that at the end of the conditioning period. Thereafter, lead levels dropped in the

next 2 weeks and settled into a consistent 50 ppb day-to-day and week-to-week.

A crucial fact about the make-ups of the first and second FSA + CL batches is that the first had a pH of 7.56 with 1 g sodium bicarbonate added while the second had a pH of 7.30 with 6 g of sodium bicarbonate added. It is doubtful that this pH difference accounts for first week lead starting on day 1 at 800 ppb, increasing to over 1000 by the third day. The fact that one sixth the amount of base in batch 1 than in batch 2 produced a higher pH in batch 1 than that of batch 2 suggests fluosilicate dissociation status was not the same for both batches.

There also could have been serious error in batch preparation or analytical technique. But these explanations are at odds with the consistency of lead extraction measured during conditioning and very low meter-to-meter variation illustrated in Figs. 6 and 7. Neither human error in batch preparation, nor flaws in instrument performance can account for the high first day lead and upward trend that followed. A better explanation is that the combined action of CL and FSA in the first water batch started out very efficient and improved in the next 2 days. This would be consistent with release of particulate lead from the brass alloy in the first week leaving the remaining potentially mobilizable lead shielded from corrosive attack.

A similar, but less dramatic, effect was observed when FSA was added to CA with excess NH. After settling at the 100 ppb level during conditioning, the first day of exposure the test water produced a stagnation lead level of 350 ppb. Then, in this case, without any change in batch formula, after the first-day 350 ppb spike, the second day lead was 120 ppb, and the third day 100 ppb. Thereafter, for 5 successive weeks, with new batches each week, stagnation lead settled down to a very consistent 40–50 ppb.

On an obviously different scale, the same sensitivity of leaded-brass to corrosion by FSA + CL or FSA + CA can be expected in a water plant. It may not be observed when very tight controls are kept on treatment chemical compositions, but the results reported here are very much like what was found in the DC experience.

Considering the several different additives used in the plant, it is a forgone conclusion that deviations from an ideal dosage of any one additive are inevitable. The important data in this report should, therefore, be treated as providing reasonable confidence, not absolute proof of what would actually occur in a water plant.

6. Conclusions

In the "fluoridation debate" proponents frequently argue that the 1 or 2 ppm of fluoride in drinking water is so trivial that it cannot be a health danger. When one translates the ppm involved into molar concentrations, 2 ppm of fluoride is about twice the concentration of 2 ppm of chloride.

Ironically, the switch from CL to CA for disinfection that was made for health reasons, may have created a high water lead health problem. Published evidence has shown that chloramine used instead of chlorine for water disinfection enhances lead extraction from leaded-brass plumbing devices and faucets. Prior to the present study, no one had looked at brass corrosion by combinations of either chlorine or chloramine with water fluoridating agents. Several factors applicable to such combinations can produce more corrosion than either of the disinfectants or fluoridating agents alone.

One such factor is that fluosilicic acid, the most widely used fluoridating agent, is a good solvent for lead. Another is that chlorine, ammonia, and chloramine are all hostile to copper in that they induce copper stress cracking and/or can dissolve it. A third factor is that ammonia added to chlorine to produce chloramine will also react with fluosilicic acid to produce ammonium fluosilicate, an established solvent for copper alloys.

Besides these chemical factors, the lead in brass is present as nodules, so that any attack on the copper matrix of brass would render particulate lead readily accessible for mobilization. Whatever the exact mechanism may be for the combined effect of CA and fluoridating agents on increased levels of waterborne lead, the fact is that SiFs (FSA and NaFSA), commonly used to fluoridate water, have been associated with elevated blood lead levels in children (Coplan et al., in press; Masters et al., 2000). In a related sense, it was recently found that the North Carolina water systems that use FSA and chloramine are associated with elevated blood lead levels in children (Allegood, 2005; Clabby, 2006; Miranda et al., 2006). EPA has claimed a year-long evaluation they conducted did not find a national problem comparable to that in DC, but EPA also acknowledged the need to update specific areas of the LCR and guidance materials (USEPA, 2006). That ought to include lead from brass (Dudi et al., 2005; Renner, 2006) (see Coplan et al., in press).

Acknowledgment

This paper is dedicated to Dr. Richard P. Maas 1952–2005.

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Impact of fluorosilicate compounds on lead levels in drinking water and on water distribution infrastructure

Petition: No. 245

Issue(s): Environmental assessment, fisheries, human health/environmental health, toxic substances, and water

Petitioner(s): Environmental Training Institute

Date Received: 2 May 2008

Status: Completed

Summary: The petitioner seeks responses from several departments on potential health concerns related to increased levels of lead in drinking water due to fluoridation. In addition, the petitioner alleges that fluorosilicates have a detrimental effect on water distribution infrastructure and asks whether the government has carried out related cost assessments.

Federal Departments Responsible for Reply: Environment Canada, Department of Finance Canada, Fisheries and Oceans Canada, Health Canada, Public Health Agency of Canada, Public Works and Government Services Canada, Treasury Board of Canada Secretariat

Petition

Petition under Section 22 of the *Auditor General Act* for the discontinuation of the addition of toxic substances to our drinking water (inorganic fluorides, inorganic arsenic, lead)
In violation of the *Fisheries Act*, section 34(1), which describe the provisions to conserve and protect fish habitat that sustain Canada's fisheries resources, the harmful alteration, section 35(1), which prohibits the harmful alteration, disruption or destruction (HADD) of fish habitat, and sections 36-42 which control the deposition of any deleterious substance to water frequented by fish

Fluorosilicate Compounds Increase Drinking Water Lead Levels, Hence Source Water Contamination.

[Original signed by Peter L.D. Van Caulart]

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Introduction

Background levels of fluoride for Lake Ontario and the St. Lawrence River are up to 0.25mg/L that is double the 0.12mg/L Canadian Water Quality Guideline (CWQG). DWSP http://www.ene.gov.on.ca/envision/water/dwsp/0002/eastern/eastern.htm

Evidence from the study by Daemker and Dey 1989 indicates that some species of fish (salmon) are harmed at levels of about 0.25mg/L.[10] Other studies that indicate that fluoride at levels below 1.5 ppm have lethal and other adverse effects on fish. Delayed hatching of rainbow trout have occurred at 1.5 ppm; [11] brown mussels have died at 1.4 ppm[12]; an alga (Porphyria tenera) was killed by a four-hour fumigation with fluoride with a critical concentration of 0.9 ppm[13]; and, levels below 0.1 ppm were shown to be lethal to the water flea, Daphnia magna.[14] These latter two studies suggest that salmon species also may be affected by fluoride-induced reduction of food supply.

Documents used in a 1961 court case involving Meader's Trout farm in Pocatello, Idaho,[15] contain evidence that between 1949 and 1950 trout damage and loss was related to fluoride contamination due to rain washing airborne particles from leaves into hatchery water at levels as low as 0.5 ppm.

Because, in soft waters with low ionic content, a fluoride concentration as low as 0.5mg/L can adversely affect invertebrates and fishes, safe levels below this fluoride concentration are recommended in order to protect freshwater animals from fluoride pollution.[16]

Such demonstrated harm of aquatic species is in violation of the Fisheries Act. The addition of toxic substances (Na_2SiF_6 and H_2SiF_6 and co-contaminants arsenic, lead, mercury etc.) into our drinking water, hence source water is harmful and not sustainable.

Fluorosilicates Increase Blood Lead Levels

The actual products used in 90% of artificial water fluoridation sites are fluorosilicates that are derived from the phosphate mining industry. These products (Na_2SiF_6, H_2SiF_6) contain trace amounts of arsenic, lead and other contaminants. The **direct and indirect contribution of the drinking water contaminant lead** from fluorosilicates and their contribution to independent health effects are assessed below.

Direct additive: Lead is the second most common co-contaminant with the silicofluorides used in water fluoridation; "The second most common contaminant found..." (NSF Fluoride Fact Sheet on Fluoridation Chemicals)

Chlorine → Chloramine

A switch from chlorine to chloramine [ammonia + chlorine] was recently recommended and adopted in some water systems for several reasons:[1]

- Chloramine is cheaper than other disinfection methods
- Easy to add ammonium to already-chlorinated water
- Chloramine produced fewer disinfection by-products [DBPs] than chlorine

Fluorosilicates &/or Chloramine + Lead or Leaded Brass = Increased Blood Levels

Two new studies[1-3] demonstrate that fluoride in various combinations with chlorinating chemicals (e.g. chlorine or chloramine) increases the release of lead from leaded brass fittings used in water pipes. There are several chemical reasons[3];

1. fluosilicic acid, the most widely used fluoridating agent, is a good solvent for lead.
2. chlorine, ammonia, and chloramine are all hostile to copper in that they induce copper stress cracking and/or can dissolve it.
3. ammonia added to chlorine to produce chloramine will also react with fluosilicic acid to produce ammonium fluosilicate, an established solvent for copper alloys/brass.

Besides these chemical factors, the lead in brass is present as nodules, so that any attack on the copper matrix of brass would make lead particles readily accessible for mobilization[3].

Drinking Water is an important source of increased lead. Increased blood levels were found in homes without lead service lines or lead soldered copper piping. The only possible lead source had to be leaded-brass plumbing and/or brass faucets.

Silicofluoride use is associated with 2 neurotoxic effects[1]:

1. Prevalence of children with elevated blood lead (PbB>10µg/dL) is about double that in non-fluoridated communities;
2. Voluntary and involuntary muscle action is stimulated by **acetylcholine** (ACh) that is cleaved by the enzyme **acetylcholinesterase** (AChE) to end the stimulation. Without AChE, muscle excitation would persist as spasm with potentially lethal effect, as caused by a nerve gas. Acetylcholine modulated by acetylcholinesterase also induces saliva flow. Intense salivation is a symptom of fluoride poisoning. Silicofluorides inhibit AChE.

"It is proposed here that SiFW [silicofluorides in water] induces protein mis-folding via a mechanism that would affect polypeptides in general, and explain dental fluorosis, a tooth enamel defect that is not merely "cosmetic" but a "canary in the mine" foretelling other adverse, albeit subtle, health and behavioral effects."

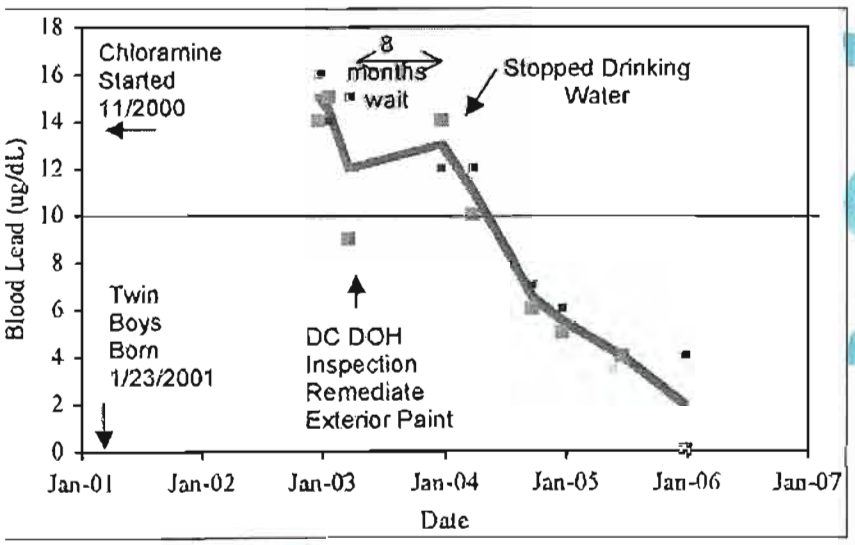
Indirect additive: "A contaminant that is extracted into drinking water through contact with surfaces of materials or products used for drinking water treatment, storage, transmission, or distribution." (NSF/ANSI Standard 60 2.10, P 3)

A growing body of research suggests that the practice of fluoridation may double the exposure of lead in our children from drinking water in two important ways:

1. **Direct additive**: Lead is the second most common contaminant found in the silicofluoride products used in >95% of water fluoridation facilities in the US.
2. **Indirect additive**: Lead is now known to leach from lead pipes, lead solder and leaded brass by mechanical and chemical interactions of fluorosilicates and/or chloramine (see below).

The University of North Carolina Environmental Quality Institute (EQI) has found that lead-bearing brass plumbing in the absence of any other source such as lead pipes is corroded by SiF treated water to such an extent that it should be considered as a serious source of ingested lead. (Contact Myron Coplan P.E., Intellequity Technology Services; Telephone: 508 653-6147)

A statistically significant association between the use of silicofluorides as water fluoridation agents (in both Massachusetts and New York State) and an increased uptake of lead into children's blood[4,5] was previously demonstrated.



Blood lead data for twin boys born in 2000.

"After the levels were found to be higher than CDC's [Centers for Disease Control] level of concern, DOH [department of health] inspected their home for lead paint and found only a remote exterior door as an admittedly unlikely source. However, painting the door failed to bring down the boys' blood lead levels. But when they stopped drinking tap water and it was no longer used in cooking their meals, the boys' blood lead levels declined rapidly."[8]

Sodium Fluoride → Silicofluorides

Silicofluorides were substituted for sodium fluoride in 1947 and endorsed in 1950 by the US Public Health Service without prior animal testing because rats teeth got as much fluoride as from sodium fluoride, and a community could save 4 cents per year per resident (McClure, 1950).[1]

Fluorosilicates shortens lifetime of water distribution infrastructure. Fluoride is the most corrosive of all known elements. (Merck Index) Fluorosilicates cause the following problems:

1. **leach lead from brass/copper fittings** (Coplan 2007, Maas 2007, Masters 1999, 2000, NRC 2006 Report on Fluorides in Drinking Water)
2. **leach lead from lead pipes and lead solder - cast iron (CI), ductile iron (DI)** piping
3. **antagonistic to the Asbestos Cement pipe matrix** used in transmission watermains, hastening decay of this important infrastructure. (IAOMT p24)
4. **corrodes stainless steel, nickel, ceramic and glass.**
5. **acidifies water, creating a need for neutralizing agents** such as lime to increase pH and restore lost alkalinity.

Quicklime is calcium oxide (CaO) and is made by heating limestone. It's the cheapest form of lime. To use quicklime it must be **slaked by adding water and allowing the insolubles to precipitate, leaving the limewater to be used for water treatment coagulation, softening or raising pH.** The slaking process is labour intensive and dirty. Quicklime must be stored in sealed silo hoppers, onsite. It's a cheap ingredient that requires a high capital cost for storage and slaking equipment, plus an ongoing operational labour cost to run the system. http://www.carmeusena.com/Markets/faqs.asp?indid=4#3

Liquid Sodium hydroxide NaOH may also be used to raise pH. It is simple to use, feed directly to pump, easier to store, but it is 5x higher price. It is used in small communities because they do not have the human resources to deal with lime sludge/limewater separation.

In **San Diego a new stainless steel HFSA vat** was destroyed within weeks of installation "After waiting four years to complete billions of dollars of improvements at five water treatment plants, the Metropolitan Water District had expected to start fluoridating in October."..."Metropolitan spokesman Bob Muir said Wednesday the latest delay came after the agency's staff discovered the galvanized steel it planned to use could corrode if it came in contact with the fluorosilicic acid that will fluoridate supplies." (Conaughton 2007)

1992 Tacoma, Washington had to shut down the fluoridation equipment due to the fact that **fluoride had eaten the pipes**. The municipal water had approximately 32 parts per billion (ppb) lead at the time of the breakdown. After the breakdown, the lead level dropped to 17 ppb. When the equipment was fixed, the lead level shot back up to 32 ppb. The city fathers decided to discontinue the use of fluoride, and the lead level again dropped. Over the next several years the lead level continued to drop, and today it is about 5 ppb. IAOMT p24-25

Thurmont, Maryland had an identical experience with fluoride raising lead levels in their municipal water system. IAOMT p25

One new study reports:[3] "Over the first test week with chlorine flushing, lead concentrations nearly doubled [from 100ppb to nearly 200ppb]. When fluorosilicic acid was added, lead concentrations spiked from 100ppb to over 900ppb. Lead concentrations from the chlorine-based waters appeared to be decreasing over the study period. Lead concentrations seemed to be increasing with the chlorine + ammonia + fluorosilicic acid combination."

Moss et al 1999[6] indicate that elevated blood lead levels may lead to increased cavities.

The quote below is from the following document:
Silicofluorides Should Not Be Added to Municipal Water Without Safety Testing Adequate to Protect Children and Other Vulnerable Populations *Resolution Submitted to American Public Health Association by Myron Coplan, P.E. & Dr. Robert Carton, Ph.D. 2001*

"Considering that data on 400,000 children in New York, Massachusetts, and in the NHANES III (National Health and Nutrition Examination Survey III) study, found that where local water is fluoridated with SiF's the prevalence of children with venous blood lead exceeding 10mcg/dL was significantly higher than in non-fluoridated areas with risk ratios of between 2.0 and 4.0 ($p<0.001$) controlling for race, housing age, poverty, congestion, and parental education);[40, 41] and

Recognizing that blood lead is believed responsible for adverse effects inflicted *in utero* such as impaired immune capacity,[42] brain damage and developmental problems,[43, 44, 45] as well as in early childhood,[46, 47, 48, 49, 50, 51] and into puberty/adolescence as impaired cognition and impulse control,[52, 53] and adulthood as nephropathy and hypertension,[54, 55] and into geriatric life;[56]"

"The atomic weight of lead being about ten times that of fluorine, for each ppm of silicon bound fluorine, 10 ppm of lead would be mobilized."[1]

Enzyme inhibition by SiF was also the subject of a German PhD thesis that focused on inhibiting acetylcholinesterase (AChE). AChE plays a vital role in proper functioning of cholinergic neural systems responsible for both voluntary and involuntary muscular processes. For instance, AChE quenches acetylcholine (ACh) activity after it has transmitted excitatory signals across a synaptic gap to a muscle end-plate. Without that quenching, muscle excitation would be prolonged, a spasm would occur that can be fatal...a shorthand description of how nerve gas works.[17]

Apart from direct adverse health problem from ingested SiF's, it should be noted that SiF treated water is a potential source of low-level internal radiation from contaminating radionuclides as a possible cause of osteosarcoma.

COSTS OF FLUOROSILICATES TO WATER DISTRIBUTION INFRASTRUCTURE

The Centers for Disease Control now states clearly that fluorides are ineffective at the levels currently used in Canada. ***"Fluoride's predominant effect is posteruptive and topical."*** *MMWR Weekly Report.* Vol. 50, No. RR-14, August 17, 2001, p. 4.

"Fluoride's caries-preventive properties initially were attributed to changes in enamel during tooth development because of the association between fluoride and cosmetic changes in enamel and a belief that fluoride incorporated into enamel during tooth development would result in a more acid-resistant mineral. However, laboratory and epidemiologic research suggests that fluoride prevents dental caries predominately after eruption of the tooth into the mouth, and its ***actions primarily are topical for both adults and children.*** *" MMWR Weekly Report.* 1999;48:933-940.

The CDC also states that the concentration of fluoride in drinking water is too low to have a topical effect. *"Studies have shown that* ***even a drop of 0.2 mg/L below the optimum (fluoride) level can reduce dental benefits significantly.*** *"* CDC Fluoridation Course 3017-G, pg. 8, Para. 3 According to the estimates by CDC, fluoride level for Ontario should be at least 1.2mg/L. According to CDC's own calculations, the concentrations (0.5-0.8mg/L) recommended by the Ontario MOE are ineffective.

Conclusions

The direct or indirect addition of lead from fluorosilicates is shown to cause measurable health harm. Lead is a known neurotoxicant. Lead has synergistic effects with chlorine/chloramine.

This evidence suggests that the "safe level" of fluoride in the fresh water habitat of susceptible species is 0.2 ppm (mg/L). The evidence suggests that artificial water fluoridation is harmful to humans and should be discontinued.

Questions

1. In legal circles when one product is advertised for use and then another product is used in its place, it is called "bait and switch". Please provide evidence that Health Canada or Public Health have informed the public that we are putting H_2SiF_6 or Na_2SiF_6 into drinking water and not "fluoride"? Provide evidence of such notices.
2. Does Environment Canada consider that removing fluoride compounds from air emissions (HF) to minimize pollution and adding them through drinking water, hence source water is a valid environmental solution to pollution? If so, explain how?
3. How has Health Canada or Environmental Canada communicated concern about increasing lead levels in drinking water, hence source water? If so, which federal agency will conduct research to determine the relative contributions of the mixtures of free chlorine or chloramine and fluorosilicates to our lead in drinking water?

4. What is Health Canada's current position with respect to the American Dental Association's explanation that fluoride works topically, not by swallowing (JADA Cover Story July 2000)? Agree or not?
5. Has Public Works or the Treasury Board made a cost assessment of how much the use of H_2SiF6 and Na_2SiF_6 are costing taxpayers in terms of infrastructure? Please provide estimates and the source of these estimates.
6. Australia and US governments are being sued for health harm caused by water fluoridation. What type of risk assessment has Health Canada or any other government agency done to assess potential liability on this issue for the government of Canada?
7. The CDC states: *"Studies have shown that even a drop of 0.2 mg/L below the optimum (fluoride) level can reduce dental benefits significantly."* CDC Fluoridation Course 3017-G, pg. 8, para. 3 According to the estimates by CDC, "optimal" fluoride levels for Ontario should be 1.2mg/L or higher. According to CDC's own calculations, the concentrations (0.5-0.8mg/L) recommended by Ont. MOE are ineffective. Why does Health Canada continue to promote this ineffective method of delivering this unregulated drug when even the Centers for Disease Control optimal dosage formula demonstrate that it is ineffective?
8. Does Health Canada still use the Galagan-Vermillion formula and the assumptions on which it was based (e.g. Galagan and Vermillion assumed that, on average, 44% of the American children's fluid intake was milk, which has negligible fluoride levels) to determine water fluoridation concentration guidelines? If so, please provide rationale and research evidence. If not, why? Galagan DJ, Vermillion JR, Nevitt GA, Stadt AM, Dart RE. Climate and fluid intake. Public Health Rep 1957;72:484–90.
9. Does Health Canada believe that 44% of Canadian children's fluid intake is milk? If so, why? Please provide rationale and research evidence. If not, why?
10. Does Health Canada contend that drinking water providers may compel the ingestion of, and dermal exposures to, fluoride through our addition of a direct water additive?
11. Which government agency is responsible for disclosing all sources of, and quantifying, potential and historically based exposures to fluoride?
12. Is the Department of Fisheries and Oceans aware that fluoridating communities adding the toxic compounds of hydrofluorosilicic acid, arsenic and lead to our fresh water and salt water systems are pushing background levels of fluoride above the CWQG? If so, what do they plan to do about it? If not, why?
13. Does the Department of Fisheries and Oceans believe that the discharge of municipal fluoridated effluents is a sustainable activity for fisheries? If so, why? If not, what are the Department's plans to halt it?
14. H_2SiF_6, Na_2SiF_6 are the primary agents used in >95% of water fluoridation schemes in the USA and assumably in Canada. These products are incorporated into the food chain through reconstituted beverages (fruit beverages, sodas) and food processing (cooking, washing). Does Health

Canada have any responsibility to prevent the sale of toxic substances, as defined by CEPA (inorganic fluorides such as H_2SiF_6, Na_2SiF_6 which are anthropogenic) for consumption? Does any other federal government agency (Environment Canada?) have a responsibility to prevent the sale of toxic substances in our food chain?

15. The Safe Drinking Water Act of Ontario, section 20, does not permit the addition of drinking water health hazards to our drinking water; dilution of drinking water health hazards is no defence. What proof Can Environment Canada or Health Canada or any other relevant government agency show that the toxic substances used in water fluoridation (H_2SiF_6, Na_2SiF_6) their complex silicate by-products and co-contaminants arsenic, lead (see Urbansky 2002, Coplan 2007, Smith, 1999) are not drinking water health hazards? (Urbansky concluded that hydroxo-fluoro SiF derivatives exist in drinking water. Coplan et al 2007 also demonstrates that many fluorosiliciates exist in drinking water H_2SiF_6, $H_2SiF_6 \cdot SiF_4$, Na_2SiF_6). Please provide references.
16. Does Health Canada, Environment Canada or any other relevant government agency disagree with the above evidence that fluorosilicates do not completely dissociate and may re-associate? If so, please provide rationale and complete references.
17. Can Health Canada, Environment Canada or any other relevant government agency prove that complete dissociation occurs despite the above evidence? If so, please provide references.
18. Which SINGLE peer-reviewed publication in an established scientific journal that establishes the SAFETY of either fluorosilicic acid or sodium silicofluoride (H_2SiF_6, Na_2SiF_6) for all individuals, over a lifetime of ingestion, using conventional animal studies of toxicology (and neurotoxicology) can Health Canada or any other relevant government agency provide as evidence of such?
19. Knowing that approximately 50% of the fluoride humans and animals ingest each day accumulates in their bones, what systematic and comprehensive attempts to measure fluoride in the bones of the Canadian population or fish species to see how close some individuals or fish species are getting to levels associated with pre-clinical, phase 1, phase II or phase III skeletal fluorosis <http://salsa.democracyinaction.org/dia/track.jsp?v=2&c=d7MRwdlM6uD1oIrwfLMfOElUQHKaz7Xd> , as well as levels associated with increased susceptibility to bone fractures <http://salsa.democracyinaction.org/dia/track.jsp?v=2&c=H1PPFrMCu0Jw2jIMpmf%2BK0lUQHKaz7Xd> in animal studies <http://salsa.democracyinaction.org/dia/track.jsp?v=2&c=88KRz7vDYWGIjuURme452klUQHKaz7Xd> and clinical trials <http://salsa.democracyinaction.org/dia/track.jsp?v=2&c=2kKMD1zB%2BTHq1ASGjcrjeUlUQHKaz7Xd> ?
20. What published studies have satisfied you that when a child has developed dental fluorosis that fluoride has caused no other damage <http://salsa.democracyinaction.org/dia/track.jsp?

v=2&c=1lvtZ6CYQhmMqAt8rVhVBUlUQHKaz7Xd> to the child's developing tissues?

Recommendations

Will Fisheries and Oceans recommend the discontinuation of this practice which is no longer considered effective in the treatment of cavities, but which is influencing the migration patterns and the destruction of some marine species such as Pacific Salmon, in violation of the Fisheries Act?

Will Public Works and Government Services Canada or Finance Canada recommend that a cost estimate of damage done to water infrastructures in cities that currently fluoridate?

Because litigation is now occurring in Australia and the USA regarding water fluoridation, will Public Works and Government Services Canada or Finance Canada recommend that a cost estimate of possible litigation on this issue be made?

Will Health Canada recommend that this practice of adding hydrofluorosilicic acid or sodium silicofluoride and associated contaminants to drinking water, hence source water, stop immediately? If not, why?

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FLUORIDE CLASS ACTION

by James Robert Deal, Attorney

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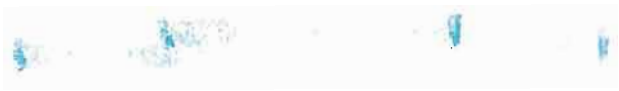

- Potentially Liable Parties

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COMMENTS TO HHS AND EPA
REGARDING LEAD, ARSENIC, AND WATER FLUORIDATION
Submitted April 19, 2011, Revised May 19, 2011

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Dear Ms. Sebelius and Ms. Jackson,

I am writing to give comments to HHS and EPA regarding their recent requests for comment on fluoridation.

To make it easier to follow links, read a web version of this letter at http://fluoride-class-action.com/hhs. Click on http://fluoride-class-action.com/hhs/comments-re-lead.

This letter is a supplement to my Fluoride Report Card for HHS and EPA. To read that letter click on http://fluoride-class-action.com/hhs/comments-re-lead.

I conclude that the research done by HHS and EPA on fluoridation is inadequate and that neither presented sufficient evidence to come to the conclusion that fluoridation should be continued but at a reduced level, that is at .7 ppm or that a new reference does of .08 mg of fluoride per kg of body weight should be set. I also conclude that HHS and EPA have been ignoring evidence that would force the conclusion that no fluoridation at any level should continue.

One of the most important lines of evidence that has been ignored is that which connects silicofluorides with increased lead in water.

Both requests for comment look back to the 2006 NRC Report, which suggested many topics which EPA should study in connection with drinking water fluoridation. The long list of topics included the following: caries, fluorosis, and bone fractures, fertility, thyroid function, increased calcitonin activity, increased parathyroid hormone activity, secondary hyperparathyroidism, impaired glucose tolerance, and possible effects on timing of sexual maturity, endocrine effects and brain function, osteosarcoma. See 2006 NRC Report, "Research Needs", pages 11-12, which mentions "brain function" as a needed area of research, and which points to lead which causes serious neurological harm. NRC also suggested on page 52-53 under "Fluorosilicates" that EPA do further research on the connection between silicofluorides and lead uptake:

> Most fluoride in drinking water is added in the form of fluosilicic acid (fluorosilicic acid, H2SiF6) or the sodium salt (sodium fluosilicate, Na2SiF6), collectively referred to as fluorosilicates (CDC 1993). Of approximately 10,000 fluoridated water systems included in the CDC's 1992 fluoridation census, 75% of them (accounting for 90% of the people served) used fluorosilicates. This widespread use of silicofluorides has raised concerns on at least two levels. First, some authors have reported an association between the use of silicofluorides in community water and elevated blood concentrations of lead in children (Masters and Coplan 1999; Masters et al. 2000); this association is attributed to increased uptake of lead (from whatever source) due to incompletely dissociated silicofluorides remaining in the drinking water (Masters and Coplan 1999; Masters et al. 2000) or to increased leaching of lead into drinking water in systems that use chloramines (instead of chlorine as a disinfectant) and silicofluorides (Allegood 2005; Clabby 2005; Maas et al. 2005).12,13
>
> Use of more sophisticated analytical techniques such as nuclear magnetic resonance has failed to detect any silicon- and fluorine-containing species other than hexafluorosilicate ion ($SiF6^{2-}$) (Urbansky 2002; Morris 2004). In drinking water at approximately neutral pH and typical fluoride concentrations, all the silicofluoride appears to be dissociated entirely to silicic acid (Si(OH)4), fluoride ion, and HF (Urbansky 2002; Morris 2004); any intermediate species either exist at extremely low concentrations or are highly transient. $SiF6^{2-}$ would be present only under conditions of low pH (pH < 5; Urbansky 2002; Morris 2004) and high fluoride concentration (above 16 mg/L according to Urbansky [2002]; at least 1 g/L to reach detectable levels of $SiF6^{2-}$, according to Morris [2004]). Urbansky (2002) also stated that the silica contribution from the fluoridating agent is usually trivial compared with native silica in the water; therefore, addition of any fluoridating agent (or the presence of natural fluoride) could result in the presence of $SiF6^{2-}$ in any water if other conditions (low pH and high total fluoride concentration) are met. Both Urbansky (2002) and Morris (2004) indicate that other substances in the water, especially metal cations, might form complexes with fluoride, which, depending on pH and other factors, could influence the amount of fluoride actually present as free fluoride ion. For example, P J. Jackson et al (2002) have calculated that at pH 7, in the presence of aluminum, 97.46% of a total fluoride concentration of 1 mg/L is present as fluoride ion, but at pH 6, only 21.35% of the total fluoride is present as fluoride ion, the rest being present in various aluminum fluoride species (primarily AlF2+ and AlF3). Calculations were not reported for pH <6.
>
> Further research should include analysis of the concentrations of fluoride and various fluoride species or complexes present in tap water, using a range of water samples (e.g., of different hardness and mineral content). In addition, given the expected presence of fluoride ion (from any fluoridation source) and silica (native to the water) in any fluoridated tap water, it would be useful to examine what happens when that tap water is used to make acidic beverages or products (commercially or in homes), especially fruit juice from concentrate, tea, and soft drinks. Although neither Urbansky (2002) nor Morris (2004) discusses such beverages, both indicate that at pH < 5, $SiF6^{2-}$ would be present, so it seems reasonable to expect that some $SiF6^{2-}$ would be present in acidic beverages but not in the tap water used to prepare the beverages. Consumption rates of these beverages are high for many people, and therefore the possibility of biological effects of $SiF6^{2-}$, as opposed to free fluoride ion, should be examined.

HHS and EPA avoided the silicofluoride and lead issue. They chose to do research only on caries, fluorosis, and brittle bones

NRC suggested that further research be done on the silicofluoride-lead issue, however, neither HHS nor EPA did such further research. They only addressed caries, fluorosis, and bone fractures. Nevertheless, HHS and EPA had the audacity to recommend that fluoridation be continued. HHS and EPA should immediately retract any

recommendation that fluoridation be continued.

Why have HHS and EPA avoided doing broad research on all topics pertaining to fluoridation? I can only speculate. The best hypothesis I can come up with is that they do not want to find what they would find if they did broad research. Their primary commitment is not to our health or pure water but to please the chemical industry which buys seats on the boards of our regulatory agencies through political donations to our representatives and senators.

A FOCUS ON LEAD IN PAINT—LEAD IN WATER IGNORED

Federal law mandates that water districts give lead notices. A water district, as

> owner or operator of a public water system .. shall identify and provide notice to persons that may be affected by lead contamination of their drinking water where such contamination results from . . lead content in the construction materials of the public water distribution system [or] corrosivity of the water supply sufficient to cause leaching of lead. . Notice under this paragraph shall be provided notwithstanding the absence of a violation of any national drinking water standard.[1] [emphasis added]

State boards of health have the responsibility under federal law to make rules pertaining to protecting state citizens, particularly children, from lead.

When the Washington Board of Health, for example, works on the lead issue, it focuses on avoiding lead in old paint [2] The Board of Health in its literature also mentions lead in brass plumbing fixtures, solder, and batteries, but goes nowhere with this aspect of lead avoidance. The "Lead Warning" card[3] distributed by the Washington Department of Health focuses almost entirely on lead in paint The section of "A Healthy Home" brochure[4] published by the Department of Health and which deals with lead focuses entirely on lead paint. Washington is typical of other states when it comes to lead disclosure requirements. Likewise, EPA efforts[5] to reduce exposure to lead focus on lead in paint. The EPA has failed to do its duty to see to it that states disclose the danger. I pointed out to the Washington Department of Health that it was failing to disclose lead problems related to water fluoridation

On both the state and federal levels the lead that enters our bodies through drinking water is ignored. I learned this when I went to a Washington Department of Health meeting on lead. The focus was on lead in paint and not on lead in water.

SDWA § 300j–24, entitled Lead contamination in school drinking water requires as follows:

> Within 9 months after October 31, 1988, each State shall establish a program, consistent with this section, to assist local educational agencies in testing for, and remedying, lead contamination in drinking water from coolers and from other sources of lead contamination at schools under the jurisdiction of such agencies.

This is another law which the EPA enforces only half heartedly.

The health issue is this: There is sometimes a small amount of lead in raw fluoridated drinking water. Fluorosilicates are being added to drinking water which contain lead at up to .6 ppb.[6] Fluorosilicates added to drinking water break down into ions, one of which is HF, which dissolves lead in pipes. Lead goes directly into the blood stream and passes all the bodies barriers, brain barrier, placental barrier, and perhaps the mammary barrier.

Like other states, Washington is failing to notify citizens of these lead issues. It fails even to look at lead in drinking water. The Washington Department of Health – like EPA, like CDC, like FDA – ignores the issue of lead in water.

Why do they ignore the lead issue? Because to look honestly at the lead issue would mean looking at the fluoridation materials that contain lead and leaches lead from brass plumbing. Because fluoridation is a sacred cow.

LEAD IN PLUMBING

Lead has long been added to almost all brass water pipes and pipe fittings[7] and to the solder used to solder brass and copper pipe. Lead has long been added to brass to soften it and lower its melting point.

The Wikipedia article on Tap Water contains this discussion under Lead Leaching:

Generally, copper tubes are soldered directly into copper or brass fittings, although compression, crimp, or flare fittings are also used. Formerly, concerns with copper supply tubes included the lead used in the solder at joints (50% tin and 50% lead). Some studies have shown significant "leaching" of the lead into the potable water stream, particularly after long periods of low usage, followed by peak demand periods. In hard water applications, shortly after installation, the interior of the pipes will be coated with the deposited minerals that had been dissolved in the water, and therefore the vast majority of exposed lead is prevented from entering the potable water. Building codes now require lead-free solder. Building Codes throughout the U.S. require the use of virtually "lead-free" (<.2% lead) solder or filler metals in plumbing fittings and appliances as well [8]

In 1977 we made lead based paint illegal.[9] In 1986 we made lead based inks illegal [10] Between 1976 and 1986 we phased out tetraethyl lead.[11] California has banned lead bullets[12] in areas where condors forage.

Newer water mains are lead free However, many older water mains are cast iron[13] and are generally soldered together with lead solder poured molten into forms set up around the joins Old cast iron water mains are common in many cities.[14] Concrete-asbestos water pipes[15] are used extensively, not only as sewer pipes but as drinking water mains[16] NaF and SiF dissolve concrete[17].

Even if there is no lead in water mains, things change when water gets to homes and businesses, where water encounters brass plumbing and fittings which contain lead, and copper pipe which is soldered with lead solder. Until recently, it was standard procedure to solder copper pipes together with solder containing 50 percent lead.[18]

In 1986 as part of the Safe Drinking Water Act.[19] the EPA required that all pipes and fittings that carry water be "lead free". However, the term "lead free" allowed water pipes and fittings to contain up to 8.0% lead and allowed solder[20] for use in plumbing to contain up to 0.2% lead, a standard which Washington follows.[21] Before 1986 water pipes were sometimes up to 30% lead. This means that we should carefully check lead levels[22] in water in old buildings, including old schools.

In 2010 California limited lead content[23] in brass pipes and fittings to a maximum of 0.25%, and in solder to 0.2%.[24] It is unfortunate that the EPA did not do the same back on a nationwide basis in 1986. The 8.0% lead level provides insufficient protection; HF – released as SiFs ionize in acid conditions – leaches the lead from pipes into our drinking water.

Many thousands have been harmed by lead[25] since 1986. We would hope that the EPA would follow California's lead and reduce lead levels in water pipes. Note, however, that limiting lead in new construction will not remove the already existing lead in plumbing in millions of homes, schools, apartment buildings, and businesses Something should be done to reduce the amount of lead consumed by children through their drinking water. The way to do that is to halt fluoridation using silicofluorides.

In 2004 the Seattle Post-Intelligencer reported that lead was showing up in water fountains in old Seattle schools, at levels up to 1,600 ppb[26], far above EPA's legally enforceable maximum contaminant level[27] (MCL) in effect at that time, which was 20 ppb. The MCL was recently reduced to 15 ppb.[28]

Two studies were done that I know of which discuss this incident, both written by Karr, Sathyanarayana, and others. The authors concluded in both their 2004[29] and 2006 [30] articles that the high lead levels were not a concern because children's blood lead levels were not higher than average, that the highest lead levels by far occurred in first draw water and dropped dramatically in running water, and the fact that the children only drank part of their water at school.

More important is the recommended maximum contaminant level goal[31] (MCLG) for lead, which is zero. Lead is a probable human carcinogen, so we should not do anything that adds lead to our water, causes lead to leach out of plumbing, or increases lead uptake or retention by the body.

Lead in pipes will often stay put relatively well and not dissolve into drinking water, particularly if the water is hard and contains a lot of calcium carbonate, which binds with lead and coats the inside of pipes and thus insulates the lead from the water. Ancient Rome declined in part because Romans were lead poisoned. Most presume that this was because their water pipes were lead. Yes, in areas outside of Rome where water came from melted snow and was soft. No, in Rome where water came from springs and was hard. It was not their pipes that poisoned the Romans; it was the lead acetate they used in copious amounts as a sweetener, which was produced by cooking grapes in lead pots to produce a must.

A problem arises when silicofluorides (SiFs) are added to water. SiFs ionize in such a way that they dissolve lead[32], as I will discuss below. Blood lead levels are higher in cities which fluoridate using SiFs.

This problem is most serious in cities such as Seattle which have soft water, snow melt water that is low in dissolved calcium and other minerals. Even the CDC admits that soft water is more prone[33] to be acidic and leach more lead because there is so little dissolved minerals in soft water to bind with the fluoride and reduce acidity. Thus, fluoride is freer to bind with lead in soft water. Seattle's snow melt water is considered very soft.

Elemental fluorine is the most electronegative of all elements, although it does not exist naturally. Elemental fluorine does not exist in nature because it reacts immediately with other substances to become fluoride, a negatively charged elemental anion

Fluoride aggressively seeks out other elements in the body and bonds with them strongly, especially with positive charged calcium and aluminum. It interacts with positively

charged lead ions. At acidic pH, hydrogen fluoride – an ionization breakdown product of SiF – dissolves lead.

Fluorine is the most powerful oxidizer, a more powerful oxidizer even than oxygen. Oxygen does not oxidize fluorine, fluorine oxidizes oxygen and forms OF_2, oxygen difluoride.

Fluoride is the most hydrogen bond disrupting of all the charged elements. Even a small amount will do damage. Fluoride can denature protein, and the extent to which it denatures protein depends on the concentration of the fluoride and on the sensitivity of the particular protein.

In people who drink water fluoridated at 1.0 ppm, blood fluoride level is around .2 ppm. Even at this low level fluoride affects the activity of many sensitive enzymes.[34] All protein, DNA, and cells need to be turned over or repaired or in some cases replicated, and they do this with the assistance of enzymes. Fluoride interferes with enzymatic activity in general and can disrupt protein, DNA, and cellular turnover, repair, and replication.

Proteins have H atoms sticking off their sides. Fluoride affects the hydrogen bonds of the protein H atoms with water, thus changing the shape of proteins or at high enough concentrations denaturing them.

It is not acceptable to say that 2 ppm fluoride is only a "small amount of fluoride" because that small amount will find hydrogen bonds in proteins to disrupt. F does not disappear when it is only present at 2 ppm. It is still there doing its mischief. It can alter the 3-dimensional shape of some proteins at even the smallest dose. Those who consume synthetic fluorides are taking a chance as to which proteins will be denatured and to what extent and how much abuse from fluoride the body can take.

Fluoride "loves" to bond with calcium. It is this bonding that prevents calcium fluoride from being labeled a poison -- although it is definitely sickening and can cause serious health problems over time.

Fluoride builds up in bone, which is where most of the calcium is. A person who has been drinking fluoridated water for a year may have around 2,500 ppm fluoride in his bones. After 20 years he may have from 4,000 ppm to 6,000 ppm, which will cause bone weakness or pain, or up to 10,000 ppm, which can render one an invalid. The fluoride level in bone will not much exceed 12,000 to 14,000 ppm, because at that level of fluoride, the bones are no longer able to release the normal surges of calcium necessary to supply the heart with the blood calcium ion which must be available to rush into heart cells each time the heart beats. Death by heart failure is likely to occur.

In a person quits drinking fluoridated water, the fluoride in blood and in soft tissues can be eliminated, but fluoride in bone is essentially permanent (NRC, 2006).

When lead is available, such as when fluoridated water sits in brass pipe overnight or through the weekend, and especially when calcium is not available to bind with and seal in the lead, fluoride dissolves and joins with lead, especially when the water is soft and acidic, and especially when the form of the fluoride is HF, hydrogen fluoride, which is formed when SiFs dissolve in water and ionize, as I will discuss below.

Fluoride is the supreme flux. The word "flux" itself derives from an old version of the name for fluoride. Fluxes such as fluoride make metals in general melt at lower temperatures.[35] Lead like fluoride lowers the melting point of the metals it is mixed with. Fluxes are also used in solder on the surface of metals being worked or welded to prevent surface oxidation and remove impurities.

In the past water pipes were used for electrical grounding. This accelerates lead corrosion and increases lead in drinking water.[36]

Further, silicofluorides attack PVC pipe,[37] causing the release of ammonia, which combines with chlorine to form chloramine, which is more aggressive than chlorine in dissolving lead in brass pipes, fittings, and solder. Fortunately Seattle, for example uses chlorine instead of chloramine, and we hope it will not follow the current trend of switching from chlorine to chloramine as a disinfectant.

SILICOFLUORIDES AS ACETYLCHOLINESTERASE INHIBITORS

There are many theories about how silicofluorides do us harm, and all of them may be true. HHS and EPA should explore all these theories; currently they explore none of them. This is an act of bad faith.

There is substantial evidence to support the theory that silicofluorides are acetylcholinesterase inhibitors. I paraphrase from an email sent to me by Dr. Roger D. Masters, who has done much work on silicofluorides, working as part of a team with Myron Coplan and others:

The fundamental problem with silicofluoride water treatment is that these compounds do NOT "dissociate" completely into component elements, as was assumed when the use of silicofluorides in place of sodium fluoride began around 1950. Hydrofluosilicic acid, H_2SiF_6, does not dissociate neatly into H+ Si + F. Westendorf (4th ed.) showed that a "residual species" of chemical remains which is biologically active. The residual is an acetylcholinesterase inhibitor, a fact which has mostly been ignored or unnoticed by mainstream fluoride scientists.

There are two effects of silicofluorides on brain chemistry. First, the action of acetylcholinesterase, the enzyme that breaks down acetylcholine, is blocked. Acetylcholine is a neurotransmitter which stimulates cellular activity. With acetylcholinesterase unable to do its work, activity once stimulated is hard deactivate, as in the case of ADHD. Second, silicofluoride residue has effects on the neurotransmitter dopamine (which is a central regulator of impulsiveness). Where silicofluorides are used, the combination of more activation and weaker inhibition results in statistically significant increases in behaviors where impulse control is essential: learning deficits, more substance abuse, and more violent crime.[38]

We have solid findings, in peer reviewed journals, that show lower scores on nine standardized tests in Massachusetts towns where drinking water is fluoridated with silicofluorides. Findings show higher rates of violent crime. The latter has been assessed using multivariate statistical analyses of up to twelve risk factors to predict county level violent crime rates for all 3141 US counties and then replicated for violent crime. These effects are related to the neurotoxicity of silicofluoride residues as well as lead. That is, where silicofluorides are used, the neurotransmitter dopamine does not function normally. The resulting behavioral problems cost American taxpayers billions of dollars. The National Toxicology Program[39] nominated silicofluorides for testing in 2003 on the ground [that] their toxicology wasn't known, and a decade of published data on harmful effects has never been contradicted. The EPA should immediately ban use of silicofluorides until such time as their safety has been demonstrated convincingly and contrary data explained.

In 2000 Masters, Coplan, and others published an article in NeuroToxicology,[40] a peer reviewed journal.[41] This article was expanded on in a 2001 article[42] and summarized in Dartmouth News.[43] The authors of the Dartmouth News article conclude that there is

evidence that public drinking water treated with sodium silicofluoride or fluosilicic acid, known as silicofluorides (SiFs), is linked to higher uptake of lead in children.

Sodium fluoride, first added to public drinking water in 1945, is now used in less than 10% of fluoridation systems nationwide.... Instead, [silicofluorides] are now used to treat drinking water delivered to 140 million people [including Seattle, Everett, and most Washington water systems].

Masters and . Coplan . studied the blood lead levels in over 400,000 children in three different samples. In each case, they found a significant link between [silicofluoride]-treated water and elevated blood lead levels. [Masters said:] 'We should stop using silicofluorides in our public water supply until we know what they do.' . The researchers found that the greatest likelihood of children having elevated blood lead levels occurs when they are exposed both to known risk factors, such as old house paint and lead in soil or water, and to [silicofluoride]-treated drinking water. [Masters said.] '[O]ur preliminary findings show correlations between SiF use and more behavior problems due to known effects of lead on brain chemistry.' Also requiring further examination is German research that shows [silicofluorides] inhibit cholinesterase, an enzyme that plays an important role in regulating neurotransmitters [Masters said.] 'If [silicofluorides] are cholinesterase inhibitors, this means that [silicofluorides] have effects like the chemical agents linked to Gulf War Syndrome, chronic fatigue syndrome and other puzzling conditions that plague millions of Americans... ' [Masters said:] '[T]his may well be the worst environmental poison since leaded gasoline.'

Masters added more detail in a letter he wrote June 17, 2001.[44]

In 2007 Masters, Coplan, and others published another article in NeuroToxicology,[45] in which they concluded:[46]

Silicofluorides ... are used to fluoridate over 90% of US fluoridated municipal water supplies [including Seattle's]. Living in communities with silicofluoride treated water... is associated with two neurotoxic effects.

(1) Prevalence of children with elevated blood lead ... is about double that in non-fluoridated communities .... [silicofluoride treated water] is associated with serious corrosion of lead-bearing brass plumbing, producing elevated water lead ... at the faucet. New data refute the long-prevailing belief that [lead in water] contributes little to children's blood lead.... [I]t it is likely to contribute 50% or more.

(2) [Silicofluoride treated water] has been shown to interfere with cholinergic function. . [Silicofluoride treated water] is a more powerful inhibitor of acetylcholinesterase than [water fluoridated with sodium fluoride, which was used when fluoridation first began in the 1950s].

Authors of another study published in NeuroToxicology reported:

This study concerns effects on water-borne lead from combinations of chlorine (CL) or chloramines (CA) with fluosilicic acid (FSA) or sodium fluoride (NaF). ... Water samples were taken for lead analysis three times per week after a 16-h stagnation period. . . [W]hen FSA was also included, lead concentrations spiked to over 900 ppb. Lead concentrations from the CL-based waters appeared to be decreasing over the study period, while for the CA+NH3+FSA combination, lead concentrations seemed to be increasing with time.[47]

THE SILICOFLUORIDE / HYDROGEN FLUORIDE LINK

Another convincing theory is that silicofluorides go through a chemical process that ends up increasing the amount of hydrogen fluoride, which in turn is especially good at dissolving lead in pipes.

Sodium fluoride, NaF, is a salt that forms alkaline or basic water. Silicofluorides such as H_2SiF_6, hexafluorosilicic acid, abbreviated here as SiF, are acids with low pH.

When SiF is used to fluoridate tap water, it breaks down into H^+, SiF_6^{2-} hexafluorosilicate anion, F-, and HF. The pH level is very low. To balance the acidity, the water company adds sodium hydroxide, NaOH, a strong base, aka Drano®. NaOH ionizes and forms Na+ and OH-. Na+ is a spectator ion and does not interact with other elements as long as it is in water. OH- combines with H+ to form water, thus reducing the H+ and neutralizing the pH. Without sodium hydroxide added, hydrogen fluoride HF will quickly dissolve water system equipment.

When a municipality fluoridates with tanker loads of silicofluorides, it must bring in tanker loads of NaOH to neutralize the low pH of the fluoride. More NaOH is needed in cities with soft, more acidic water – such as Seattle.

Workmen who handle these chemicals must wear hazardous materials suits for self-protection.

NaOH ionizes into Na+ and OH-, and the OH- can combine with metals in pipes such as iron and copper. It can also combine with aluminum, which the water district adds to precipitate dirt. As a result, there may not be sufficient OH- remaining to raise the pH of SiF fluoridated water at the time the water arrives at the point of use. Out in the street main pipe, the water pH may be neutral and low in HF. However, once the water is in the building plumbing, especially when the water sits in the pipes overnight or through the weekend, the water pH drops as OH- combines with iron, copper, and aluminum, and the HF dissolves lead and other metals. This negative pH plunge is more pronounced in soft, more acidic water because it has less calcium to bind with and neutralize fluoride. This is how lead levels in water in old Seattle schools got up to 1,600 ppb[48].

If you remember anything from this chemistry lesson, remember that fluoride loves calcium. It will make a bee line for wherever calcium is found in the body and be locked there permanently, in teeth, bones, pineal, and other areas.

When NaF ionizes in water, little HF is produced. But when SiF ionizes, HF is produced along with H^+, F- and $(SiF_6)^{2-}$. The lower the pH, the more HF is produced, which is especially effective at dissolving the lead out of brass plumbing.

NaOH is the line of defense. It's job is to keep pH neutral. NaOH is an unreliable defender. NaOH levels can drop if there is an NaOH underfeed. If there is a SiF overfeed, SiF will overwhelm the NaOH. Or NaOH can be lost when water reaches pipes and the OH- combines with iron in pipes to produce $Fe(OH)3$. It can combine with aluminum to make $Al(OH)3$. Aluminum is often added to water to precipitate dirt so it can be filtered out. It can combine with copper to form $CuOH_2$. With less OH- ion available to combine with H+ and form water and thus raise pH, there is more H+ available to join with F- to make HF.

As stated above, it is the HF which does the mischief in the pipes, dissolving the lead. Likewise, when SiF fluoridated water or SiF fluoridated soda pop or orange juice reconstituted with SiF fluoridated water or bread made with SiF fluoridated water reaches the stomach where the pH is low, more H+ binds to F- to form more HF, which is hard on stomach lining, especially for a person with a thin stomach lining.

HF is a small molecule which will burrow into and easily dissolve lead and other metals in pipes and easily burrow into and attack stomach lining. Some theorize that through this mechanism SiF causes stomach ulcers and stomach cancer.

At one atmosphere pressure HF has a boiling point of 19.5°C (67.1° F). As such, it is very tissue penetrating and causes severe, deep "burns" on contact with the skin or respiratory system. In water it is hydrofluoric acid, which is very corrosive and is able to etch glass.[49]

SiF dissociates almost completely in water at a pH of 7.4 or above. It forms silicic acid $Si(OH)4$ or its silicate anions after dissociation of H^+. The lower the pH, the less that SiF dissociates. While pH may be neutral out in the water main, in the old school or apartment building, with pipes that are 30% lead, the water may have a more acidic pH, depending on injected levels of NaOH with the SiF and depending on metals such iron, aluminum, and copper that can precipitate OH^- to varying degrees. In the stomach the pH is 3.0 to 5.0. In the small intestine the pH of stomach contents gradually increases along its initial length until it becomes basic as a result of the pancreatic release of bicarbonate. In the blood stream pH is 7.4, with the pH of arterial blood slightly lower than the pH of venous blood. In the gaps between synapses the pH is 7.4. Inside the cell the pH is 6.9. As the SiF travels about the body, pH changes and so SiF forms vary.

SiF scrubber liquor is composed of dozens of different constantly changing ions and compounds, depending on pH. Read what Wikipedia has to say about hexafluorosilicic acid:

Like several related compounds, hexafluorosilicic acid does not exist as a discrete species, that is, a material with the formula H_2SiF_6 has not been isolated. Hexafluorosilicic acid refers to an equilibrium mixture with hexafluorosilicate anion (SiF_6^{2-}) in an aqueous solution or other solvents that contain strong proton donors at low pH (acids described similarly include chloroplatinic acid, fluoroboric acid, and hexafluorophosphoric acid, and, more commonly, carbonic acid) Distillation of hexafluorosilicic acid solutions produces no molecules of H_2SiF_6, instead the vapor consists of HF, SiF_4, and water. Aqueous solutions of H_2SiF_6 contain the hexafluorosilicate anion, SiF_6^{2-} and protonated water.[50]

Workmen who handle these chemicals must wear hazardous materials suits for self protection. Spills are inevitable.

Water fluoridation plants take up acres. They are surrounded by tall electrified fences with guards who watch your every move when you approach them. They are like armed outposts Not even local policemen are allowed inside

But the worst part of fluoridation is the steady trickle of toxic waste into pretty much every river in the Lower 48 States.

The slurry liquor is dilute but there is a lot of it and it contains the most toxic wastes imaginable, including lead, arsenic, uranium, radium, polonium 210, and other radionuclides [51]

HYPERSENSITIVES, THE FLUORIDE ALLERGIC

Being an attorney, people confess all kinds of things to me. They know I cannot tell anyone what they confess People call me who are fluoride hypersensitives. Many of the hypersensitives went through an episode in which they were hypersensitized to fluoride. They confess these episodes to me.

The most common story is about the hypersensitive adult who as a child discovered the wonderful taste of Ipana toothpaste and would sneak into the bathroom at night and eat toothpaste. They tell how they did it for months Or my friend who was part of the first test of sodium fluoride in Grand Rapids Michigan in 1945 and was seriously overdosed and sickened as a child, and who now has reactions when she consumes even a small amount of fluoride.

Parents of mentally disabled children tell me how fluoridated water worsens their symptoms:

People tell me how they had to move away from their fluoridated town to one not yet fluoridated. They tell me how they get sick when they return and how they get better again when they leave. They tell of the rashes they develop when they shower in fluoridated water

Many tell how they were sensitized as children and how they can react strongly to fluoride Perhaps their body is panicking and trying to communicate to the conscious mind that the person must stop drinking the fluoridated water.

Could the effect be psychosomatic? Not according to Dr. Bruce Spittle, co-editor of the journal Fluoride Dr. Bruce Spittle has written about the hypersensitives, and he reports objective, double blind tests which confirm the fact that around one percent of us are very sensitive to fluoride.

In his free eBook, Spittle discusses fluoride studies done on animals, starting on page 50. Fluoride affects different species in different ways. Some species are more sensitive to fluoride. For example, rats are less sensitive. It takes more fluoride to poison a rat than to poison a human. Perhaps this is the result of selective evolutionary pressures; they are descended from the few rats that survived the NaF rat and roach poison we formerly spread around. Some species are more sensitive to it. Horses are very sensitive to fluoride. Horses drink a lot of water. A horse that always drinks from his trough will start drinking from muddy ruts in the road and eating snow Horses get sick from fluoride and die if they are not given fluoride free water All species are sensitive to fluoride, and different amounts of fluoride will kill all of them.

The NRC asked the EPA to study: endocrine effects of fluoride, decreased thyroid function, increased calcitonin activity, increased parathyroid hormone activity, secondary hyperparathyroidism, impaired glucose tolerance – these being possible mechanisms through which the hypersensitives are made to react. However, HHS and EPA have not done any research into these important areas. They have only studied caries, fluorosis, and bone fractures.

My sharpest criticism of those who run HHS and the EPA, is for their insensitivity to those who are fluoride allergic.

SILICOFLUORIDES CONTAIN LEAD

Silicofluorides not only produce HF which dissolves lead. Silicofluorides contain lead. NSF, the National Sanitation Foundation, puts out its analysis of silicofluorides, and admits that the liquid flurosilicic acid scrubber liquor (26% SiF and other toxins) after being diluted down to the point where the SiF level is 1 ppm, contains sometimes as much as .6 ppb lead.[52]

Lead is so nasty that we should not knowingly be adding any amount of lead to drinking water. The MCLG, maximum contaminant level goal for lead is zero. Bear in mind that flurosilicic acid is a mixture of hundreds of elements and lead is just one of them.

Silicofluorides come from super-phosphate fertilizer plants in Florida, Louisiana, and increasingly from China. To make super-phosphate fertilizer, processors cook rock phosphate with sulfuric acid. Sulfuric acid contains lead because the sulfuric acid is produced in gigantic lead pots, and part of the lead remains in the sulfuric acid, as NSF International[53] admits

Lead is a probable human carcinogen[54]. With both known and probable carcinogens the MCLG.[55] is always zero. That means none at all should be added to drinking water.

The fact that there is an MCL for lead of 15 ppb does not mean that a water district can therefore add any amount of lead it wants to add up to 15 ppb

There is an MCL, maximum contaminant level, which is the level at which the federal or state governments will file suit. This 15 ppb limit is the legally enforceable limit. If the MCL for lead is exceeded, then the water district must pay the cost of filtering out the lead.

The 2006 NRC Report at page 13 says the following about fluoride, and the same would apply to lead:

EPA's drinking-water guidelines are not recommendations about adding fluoride to drinking water to protect the public from dental caries. .. Instead, EPA's guidelines are maximum allowable concentrations in drinking water intended to prevent toxic or other adverse effects that could result from exposure to fluoride.

Nevertheless, we are knowingly adding lead to our drinking water. This is one more example of laws which are being broken just so we can preserve the chemical, fertilizer, and fluoride business

HHS & EPA POLICY: DILUTION IS THE SOLUTION

We quote[56] from Fluoride and Lead by Frances Frech:

> Let us tell you a tale of two cities–Tacoma, Washington, and Thurmont, Maryland. Both of them saw significant decline in [blood] lead levels only six months after fluoridation was stopped. (In Tacoma, that was due to equipment problems; in Thurmont, it was a temporary ban by the city council.) Tacoma registered a drop of nearly 50% ... ; in Thurmont it was 78%. To the best of our knowledge, no other explanations were offered. In Thurmont the ban is now permanent."

Unfortunately, Tacoma returned to fluoridating its drinking water and a battle continues over whether to reverse this policy.

Super-phosphate fertilizer is used to grow corn, soybeans, wheat, and other industrial food crops as rapidly as possible. To make super-phosphate fertilizer sulfuric acid is mixed with rock phosphate. Clouds of fluoride-rich[57] vapor go up the stacks. Before EPA intervention in the 1970s, the toxic smoke poisoned plants, animals, and people for miles around

The EPA required fertilizer plants to begin using wet scrubbers to filter out the fluoride along with the lead, arsenic, and many other contaminants from the smoke. The silicofluorides are the unfiltered and unprocessed scrubber liquor from the fertilizer production process. Silicofluoride scrubber liquor goes directly into tanker trucks and is delivered to the headwaters of the Tolt and Cedar Rivers, where it is poured into Seattle drinking water. The scrubber liquor is the most filthy substance imaginable. The idea that we dilute it and drink it is amazing.

The greatest irony of all this is that the toxic smoke that was illegal to go up the smoke stacks as air pollution and was illegal to dump as a liquid into rivers and oceans, was grandfathered in as a de facto legal medical additive to drinking water. It still gets dumped into rivers and oceans but only after passing through city water systems.

LEAD NOTICE

The EPA grants primacy on a state-by-state basis to each state which qualifies to carry out the role of implementing the SDWA, and Washington has been granted primacy. See 40 CFR 42.10. In each state there is a lead agency which is empowered to administer the SDWA, and in Washington that agency is the Department of Health. RCW 70.119A.080, RCW 43.21A.445. In RCW 43.21A.445 several Washington agencies led by the Department of Health are "... authorized to participate fully in and are empowered to administer ... " the SDWA.

Because the SDWA requires that state ". . drinking water regulations" be "no less stringent than the national primary drinking water regulations,"[58] state regulations likewise must be so limited. Therefore, the Department of Health must see to it that water districts disseminate notice regarding lead which the Safe Drinking Water Act

requires water districts to give.[59]

This is what the SDWA says regarding lead notice:

Public notice requirements

(A) In general

Each owner or operator of a public water system shall identify and provide notice to persons that may be affected by lead contamination of their drinking water where such contamination results from either or both of the following:

(i) The lead content in the construction materials of the public water distribution system.

(ii) Corrosivity of the water supply sufficient to cause leaching of lead

The notice shall be provided in such manner and form as may be reasonably required by the Administrator. Notice under this paragraph shall be provided notwithstanding the absence of a violation of any national drinking water standard.

(B) Contents of notice

Notice under this paragraph shall provide a clear and readily understandable explanation of—

(i) the potential sources of lead in the drinking water,

(ii) potential adverse health effects,

(iii) reasonably available methods of mitigating known or potential lead content in drinking water,

(iv) any steps the system is taking to mitigate lead content in drinking water, and

(v) the necessity for seeking alternative water supplies, if any.

The law is very clear on this point: The SDWA requires the EPA to write lead disclosures and see to it that states disseminate them. Water systems must give an honest notice to water drinkers regarding lead, and the Department of Health as the lead agency in enforcement of the SDWA[60] as set forth in RCW 70.119A.080 must pass and enforce a regulation requiring that water districts give such notice.

EPA is failing to enforce this part of the SDWA and failing to insist that municipalities send out truthful lead notices.

Silicofluorides contain more lead than does sodium fluoride. Compared with NaF, SiFs cause more lead to be leached from brass pipe and fittings and from the lead solder used to solder copper pipe and cast iron water mains. For all these reasons SiFs should be disallowed as fluoridation materials.

The lead notice section of the SDWA requires every municipality to disclose in writing to water drinkers

the lead content in the construction materials of the public water distribution system [and] the corrosivity of the water supply sufficient to cause leaching of lead.

The EPA has not required municipalities to issue such reports, nor has it properly informed them as to how to measure their waters' corrosivity.

The EPA is falling down on its job when it comes to honestly doing what needs to be done to get lead out of peoples' diets: Get all the fluorosilicates out of the drinking water, immediately and in all municipalities throughout the country. There is sufficient evidence for the EPA right now to issue an order to take effect immediately. Hearings would be held but they should be held after and not before the order goes into effect

LEAD – PROBABLE HUMAN CARCINOGEN

The EPA classifies lead[61] as a "probable human carcinogen" and adds.

Health effects associated with exposure to inorganic lead and compounds include, but are not limited to, neurotoxicity, developmental delays, hypertension, impaired hearing acuity, impaired hemoglobin synthesis, and male reproductive impairment. Importantly, many of lead's health effects may occur without overt signs of toxicity. Lead has particularly significant effects in children, well before the usual term of chronic exposure can take place. Children under 6 years old have a high risk of exposure because of their more frequent hand-to-mouth behavior.

Once lead is consumed it pervades the entire body, passing through brain, placental, and mammary barriers.

The need to add no lead to drinking water is especially true because there are other vectors of lead exposure which are hard to eliminate such as lead paint, batteries, bullets, and many industrial uses. However, the most serious source of lead is brass plumbing, as I discuss above on page 8, at levels up to 1,600 ppb.

EPA defines MCLG[62] as "the level of a contaminant in drinking water below which there is no known or expected risk to health." The MCLG for lead is zero. This means that any level of lead added to water may cause or increase risk to health. Therefore, no lead may be added to water and therefore fluoridation, which adds lead to drinking water, must cease.

The authors of the 2006 NRC Report at page 285 say:

> The EPA Office of Drinking Water establishes MCLGs of zero for contaminants that are known or probable human carcinogens. Chemicals for which cancer hazard is judged to be absent are regulated via the reference dose (RfD) method (see Chapter 11).

The Wikipedia article[63] on "lead poisoning" says:

> No safe threshold for lead exposure has been discovered—that is, there is no known amount of lead that is too small to cause the body harm . The US Centers for Disease Control and Prevention and the World Health Organization state that a blood lead level of 10 µg/dL or above is a cause for concern; however, lead may impair development and have harmful health effects even at lower levels, and there is no known safe exposure level.

EPA and HHS have acted in bad faith by ignoring the fact that the fluoridation materials they endorse routinely contain lead and dissolve lead from plumbing. No amount of lead should routinely be added to water, and no amount of fluoride should be added to water of the type which contains lead or dissolves the lead out of plumbing. For this reason alone EPA and FDA have grounds to terminate fluoridation with silicofluorides immediately, and they should do so

ARSENIC – CONFIRMED CLASS A HUMAN CARCINOGEN

NSF reports that 43 percent of tanker truck loads of silicofluoride contain arsenic, and that those loads can contain levels, which after the scrubber liquor is diluted to the point where fluoride concentration goes from 26.0% to 1.0 ppm, arsenic will be present at levels of up to .6 ppb.[64]

Arsenic is a poison and a known human carcinogen.[65] The smallest amount can kill in many ways. Regarding arsenic, CDC's ATSDR[66] has this to say:

> Prolonged arsenic exposure causes skin and lung cancer and may cause other internal cancers as well. (page 2)

> A small molecule [sic, actually arsenic is an atom] that can easily get into cells, arsenic can cause cell injury and death by multiple mechanisms. Interference with cellular respiration explains the potent toxicity of arsenic. In addition, arsine gas may interact directly with red cell membranes. Arsenic is a known human carcinogen, but the specific mechanisms by which it causes cancer are less well understood. (page 46)

> A scientific consensus has not yet been reached on the many suggested modes of arsenic carcinogenesis that exist in the literature. These include modes that are predominately genotoxic (i.e., chromosomal abnormalities, oxidative stress, and gene amplification) vs. more nongenotoxic (i.e., altered growth factors, enhanced cell proliferation and promotion of carcinogenesis, and altered DNA repair). Likewise, the dose-response relationship at low arsenic concentrations for any of these suggested modes is not known [Kitchin 2001]. (page 48)

> Arsenic can cause serious effects of the neurologic, respiratory, hematologic, cardiovascular, gastrointestinal, and other systems. Arsenic is a carcinogen in multiple organ systems. Interindividual and population differences in arsenic methylation and nutritional status may be factors in susceptibility to arsenic toxicity. (page 68)

Arsenic can be excreted through hair, skin, feces, or urine, but primarily through kidneys. Kidneys do not excrete all arsenic consumed, especially for those who have weak kidneys. Arsenic, whether eaten, drunk, or inhaled may be deposited throughout the body. It may wind up in the lungs where it may cause lung cancer or in the skin where it may cause skin cancer.[67]

The MCL for arsenic in water is 10 ppb. This does not mean it is acceptable for water districts to add arsenic knowingly at any level up to 10 ppb. The MCLG for arsenic is zero,[68] as it is for lead.[69] When the MCLG for an element or compound is zero,[70] none should be added at all.

The need to add no arsenic to drinking water is especially true because there are other vectors of exposure which are hard to eliminate. Until recently arsenic was used as a wood preservative. Lead arsenate was used for example, in Wisconsin in agriculture until the 1950s.[71] Once there is arsenic in the soil it does not disappear. Homes and schools are built on old farmland, and it is easy for children and gardeners to be exposed to arsenic. Arsenic occurs naturally in some water sources. It is used in making lead batteries and in various industrial applications. Amazingly, it is legal in the United States to feed arsenic to non-organic chickens.[72] The body is poor at excreting arsenic, as noted above

EPA defines MCLG[73] as "the level of a contaminant in drinking water below which there is no known or expected risk to health." The MCLG for arsenic is zero (as it is for lead). This means that any level of arsenic added to water may cause or increase risk to health. Therefore, no arsenic should be added to water and therefore fluoridation, which adds arsenic to drinking water, must cease.

EPA and HHS have acted in bad faith by ignoring the fact that the fluoridation materials they endorse routinely contain arsenic (as well as lead). No amount of arsenic should routinely be added to water. For this reason alone EPA and FDA have grounds to terminate fluoridation with silicofluorides.

FLUORIDE, LEAD, ARSENIC, ALUMINUM TOGETHER – SYNERGISTIC EFFECT

It is entirely possible that fluoride, lead, arsenic, aluminum, and several other heavy metals and strange chemicals, all of them present together in SiF, act more powerfully together than they do individually. We know of one example of a synergy involving these elements: Fluoride interferes with the body's ability to process and eliminate arsenic. So arsenic and fluoride together are more toxic than either acting alone.

When you add up the MCLGs for F, Pb, As, Al, U, and other metals and compounds found in SiF, the sum of the MCLG equivalents becomes a large number.

A WAY OUT

Today there are more contaminants in the source waters used to make drinking water, so it is time for EPA to respond to this change in the environment by drawing the logical conclusion: With all the competing heavy metals joining the fluoride, water fluoridation is no longer the safe way to deliver fluoride. EPA could advise people that if they want fluoride they can get it easily by brushing their teeth more frequently with fluoridated toothpaste and even swallowing some of it.

Fluoridation is dying out – just like tetraethyl lead, asbestos, cigarettes, and mercury amalgam. In most cities and towns where the issue is voted on, a majority votes to end fluoridation. Former Georgia Governor Andrew Young is pressing for a Fluoride-Gate investigation. Minorities and those who eat a less nutritious diet are more subject to being harmed by fluoride, lead, and arsenic.

The possibility that people and corporations are going to be sued is becoming clearer. I discuss mass toxic tort actions with fellow attorneys. There are tens of thousands of people now dead who would still be alive if National Kidney Foundation, EPA, and state boards of health had disclosed what they knew, that those with weak kidneys especially should not drink fluoridated water. The suits will come, so those who are pushing fluoridation had better switch sides and try to mitigate the damages caused.

The administrators at HHS and EPA who support fluoridation – in conflict with the scientists who oppose it – should get off the sinking fluoride ship while the getting is good. If losing face is a concern, there is a way to change positions on fluoridation without losing face or admitting liability. That is to rely on a reason which has just become clear: the synergistic effects of many contaminants acting together.

There is not only more artificial fluoride in water, there is also more artificial fluoride in foods and beverages. There is also more lead and arsenic in the environment. Arsenic was spread in tens of thousands of agricultural sites around the country over the last century, and arsenic never goes away. F-Pb-Al-As – all work together synergistically and constitute a new group of troublesome chemicals. EPA should create an MCL and an MCLG for F-Pb-Al-As combined.

If HHS and EPA rely on the synergistic effect to change their position on fluoridation, they will be able to take credit for deciding that fluoridation has to stop without ever admitting they were wrong about fluoride in the first place.

HHS and EPA could declare that drinking water is no longer a safe vehicle for delivering fluoride. They can say that the chemical ecology of water has changed and so HHS and EPA is having to change. People will not have to give up their fluoride: They can just brush more frequently and swallow a little toothpaste if they want to

There's your open door. Go through it while you can

NOTICES WHICH WATER DISTRICTS SHOULD GIVE

If the EPA were to allow the continued use of silicofluorides, it should require that municipalities give notices which include the following warnings:

> Those who drink tap water and eat food made with tap water should be aware of the following: Tap water in this water district is fluoridated with silicofluorides Silicofluorides contain lead and arsenic. Silicofluorides leach lead from brass pipe, from brass fittings, and from the lead based solder used to solder together brass and copper pipe Silicofluorides leach lead from the lead solder used to solder cast iron water main pipes. The lead content of your drinking water may vary from zero to .6 ppb from the silicofluoride added, and the level may be more as a result of lead leached from pipes as a result of silicofluorides added. The federal MCL, maximum contaminant level for lead, is 15 ppb. The federal MCLG, maximum contaminant level goal, is zero, meaning that any and all amounts of fluoride intake should be avoided where ever possible.
>
> Homes, apartments, schools, and other buildings built before 1986 generally utilize brass pipes containing up to 30.0% lead, and lead levels in such buildings, have been known to be as high as 1,600 ppb, especially when water sits in lines for long periods of time. Brass pipes in buildings built after 1986 generally use brass containing up to 8.0% lead (except in California where limits are lower).
>
> Those who wish to avoid consuming lead and who wish to avoid having their children consume lead should not drink tap water or use it to cook food and instead should use a source of water known not to contain lead such as spring water, distilled water, or water filtered with a reverse osmosis filter. Lead is known to cause brain damage

A better solution than requiring disclosure would be for EPA to ban the use of silicofluorides as fluoridation materials, a power which the EPA has, given the harm that silicofluorides are causing, particularly in connection with lead poisoning, under SDFA § 300 g-1 (b)(1)(d):

> Urgent threats to public health — The Administrator may promulgate an interim national primary drinking water regulation for a contaminant without making a determination for the contaminant under paragraph (4)(C), or completing the analysis under paragraph (3)(C), to address an urgent threat to public health as determined by the Administrator after consultation with and written response to any comments provided by the Secretary of Health and Human Services, acting through the director of the Centers for Disease Control and Prevention or the director of the National Institutes of Health.

CONCLUSION: BAD FAITH

HHS and EPA are not monolithic entities There are people of all different persuasions in those agencies. Some therein – such as the EPA union – even agree with me. So when I hurl accusations of bad faith against HHS and EPA, try not to take it personally because I am only addressing the stubborn defenders of fluoride among you.

HHS and EPA have acted in bad faith by failing to study all the research areas listed in the 1993 and 2006 NRC Reports, including the connection between silicofluorides, lead, and arsenic.

Given the fact that HHS and EPA have recommended continued fluoridation at .7 ppm and have done so without studying all the issues NRC identified, including issues pertaining to the connection between silicofluorides, lead, and arsenic, HHS and EPA have acted in bad faith

HHS and EPA have acted in bad faith by implying that they have done sufficient research to be confident that all may drink all the tap water they want at .7 ppm and not suffer any harm, particularly in light of the increase in lead uptake resulting from silicofluorides.

HHS and EPA have acted in bad faith by failing to give clear and correct notice to those who would have benefitted most from receiving it: infants and children, those with kidney disease (who drink large quantities of water), diabetics (who drink large quantities of water), to the effect that they should avoid drinking fluoridated water

Fluoridation is a fraud. It should be stopped

ACTION PROPOSED

HHS (including the CDC) and the EPA should retract their endorsement of water fluoridation.

The EPA should commission the NRC to write a report dealing with artificial water fluoridation of drinking water. The new report should ask whether it is safe to fluoridate and if so how water fluoridation should be conducted and at what level and with which type of fluoride. The report should be due in one year.

The EPA should exercise its authority under the Safe Drinking Water Act to order an immediate ban on artificial water fluoridation throughout the United States. This ban should remain in place until the new report has been received from the NRC.

HHS and EPA should commit themselves to airing all sides of the fluoridation debate, particularly as it applies to the link between SiFs and lead poisoning. They should post the debate on their web sites. They should correct all the many errors on their websites pertaining to fluoridation, including those relating to the link between SiFs and lead. This policy of openness should apply to all health and environmental issues.

CDC should deal forthrightly with the serious ethics charges laid against it.

The EPA should retract its support of the NSF, including its financial support and its "imprimatur" on NSF publications. The EPA should instruct the NSF to cease making any statements which would imply that the EPA agrees with NSF's certification of SiFs as acceptable fluoridation materials.

The EPA should obtain rights to the NSF 60 book, which says almost nothing and sells for only $325, and make it available on its website so that water districts and everyone else can see what a fraud the NSF 60 certification is.

The EPA should declare in plain and simple English that an MCL is not an authorization to add any level of a particular contaminant, including fluoride, but is to the contrary a requirement to remove that contaminant if its level exceeds the MCL.

The EPA should declare in plain and simple English that an MCLG is a rule against adding any amount of a particular contaminant above the MCLG level. Thus, if the MCLG for lead and arsenic are zero, a water district may not add any lead or arsenic to drinking water whatsoever, including the tiny amount of mercury and lead found in SiF fluoridation materials.

The FDA should ban fluoridation if the EPA does not do it first.

The FDA should require that all bottled water containing fluoride be labeled to disclose the fluoride level and the type of fluoride in the water. It should be presumed that bottled water which says nothing on the label about fluoride contain no NaF or SiF or a minimal amount of CaF_2.

Likewise, all reconstituted juices, all beer, all bread, all foods made using fluoridated water should disclose the fluoride level of the water used to make the product. It shall be presumed that all reconstituted juices, all beer, all bread whose label says nothing about fluoride contains no fluoride.

The FDA should ban fluoridated toothpaste. The risk of children eating it is too great for such a product – one which does nothing to protect teeth against decay – to be found in millions of bathrooms in easy reach of children.

If the FDA should allow continued sale of fluoridated toothpaste, it should require big print warnings that fluoridated toothpaste be kept out of the hands of and not used by children under eight years of age.

If the FDA should allow continued sale of fluoridated toothpaste, the FDA should require that fluoridated toothpaste have a taste that children dislike in order to discourage children from eating it.

EPA, HHS, CDC, and FDA should recommend to the Attorney General of the United States that he appoint special counsel to investigate "Fluoride-Gate".

CLOSING

This letter is also intended to be read by all those who are forced to drink highly dilute toxic waste and want to see this crude practice ended. For that reason I have gone into detail explaining the relevant legal and scientific issues. Fluoridation should be a political issue in the 2012 campaign and if Andrew Young of Atlanta has his way, it will be.

I want to express appreciation to Dr. Richard Sauerheber for tutoring me in basic chemistry.

Sincerely,

James Robert Deal, Attorney

WSBA Number 8103

President, http://Fluoride-Class-Action.com

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1. Jim
September 1st, 2011 at 07:02 | #1
Reply | Quote

This is a great letter. Marc Edwards of Virginia Tech is one of the top corrosion experts for water. He warned agencies of the lead leaching risks of the chloramine H2SiF6 combination when the EPA started advising cities to switch from straight chlorine. They ignored him and his many warnings. Then the Washinton DC changeover was on Nov 1 2000. DC had been double dosing with chlorine and created lots of lead blisters. Much of the system was even lead pipes. They were at the end of their three year lead survey as required and noticed the jump but never reported it to anyone. They allowed lead levels to remain toxic unmentioned to the public. They did get Marc Edwards to come up with proof with three grad students testing water for lead for like six months. They told Marc he had the contract. He never got paid a dime and was warned he would never work again if he was not silent on this problem.
A 300 million program to change out lead lateral lines started and done incorrectly which caused higher lead levels and halted after 100 million was wasted. Bottled water and fliulters were given to 25,000 homes that had lead supply pipes. They never mentioned another 100,000 also had the high lead levels. This was front page "Washington Post for 30 days running. DC Watch had the hearings to read. Hearings are still being held as who to blame. CDC did a small study of 201 kids who were receiving the bottled water on lead blood levels. They tested fine as this was many months and up to a year later on safe water. Lead levels in blood fall 50% per month after toxic intake ends. This was designed to be fraud.
Edwards years later won the Mac Arthur engineering award and 500,000 no strings cash. He used some cash to prove other data showed many hundreds if not thousands of kids were lead toxic because of this government caused lead screw up.
He also helped discover lead contamination problems in new pipes in a school building. It was traced to low lead 8% brass shut offs. It was proven the castings had much higher lead levels on the surface, thus more lead leaching.
Old schools often have sky high lead levels in water. As do lots of other buildings but the older are usually the worst. But exceptions do exist ignored and untested. The engineering department at Virginia Tech does have a contact number for him I think. He consults worldwide of water corrosion. He is tops on Potomic water. Cities only test samples every three years. Maas 2007 tested over 150,000 homes in North Carolina for the EPA. Coplan 2007 also shows bench testing for lead levels from fluoridation and chlorine, chloramine.

2. Jeanette Bajorek
April 20th, 2011 at 12:29 | #2
Reply | Quote

James,
Since making clear to the reader just what point you are making when you reference certain pages in the NRC report is difficult if the actual word "lead" does not appear in the NRC reference, and not having researched all your references for myself,(to see if you used your own paper as reference at all), I am thinking that your own paper on lead is so good and thorough and has so many references – why don't you just reference your own paper? or at least use the references you provided in your paper rather than using any more NRC page numbers. Or isn't that done? Everything you have presented thus far is so logical and thorough and am impressed.

That's the best I can think to do, outside of re-reading the NRC book to find something that would fit, and I don't want to take the time to do that.
Best,
Jeanette

3. Tim
April 17th, 2011 at 20:31 | #3
Reply | Quote

The best information I've come across on fluoride and fluoridated water is at the website http://www.MacBrussell.com. Just go to the bottom of the homepage. Dr. John Lee, Dr. John Yiamouyannis, Dr. Phyllis Mullenix, and Joanie Greggains explain the history, origin, and the health hazards of this toxic waste. The audio is 75 minutes long. Hopefully people will burn cds of the audio and pass them around.

4. Audrey Adams
April 17th, 2011 at 18:41 | #4
Reply | Quote

Excellent, James! Have you thought about sending a copy to a Seattle Public Utilities? Perhapas Everett, too?

5. Jeanette Bajorek
April 17th, 2011 at 15:07 | #5
Reply | Quote

dear James,

I am a great admirer of your work on LEAD. Just one little comment here: I tried to follow up your page number links in the NRC 06 book on recommendations to research the lead problem in drinking water, but the pages indicated 6-9and 43-44 made no mention at all of lead.

Just wonder what I may be missing ?
These letters to the HHS and EPA are wonderful in my opinion. With these issues all out in the open this way, I don't see how they can keep stonewalling .
Jeanette

anette Bajorek

1. June 3rd, 2011 at 22:53 | #1
   FLUORIDE CLASS ACTION » Involving Students
2. June 3rd, 2011 at 22:55 | #2
   WASHINGTON ACTION FOR SAFE WATER » Involving Students
3. June 5th, 2011 at 03:47 | #3
   FLUORIDE CLASS ACTION » Washington Board Of Health Put On Notice
4. June 5th, 2011 at 05:35 | #4
   WASHINGTON ACTION FOR SAFE WATER » Washington Board of Health Put on Notice
5. August 20th, 2011 at 06:44 | #5
   FLUORIDE CLASS ACTION » Hempfest Flier
6. August 27th, 2011 at 02:46 | #6
   FLUORIDE CLASS ACTION » Questions Regarding Lead in Seattle Water
7. October 10th, 2011 at 05:15 | #7
   FLUORIDE CLASS ACTION » Occupy Seattle Flier 10-10-11
8. October 11th, 2011 at 15:00 | #8
   Reasons to Oppose Flouridation : gohealthynext com
9. October 13th, 2011 at 09:45 | #9
   FLUORIDE CLASS ACTION » FOIA to Everett Water District 10-13-11
10. October 15th, 2011 at 11:04 | #10
    FLUORIDE CLASS ACTION » Questionaire To Candidates
11. October 16th, 2011 at 13:27 | #11
    FLUORIDE CLASS ACTION » Research Should Be Done In Now Non-Fluoridated Calgary
12. October 22nd, 2011 at 04:57 | #12
    WASHINGTON ACTION FOR SAFE WATER » Occupy-Seattle-Flier-10-21-11
13. October 22nd, 2011 at 05:12 | #13
    FLUORIDE CLASS ACTION » Occupy Seattle Flier 10-21-11
14. October 30th, 2011 at 03:17 | #14
    WASHINGTON ACTION FOR SAFE WATER » Press Release – WASW to Occupy Seattle City Hall Monday at Noon to Protest Lead, Arsenic, Silicofluoride Added to Water
15. October 30th, 2011 at 17:37 | #15
    FLUORIDE CLASS ACTION » Press Release - Showdown At City Hall – Monday 10-31-11 – Please come

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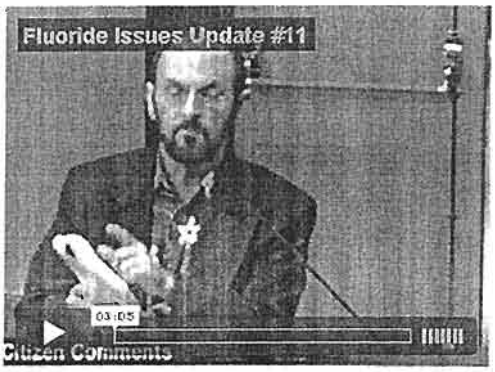
www.Fluoride-Class-Action.com/sham



Fluoride Issues Update #11 from Golda Starr on Vimeo.

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- 2999
- Dentists try to buy off California agency
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- EPA to regulate waste water from fracking
- Post Links to Facebook-Twitter
- News From Quebec
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- Fluoridation in Arkansas – Expensive and Complex
- Press Release - Showdown At City Hall – Monday 10-31-11 – Please come
- Questions about Lead Leaching

- Occupy Seattle Flier 10-21-11
- Research Should Be Done In Now Non-Fluoridated Calgary
- Questionaire To Candidates
- Collect Email Addresses of Office Holders and Candidates and Put them on Notice
- New York Times – More Communities End Fluoridation
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- Where does your politician stand on silico-fluoridation?
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- Federal Fluoride Suits
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- Letter to Snohomish County Candidates
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- Hempfest
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- Citizens for Safe Drinking Water Sue
- Demands to De-Fluoridate Issaquah
- Hereford Texas - The Town Without A Toothache
- NSC 1997 Report: Fluoride As Nutrient
- ADA – FI Does Not Cause Bone Cancer – FAN Disagrees
- Golda Starr Addresses Everett City Council
- Everett Blog is Covering City Council Fluoride Debate
- Notice to Everett of Liability 7-20-2011
- Fluoride And Hypothyroidism
- ADA and HHS Conspire to Squelch Fluoridegate
- Cuba stopped fluoridation in 1997, and cavities did not rise
- Dr. Hirzy of EPA Union
- Most Dentists Have Not Studied Fluoridation
- Alveda King & Andrew Young Against Fluoride
- Fairbanks Ends Fluoridation
- ADA, CDA $200,000 to remove fluoride from Cal list
- Bill Gates' Vaccines
- Dr. Sauerheber on Salt and Fluoridation
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- Fluoride-Gate–CDC-Ethics-Complaint
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- Sauerheber to EPA 4-9-2011
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- JRD Presentation at FAN Conference
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- Nuclear Power is Not Green
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- Fluoride in the New York Times
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- Beyond Nuclear - Videos
- Fluoride Action Network
- Mortgage Modification Attorney
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- Rebuttal to American Dental Association
- Sodium Fluoride - MSDS
- Spots on My Teeth
- Uranium is Filthy to Mine and the Contamination is Permanent
- Vaccines that Backfire

Tags

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- Vaccines that Backfire

Thanks, Wikipedia.

Fluoride Is Poison, Says Dartmouth Doctor

New evidence seems to confirm that by fluoridating our water, we are poisoning our children.

By Tom Valentine

More than two years ago, the court-killed *Spotlight* wrote about George Glasser, a citizen researcher who blew the whistle on the use of highly-toxic fluorosilicic acid from rock fertilizer processing as the primary source of community water fluoridation.

Now, a massive study of young children who have been subjected to fluorosilicic acid fluoridation in their New York communities shows that the water additive does not improve kids' teeth and could even be poisoning them.

Until that time, most people were under the impression that water fluoridation used sodium fluoride, rat poison, a by-product of aluminum manufacturing.

Glasser, however, pointed out that more than 75 percent of the U.S. water fluoridation communities have been using the even more toxic fluorosilicic acid since the late 1970s.

Glasser was the first to stress the excessive toxicity inherent in using the hydrofluorosilicic acid residue that is removed from the industrial pollution control "scrubbers" in the manufacture of phosphate fertilizers.

The chemists refer to this material as silicofluorides and have now conclusively shown that the fluoridation material is linked to other heavy metal toxins that are found in drinking water—lead, arsenic, aluminum and cadmium for example.

In the March 2001 issue of the journal *Neuro Toxicology*, a team of researchers led by Dr. Roger Masters of Dartmouth College reported evidence that public drinking water fluoridated with fluorosilicic acid is linked to higher levels of lead in children.

After pointing out that since 1992 only about 10 percent of America's fluoridated communities use sodium fluoride and 90 percent use fluorosilicic acid, the researchers stated that about 140 million Americans have this chemical placed in their water.

They also pointed out that sodium fluoride was tested on animals and approved for human consumption, but fluorosilicic acid had not been so tested and approved.

The research team studied the blood-lead levels in more than 400,000 children in three different samplings. In each case they found a significant link between fluorosilicic acid-treated water and elevated blood levels of lead.

In the latest study, the blood levels of about 150,000 children ranging in ages from infant to 6 were analyzed.

The samples were collected by the New York State Department of Children's Health from 1994 through 1998.

Researchers concluded that the fluorosilicic acid-treated water was equal to or worse a contributor of blood-lead levels as old house paint.

Dr. Masters said these preliminary findings correlate the fluorosilicic acid water treatment and behavior problems that are due to known effects of lead on brain chemistry.

Additionally, a study in Germany showed the fluorosilicic acid water (SiFs) may inhibit the enzyme cholinesterase which plays a key role in regulating neurotransmitters.

"If SiFs are cholinesterase inhibitors, this means that SiFs have effects like the chemical agents linked to Gulf War Syndrome, chronic fatigue syndrome and other puzzling conditions that plague millions of Americans," Masters said. "We need a better understanding of how SiFs behave chemically and physiologically."

Last March, Dr. Masters testified before New Hampshire legislators in favor of the Fluoride Product Quality Control Act. The bill would put the SiFs to a series of tests, and perhaps further research on neurotoxicity and behavior.

"If further research confirms our findings," Masters said, "this may well be the worst environmental poison since leaded gasoline."

The EPA admits it has no data on the health and behavioral effects of SiFs.

Dr. Masters asked: "Shouldn't we stop intentionally exposing 140 million Americans to an untested chemical until the risks are extensively and objectively evaluated by independent researchers?"

And, the final insult: There is no conclusive evidence that fluoridation of drinking water significantly improves the teeth of children at all. ™

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Neurotoxicology. 2000 Dec;21(6):1091-100

Association of silicofluoride treated water with elevated blood lead.

Masters RD, Coplan MJ, Hone BT, Dykes JE.

Foundation for Neuroscience and Society, Dartmouth College, Hanover, NH 03755-3547, USA. roger.d.masters@dartmouth.edu

Abstract

Previous epidemiological studies have associated silicofluoride-treated community water with enhanced child blood lead parameters. Chronic, low-level dosage of silicofluoride (SiF) has never been adequately tested for health effects in humans. We report here on a statistical study of 151,225 venous blood lead (VBL) tests taken from children ages 0-6 inclusive, living in 105 communities of populations from 15,000 to 75,000. The tests are part of a sample collected by the New York State Department of Children's Health, mostly from 1994-1998. Community fluoridation status was determined from the CDC 1992 Fluoridation Census. Covariates were assigned to each community using the 1990 U.S. Census. Blood lead measures were divided into groups based on race and age. Logistic regressions were carried out for each race/age group, as well as above and below the median of 7 covariates to test the relationship between known risk factors for lead uptake, exposure to SiF-treated water, and VBL >10 microg/dL. RESULTS: For every age/race group, there was a consistently significant association of SiF treated community water and elevated blood lead. Logistic regressions above and below the median value of seven covariates show an effect of silicofluoride on blood lead independent of those covariates. The highest likelihood of children having VBL> 10 microg/dL occurs when they are both exposed to SiF treated water and likely to be subject to another risk factor known to be associated with high blood lead (e.g., old housing). Results are consistent with prior analyses of surveys of children's blood lead in Massachusetts and NHANES III. These data contradict the null hypothesis that there is no difference between the toxic effects of SiF and sodium fluoride, pointing to the need for chemical studies and comprehensive animal testing of water treated with commercial grade silicofluorides.

PMID: 11233755[PubMed - indexed for MEDLINE]

Publication Types, MeSH Terms, Substances

Fluoride chemicals may increase lead accumulation, say researchers

Published on March 16, 2010 at 3:11 AM

Fluoride chemicals added to public water supplies, boosts lead absorption in lab animals' bones, teeth and blood, report Sawan, et al. (*Toxicology* 2/2010). Earlier studies already show children's blood-lead-levels are higher in fluoridated communities, reports Sawan's research team.

"...exposure to increased amounts of lead and fluoride occurs at about the same age (1-3 years)... Therefore, this is a critical time when systemic exposure to fluoride should be minimized since fluoride may increase lead accumulation," the researchers caution.

Low-level lead exposure is associated with lower IQ, ADHD and many health and behavior ailments.

Fluosilicic acid (fluoride) is added to water supplies ostensibly to reduce tooth decay.

Sawan's team put fluosilicic acid, with and without lead, into lab animals' drinking water. They found more lead in tooth enamel, surface bone, whole bone, and tooth dentine in rats co-exposed to fluoride and lead.

Possibly anticipating criticism that rats were fed higher fluoride-concentrated water than people drink, the authors write, "This concentration was chosen because it produces plasma fluoride levels that are comparable with those commonly found in humans..."

Increased prevalence and severity of fluoride-discolored teeth (fluorosis) proves U.S. children are already fluoride-overexposed, "which may cause their blood-lead levels to increase and produce more lead toxicity," they write.

"These findings suggest that a biological effect, not recognized so far, may underlie the epidemiological association between increased blood-lead levels in children and water fluoridation," concludes Sawan's research team.

"[O]ur findings may have serious implications for populations exposed to increased amounts of both lead and fluoride, particularly young children," the research team writes.

Fluoridation chemicals often contain lead (NSF International).

Attorney Paul Beeber, President, New York State Coalition Opposed to Fluoridation (NYSCOF), says, "People need to lobby and petition their legislators to stop fluoridation in their towns, cities and states. Legislators are ignoring the science proving fluoridation is endangering our health, our water supplies and wasting tax dollars while denying freedom of choice." (see: http://www.fluoridealert.org/health/sitemap.html)

Masters and Coplan's landmark studies show higher blood-lead-levels in children living in silico-fluoridated communities (*Neurotoxicology* 2000, 2007). Macek's research shows children's higher blood-lead-levels are associated with water fluoridation when lead is already in the environment (*Environmental Health Perspectives*, 2006).

Some fluoridation chemicals originate in China, Mexico and Japan, reports the CDC.

SOURCE New York State Coalition Opposed to Fluoridation, Inc.

Costs of Fluoridation

In a typical city, people consume less that 1% of all water that passes through the public water system. The rest of the water is used for washing, toilet flushing, industry, gardening, and so on. Even when many found it reasonable to believe that fluorides reduced tooth decay, fluoridation of public water supplies was an extremely costly way to deliver 1.0 milligram fluoride per day to the target population of children from birth to age 12.

For example, the annual projected cost for water fluoridation chemicals alone for the city of Tacoma, Washington in 1992 was $125,000. (C.R. Myrick, Water Quality Coordinator, City of Tacoma, telephone conversation with Wini Silko, citizen of Tacoma, November 15, 1991.) By contrast, the cost of fluoride tablets and drops for all children aged 12 and under in Tacoma would have been less than $25,000 (based on a cost of $1.20 per thousand 1.0 milligram tablets).

This comparison does not take into account the capital and labor costs of fluoridation or the substantial hidden costs, which include corrosion of water mains and plumbing, environmental pollution and degradation of the health of the general population.

Fluorides are so highly corrosive that they cannot be contained in metal or glass. Even at dilutions of 1.0 ppm, fluorides increase corrosion rates and cause leaching of lead and other metals from plumbing. In the first half of 1992 Tacoma, Washington failed to meet EPA standards for lead contamination in its water. When equipment failure forced a halt to fluoridation of Tacoma's water supply, tests showed a nearly 50% drop in lead contamination. (Letter to Michael Heath, Washington State Department of Health, from C.R. Myrick, Water Quality Coordinator, City of Tacoma, December 2, 1992.) In February 1994, Thurmont, Maryland reported a similar drop in lead levels when fluoridation was halted there. ("Lead levels in Thurmont water drop" by Julia Robb in The Frederick (MD) Post, February 3, 1994.)

When a claimed 20% decrease in tooth decay is compared to a 600% increase in bone cancer or a 41% increase in hip fractures, when the cost of a tooth filling is compared to the cost of a hip fracture or cancer treatment, it is obvious that the human and economic costs of fluoridation are staggering.

FLUORIDE CIVIL RIGHTS ISSUES

NOTE: Yellow highlights below in the copy of the text from Ambassador Young's March 29, 2011 letter have been added by The Lillie Center Inc. Condensed to display on one page.

ANDREW YOUNG

Chip Rogers, Senate Majority Leader, Rm. 236
Georgia State Capitol
Atlanta GA 30334

Subject: Withdrawal of Law Requiring Water Fluoridation in Georgia

Dear Senators and Representatives:

I am writing to convey my interest in seeing that Georgia's law mandating water fluoridation for Georgia communities be repealed.

My father was a dentist. I formerly was a strong believer in the benefits of water fluoridation for preventing cavities. But many things that we began to do 50 or more years ago we now no longer do, because we have learned further information that changes our practices and policies. So it is with fluoridation. We originally thought people needed to swallow it, so the fluoride would be incorporated into teeth before they erupted from the gums. Our belief in the need for systemic absorption was why we began adding fluoride to drinking water. But now we know that the primary, limited cavity fighting effects of fluoride are topical, when fluorides touch teeth in the mouth. We know that fluorides do little to stop cavities where they occur most often, in the pits and fissures of the back molars where food packs down into the grooves. This is why there is a big push today to use teeth sealants in the molars of children. We also have a cavity epidemic today in our inner cities that have been fluoridated for decades.

So now we know that fluoride's impacts are primarily topical and are very limited where needed most in the teeth. And on top of this we are learning that fluorides do not simply affect teeth, but can also harm other tissues and systems in the body. So we must weigh the risks to kidney patients, to diabetics, and to babies against the small amount of cavities prevented by swallowed fluorides. The National Research Council has acknowledged that kidney patients, diabetics, seniors, and infants are susceptible groups that are especially vulnerable to harm from fluorides. There are millions of these persons who have these health conditions or who meet the criteria for concern.

The National Center for Health Statistics says that 41% of 12-15 year old adolescents now have the teeth staining called "dental fluorosis" that shows overexposure to fluorides as a child, and that 3.6% have the very visible moderate and severe forms of the condition. This translates into millions of persons with disfiguring impacts from fluorides. How many of these persons can afford the tens of thousands of dollars to have veneers or other cosmetic dental work performed?

There is growing bipartisan support across the country for halting water fluoridation. And eleven unions of EPA workers, representing 7,000 EPA lab workers, scientists, and others have called for a halt to fluoridation. The recent suggested lowering of fluoride levels in water does not address the fact that we still cannot control the amount of fluorides that sensitive individuals ingest. People are calling for investigative Fluoridegate hearings, and one can understand why, given the fact that the story about fluorides keeps changing.

I am most deeply concerned for poor families who have babies: if they cannot afford unfluoridated water for their babies milk formula, do their babies not count? Of course they do. This is an issue of fairness, civil rights, and compassion. We must find better ways to prevent cavities, such as helping those most at risk for cavities obtain access to the services of a dentist.

Peace and Blessings,

Andrew Young

PROVIDENCE
BAPTIST CHURCH
"Winning the Lost...Developing the Saved"

Dr. Gerald L. Durley
Pastor

March 9, 2011

Senator Chip Rogers
Senate Majority Leader
Georgia State Capitol – Room 236
Atlanta, Georgia 30334

RE: Repeal of Georgia's Mandatory Fluoridation Law

Dear Senator Rogers:

As a citizen, a minister, and a community leader, I am writing to state my opposition to the practice of water fluoridation, and to ask that the current Georgia law mandating water fluoridation throughout our state be repealed.

First and foremost, water fluoridation takes away people's choice. We have a God-given right to not have fluoride forced into our bodies or the bodies of our children. Fluoridation supporters attempt to say that people are not forced to drink fluoridated water, but that is a disingenuous statement that ignores reality. Many families do not have funds to buy an expensive home water fluoride removal system, or to buy unfluoridated bottled water for making their babies' milk formula, so in truth they are forced to drink fluoride in their water simply because of their economic status or household income.

Second, fluoridation disproportionately harms members of the black community. The Centers for Disease Control's own information acknowledges that blacks have significantly more "dental fluorosis" teeth staining than whites. For many, the stains are not simply "barely visible" or "faint" in color, or "just a cosmetic issue" as fluoridation promoters call it. Common sense tells us that if fluorides affect the teeth, which are the hardest surfaces of the body to cause permanent staining, certainly other soft tissue organs in the body are affected. Also, the National Research Council of the National Academy of Sciences has designated kidney patients, diabetics, seniors, and babies as "susceptible subpopulations" that are especially vulnerable to harm from ingested fluorides. Black citizens are disproportionately affected by kidney disease and diabetes, and are therefore more impacted by fluorides.

Third, we cannot control the dose of fluoride people ingest if we put fluoride in drinking water. Layered on top of this, we do not know what each person's medical history or nutritional status is. Therefore, the "one size fits all" approach to fluoridation makes no sense.

We need to focus on helping people get access to dentists. Lack of fluoride does not cause cavities. Too many sugars on the teeth, lack of access to dental care, and lack of dental health education–these cause cavities.

We also need to know why the full story about harm from fluorides is only just now coming out. I support the holding of Fluoridegate hearings at the state and national level so we can learn why we haven't been openly told that fluorides build up in the body over time, why our government agencies haven't told the black community openly that fluorides disproportionately harm black Americans, and why we've been told that decades of extensive research show fluoridation to be safe, when the National Research Council in 2006 listed volumes of basic research that has never been done. This is a serious issue for all Americans, of every race and in every location.

Sincerely,

Servant/ Leader Gerald L. Durley

2295 Benjamin E. Mays Drive, SW • Atlanta, Georgia 30311
Phone: *404-752-6869* • FAX: *404-752-5284*
www.pmbcatlanta.org

Civil Rights Violation Regarding Forced Medication

LEAGUE of UNITED LATIN AMERICAN CITIZENS

WHEREAS, the League of United Latin American Citizens is this nation's oldest and largest Latino organization, founded in Corpus Christi, Texas on February 17, 1929; and

WHEREAS, LULAC throughout its history has committed itself to the principles that Latinos have equal access to opportunities in employment, education, housing and healthcare; and

WHEREAS, LULAC advocates for the well-being of, but not exclusively of, Hispanics throughout our country; and

WHEREAS, safe drinking water is a necessity for life; and

WHEREAS, the purpose of a public water supply is to supply water to the entire community which is composed of people with varying health conditions, in varying stages of life, and of varying economic status; not to forcibly mass medicate the population which is a civil rights violation; and

WHEREAS, fluoridation is mass medication of the public through the public water supply; and

WHEREAS, current science shows that fluoridation chemicals pose increased risk to sensitive subpopulations, including infants, the elderly, diabetics, kidney patients, and people with poor nutritional status; and

WHEREAS, minority communities are more highly impacted by fluorides as they historically experience more diabetes and kidney disease; and

WHEREAS, minorities are disproportionately harmed by fluorides as documented by increased rates of dental fluorosis (disfiguration and discoloration of the teeth); and

WHEREAS, the National Research Council in 2006 established that there are large gaps in the research on fluoride's effects on the whole body; a fact that contradicts previous assurances made by public health officials and by elected officials, that fluorides and fluoridation have been exhaustively researched; and

WHEREAS, a growing number of cities and health professionals have rejected fluoridation based on current science and the recognition of a person's right to choose what goes into his/her body; and

WHEREAS, the CDC now recommends that non-fluoridated water be used for infant formula (if parents want to avoid dental fluorosis – a permanent mottling and staining of teeth), which creates an economic hardship for large numbers of families, minority and otherwise; and

WHEREAS, the League of United Latin American Citizens (LULAC), founded in 1929, has historically been a champion of the disenfranchised and a leader in the fight for social and environmental justice; and

WHEREAS, City Council Districts I-6 of San Antonio (predominantly minority districts) voted overwhelmingly that the public water supply should not be contaminated with fluoridation chemicals; and

WHEREAS, the election to fluoridate the water, essentially disenfranchised the right of these minority Districts to safe drinking water for all; and

WHEREAS, the U.S. Health and Human Services and the EPA (January 2011) have recently affirmed the NRC Study results that citizens may be ingesting too much fluoride and that the exposure is primarily from drinking water; and

WHEREAS, the proponents of fluoridation promised a safe and effective dental health additive, but the San Antonio Water System's

(SAWS) contract for fluoridation chemicals proves a "bait and switch"; as SAWS is adding the toxic waste by-product of the phosphate fertilizer industry, that has no warranty for its safety and effectiveness for any purpose from the supplier (PENCCO, Inc.) or the source (Mosaic Chemical); and

THEREFORE, BE IT RESOLVED, that LULAC commends efforts by organizations that oppose forced mass medication of the public drinking supplies using fluorides that are industrial grade, toxic waste by-products which contain contaminants (arsenic, lead, mercury) which further endanger life; and

BE IT FURTHER RESOLVED, that LULAC supports efforts by all citizens working to stop forced medication through the public water system because it violates civil rights; and

BE IT FURTHER RESOLVED, that LULAC opposes the public policy of fluoridation because it fails to meet legislative intent; and

BE IT FURTHER RESOLVED, that LULAC demands to know why government agencies entrusted with protecting the public health are more protective of the policy of fluoridation than they are of public health.

Approved this 1st day of July 2011.

Margaret Moran
LULAC National President

New Evidence Confirms Dentists Controlled Statements by CDC On Fluoridation Toxicity Concerns

Planned Parenthood, founded by Margaret Sanger, once advocated "Birth Control" water. Some scientific studies show that flouride can be harmful to the human reproductive process – Alveda King

FOR IMMEDIATE RELEASE
June 22, 2011

CONTACT: Daniel G. Stockin, MPH The Lillie Center, Inc. P.O. Box 839 Ellijay GA 30540
Ph: 706-669-0786 email: stockin2@yahoo.com web: www.SpotsOnMyTeeth.com

Another King Family Member Speaks Out as Fluoridegate Scandal Builds in Atlanta

Ellijay, GA – Swirling questions about conflicts of interest and improper influence grew rapidly today as Freedom of Information Act documents showed that since the 1970s, dental health professionals alone in the Centers for Disease Control (CDC) have controlled the agency's stance supporting water fluoridation.

A response to a request for the names and job descriptions of all persons in CDC that have had input into CDC's decision to support fluoridation listed no CDC toxicologists, minority health professionals, experts in diabetes, or others outside the Oral Health Division.

CDC says its administrative structure is set up to address what the agency calls "cross cutting issues." Yet only CDC's directors of oral health were listed over several decades as being responsible for the agency's fluoridation stance, a disquieting disclosure for water, health, and political leaders that believed CDC utilized its broad array of internal expertise in assessing research on whole-body, outside-the-mouth harm from fluoridation.

The documentation intensifies focus on the motivations behind CDC's and EPA's fluoride safety statements that appear at odds with current scientific knowledge.

After a 2006 report from the National Research Council documented extensive amounts of basic research never conducted on whole-body fluoride impacts, CDC continued promoting fluoridation while stating on its website, "Extensive research conducted over the past 60 years has shown that fluoridation of public water supplies is safe and effective for all community residents."

The disclosures come as yet another prominent member of the Atlanta black community is calling for a halt to water fluoridation and highlighting concerns about the CDC's role in promoting it.

Alveda King, nationally known minister and niece of civil rights leader Martin Luther King Jr., joins the civil rights leader's daughter, Bernice King, former Atlanta mayor and U.N. ambassador Andrew Young, and civil rights leader and minister Dr. Gerald Durley in drawing attention to risks from fluoridation.

Alveda King posted information on her blog today. "The Centers for Disease Control has clearly been trying to preserve fluoridation at all costs, but the facts about fluoride harm are coming out anyway," she says.

"This is a civil rights issue," she continues. "No one should be subjected to drinking fluoride in their water, especially sensitive groups like kidney patients and diabetics, babies in their milk formula, or poor families that cannot afford to purchase unfluoridated water. Black and Latino families are being disproportionately harmed."

A growing body of published research shows that minorities, kidney patients, diabetics, babies and seniors are particularly at risk for harm from ingested fluorides.

Law firms are now reviewing old and new documents believed to highlight a pattern of attempts to curtail discussions on fluoride toxicity and downplay the importance of professionals personally reviewing scientific reports about fluorides.

One such document is an explosive transcript of a 1951 meeting of state dental directors on file at the Library of Congress.

State dental leaders at the meeting were encouraged to promote fluoridation were told, "The question of toxicity is on the same order. Lay off it altogether. Just pass it over. 'We know there is absolutely no effect other than reducing tooth decay,' you say, and go on. If it becomes an issue, then you will have to take it over, but don't bring it up yourself."

A white paper issued by the American Dental Association in 1979 stated that, "Individual dentists must be convinced that they need not be familiar with scientific reports of laboratory and field investigations on fluoridation to be effective participants in the promotion program and that nonparticipation is an overt neglect of professional responsibility."

"I think it's pretty clear that the public, the media, and health providers were given soothing talking points about fluoridation, and in many cases dissuaded from personally looking at toxicity data," says Daniel G. Stockin, a career public health professional who is opposed to fluoridation.

"How can CDC oral health professionals in a department that has promoted fluoridation for decades be objective, let alone competent to assess research and draw conclusions about the toxicity of fluorides on thyroid glands, kidneys, and the pineal gland?" he asks.

"There is a reason we're seeing calls for Fluoridegate investigations," Stockin continues. "The legal community and the media are waking up to this. I believe jurors will see a clear pattern of disinformation, half-truths, misdirection, and omission of critical material facts concerning harm from fluoridated drinking water."

###

Reference Links / Sources:

* Freedom of Info. Act Request & Response
* CDC statement on structure set up to address cross cutting issues
* CDC's "60 years of extensive research" statement (see "Safety and Fluoridation")
* Alveda King's blog: see June 22, 2011 post
* National Research Council report on fluorides: see "Susceptible Subpopulations" pp. 350-51, Harm to minorities – see Table 23 from CDC MMWR publication and other research references
* Transcript of 1951 meeting of state dental directors (see p. 23), Original document on file at Library of Congress: call number RK21.C55 LC, control no.: 59062243. LCCN permalink Meeting name: Conference of the State and Territorial Dental Directors with the Public Health Service and the Children's Bureau. Main title: Proceedings. Published/Created: [Washington] U. S. Dept. of Health, Education, and Welfare.
* ADA White Paper on Fluoridation: see bottom p. 10
* Atlanta leaders on fluoridation: Bernice King Facebook page, see May 10, 2011 post; Rev. Durley & Ambassador Young
* Fluoridation Litigation Article in American Association for Justice newsletter

Related posts:

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This entry was posted on Friday, June 24th, 2011 at 12:05 PM and is filed under Medical, Planned Parenthood. You can follow any responses to this entry through the RSS 2.0 feed. You can leave a response, or trackback from your own site.

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Website

DECEMBER 21, 2011

Black Pastors Speak Out Against Fluoride In Water

MEMPHIS - Rev. William Owens, President of the Coalition of African American Pastors, is joining a growing chorus of leaders calling for federal and state hearings and investigations into new revelations about risks from drinking fluoridated water.

Owens wants to know why important fluoride information wasn't shared with African American leaders and citizens, and is especially concerned about fluoride impacts on poor and inner city families.

"There are so many issues that need to be addressed in Fluoridegate investigations," he states. "This is very disturbing."

"Why wasn't the black community told that blacks are disproportionately impacted by harm from fluorides and fluoridated water? Nobody told me until I learned about this a year ago. African Americans have more kidney disease and more diabetes, but nobody elected to tell us that kidney patients and diabetics are more susceptible to harm from ingested fluorides."

In 2006 the National Research Council published a report that listed diabetics, kidney patients, babies and children, seniors, and outdoor workers as "susceptible subpopulations" that are especially vulnerable to harm from fluorides.

Owens also wants to know why the Centers for Disease Control didn't issue a press release when it changed its stance about mixing baby formula with fluoridated water after the NRC report was issued.

Shortly after the NRC report was published the CDC posted deep inside its website a statement about children potentially developing permanent staining of teeth called "dental fluorosis" if fluoridated water is used to mix baby formula.

"Do you know how many millions of moms and dads are going to their kitchen sink every day to get water for baby formula? This affects a lot of people. Why no press release?" he asks.

"What are poor families to do if they can't afford unfluoridated water? How are they to learn the information about fluorosis? How are they to pay to repair the stains on their teeth?"

"What about the kidney risks and concerns about IQ impacts?" he continues. "Does the Center for Disease Control really think that fluorides miraculously only affect teeth in the mouth and don't impact the rest of the body?"

Owens is an outspoken advocate for assisting children in their education and is concerned about reports of IQ impacts from children ingesting fluorides.
A recent study published in Environmental Health Perspectives, a publication of the National Institute for Environmental Health Sciences, documented diminished IQ in children from fluorides in water.

In 2009 a study in the Journal of Public Health Dentistry noted that black children ingest significantly more fluorides than white children.
The 2006 NRC report included a statement that, "More research is needed to clarify fluoride's biochemical effects on the brain."

CDC's Morbidity and Mortality Weekly Report published data in 2005 showing that blacks have significantly more of the worst forms of dental fluorosis than whites. Owens wants to know why African Americans leaders weren't openly given this and other important information.

The U.S. Department of Health and Human Services proposed somewhat reducing the level of fluoride in water in a statement on January 7, 2011.
The HHS actions don't go far enough, according to Rev. Owens, citing the issue that some people drink dramatically more water than others, have medical susceptibilities to fluorides, and have numerous other uncontrolled sources of fluoride in their diet.

"We need to investigate this Fluoridegate mess. This is a civil rights and environmental justice issue. We don't need just a little less fluoride in water. Fluoridation needs to end," he says firmly.

STORY TAGS: BLACK NEWS, AFRICAN AMERICAN NEWS, MINORITY NEWS, CIVIL RIGHTS NEWS, DISCRIMINATION, RACISM, RACIAL EQUALITY, BIAS, EQUALITY, AFRO AMERICAN NEWS

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Civil Rights Leaders Call End To Water Fluoridation

CANTON, NY - Because fluoride can disproportionately harm poor citizens and black families, Atlanta civil rights leaders, Andrew Young and Dr. Gerald Durley, have asked Georgia legislators to repeal the state's mandatory water fluoridation law.

Andrew Young, former U.N. Ambassador and former Atlanta Mayor, along with Reverend Dr. Gerald Durley, Pastor of Providence Baptist Church in Atlanta, both inductees in the International Civil Rights Walk of Fame, expressed concerns about the fairness, safety, and full disclosure regarding fluoridation in letters to the state's minority and majority legislative leaders.

Fluoride chemicals, added to 96% of Georgia's public drinking water supplies are meant to prevent tooth decay, especially in the poor. Yet, 61% of low-income Georgia third-graders have tooth decay compared to 51% from higher income families - and 33% and 20%, respectively, have untreated cavities showing a dire need for dental care.

"We also have a cavity epidemic today in our inner cities that have been fluoridated for decades," wrote Ambassador Young.

Studies show that despite fluoridation, tooth decay is higher in blacks (4) along with fluoride overexposure symptoms - dental fluorosis or discolored teeth.(5)

Dr. Durley wrote, "The National Research Council (NRC) of the National Academy of Sciences has designated kidney patients, diabetics, seniors, and babies as 'susceptible subpopulations' that are especially vulnerable to harm from ingested fluorides. Black citizens are disproportionately affected by kidney disease and diabetes, and are therefore more impacted by fluorides."

Ambassador Young wrote, "I am most deeply concerned for poor families who have babies: if they cannot afford unfluoridated water for their babies' milk formula, do their babies not count? Of course they do. This is an issue of fairness, civil rights, and compassion. We must find better ways to prevent cavities, such as helping those most at risk for cavities obtain access to the services of a dentist."

Dr. Durley's letter to the legislators also says, "I support the holding of Fluoridegate hearings at the state and national level so we can learn why we haven't been openly told that fluorides build up in the body over time (and) why our government agencies haven't told the black community openly that fluorides disproportionately harm black Americans..."

An American Association for Justice Newsletter for trial lawyers describes potential fluoride legal actions based on personal injury, consumer fraud, and civil rights harm.

In a letter to their state's Health Commissioner, a bipartisan group of Tennessee legislators expressed their concern about fluoridation's undesirable impact on babies and other groups.

A bipartisan group of New York City Council Members has also introduced legislation to stop fluoridation in NYC.

Daniel G. Stockin of The Lillie Center Inc., a Georgia-based firm working to end the practice of fluoridation says, "You can look for even more leaders and persons harmed by fluoridation to speak out now."

The Department of Health and Human Services (HHS) proposes to lower water fluoride levels to alleviate the growing dental fluorosis epidemic. The Fluoride Action Network (FAN) submitted scientific evidence to HHS indicating that fluoridation must stop completely to preserve health, documenting that:

- HHS has failed to consider fluoride's impact on the brain. Fluoride has been linked to lowered IQ in 24 human studies, and over 100 animal studies have reported damage to the brain.
- Infants who are fed formula made with fluoridated tap water will receive up to 175 times more fluoride than breast-fed infants. Infants 0-6 months old, the smallest and most vulnerable in our population, were completely excluded from risk calculations in HHS's proposal.
- African-American children and low-income children suffer from the highest rates of dental fluorosis, including the most severe forms of the condition. The HHS has failed to take any steps to redress this inequity, thereby making fluoridation an Environmental Justice issue.

Young stated, "My father was a dentist. I formerly was a strong believer in the benefits of water fluoridation for preventing cavities. But many things that we began to do 50 or more years ago we now no longer do, because we have learned further information that changes our practices and policies. So it is with fluoridation."

Paul Connett, PhD, Director of FAN says "Fluoridation is unnecessary, unethical, the benefits wildly exaggerated and the risks minimized."

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FEDERAL AGENCIES AND FLUORIDE

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Fluoride - Not FDA Approved for Ingestion
By Sally Stride
June 25, 2004

Amazingly enough, children's sodium fluoride anti-cavity drug products have never been found safe or effective by the Food and Drug Administration (FDA). They were never even tested. The reason will astound you.

As you already know, sodium-fluoride drug products are routinely given to children to prevent tooth decay. They are drugs requiring a dentist's or physician's prescription.

Earlier this year, I wrote the FDA in order to find out why these drugs are not listed on the FDA's approved drug list. My exchange of e-mail correspondence with the FDA (see below) shows that fluoride supplements were "grandfathered in" before the 1938 law requiring drug testing was enacted.

Drug products on the market prior to 1938 were presumed safe by the FDA and were allowed to be sold without any testing. As long as they meet certain conditions, these pre-1938 drugs can continue to be sold without the post-1938 testing requirement. However, once a drug is on the market for any reason, doctors can use that drug to treat any disease or condition.

But in the case of sodium fluoride, its continued, post-1938 use defies logic. Sodium fluoride was on the market prior to 1938, but it was not used at that time to stop cavities, nor was it used for any medical reason. Sodium fluoride was sold as a rat poison. So, in effect, the FDA says that since sodium fluoride was used to kill rats safely and effectively before 1938, the FDA considers it is safe to give to children to prevent tooth decay.

Over 91% of U.S. fluoridating communities now use cheaper silicofluorides , another chemical which was never approved by the FDA. This chemical, too, was never safety tested in animals nor in humans, but it was recently found to increase children's blood lead levels.

And how times change! The following statement is from a 1951 American Dental Association brochure: "There is no proof that commercial preparations such as tablets, dentifrices, mouthwashes or chewing gum containing fluorides are effective in preventing dental decay. Unfortunately such preparations are being offered to the public without adequate scientific evidence of their value."

So, it was in this context and with this knowledge that I wrote the FDA on March 5, 2004, by e-mail:

"Comments: I don't see fluoride supplements, which require a prescription, listed on your approved drugs list. They are prescribed to children to prevent tooth decay. Why aren't they approved? They aren't nutritional supplements, so they can't be excluded. Is it safe to give children drugs that haven't been FDA approved?

Fairly quickly, four days later, the FDA responded to my e-mail as follows:

"Subject: RE: DrugInfo Comment Form FDA/CDER Site

Date: 3/9/2004 3:56:03 PM

From: DRUGINFO@cder.fda.gov

Sodium fluoride has been marketed in the United States since before 1938, when the Food, Drug, and Cosmetic Act (the Act) was enacted. The Act is the basic food and drug law of the United States and is intended to assure the consumer that foods are pure and wholesome, safe to eat, and produced under sanitary conditions; that drugs and devices are safe and effective for their intended uses; that cosmetics are safe and made from appropriate ingredients; and that all labeling and packaging is truthful, informative, and not deceptive.

With the passage of the Act, an approved New Drug Application (NDA) was required for marketing any new drug product (drug products introduced after 1938), as the regulatory mechanism for ensuring that all new drugs were cleared for safety prior to distribution. An amendment to the Act in 1962 required that, before marketing a drug, a manufacturer also had to provide substantial evidence of effectiveness for the product's intended uses. Drugs on the market prior to enactment of the 1938 law were exempted, or "grandfathered," and manufacturers were not required to file an NDA. The premise was that all pre-1938 drugs were considered safe, and if the manufacturer did not change the product formulation or indication, then an NDA was not required. However, once a manufacturer made any change to a pre-1938 drug, that drug was considered by the FDA to be a "new drug" and the manufacturer was required to prove that the drug was safe for its intended use.

The FDA is aware of sodium fluoride-containing products in various dosage forms that are currently marketed. At the present time, the FDA is deferring any regulatory action on sodium fluoride products that were marketed prior to 1962 as long as the currently marketed product is identical to the pre-1962 product.

Any prescription sodium fluoride-containing product coming into the marketplace after 1962 that is not identical to the pre-1962 labeling and that has drug claims, is subject to the FDA drug review process prior to marketing. Drug sponsors, generally manufacturers, develop new drugs, from the earliest laboratory discoveries through various phases of animal and human safety testing as well as clinical testing for effectiveness and appropriate dosing.

The FDA reviews data collected during drug testing at two key points: first, at the time the sponsor believes that the drug is ready for human testing and submits an Investigational New Drug Application (IND); and second, at the time the sponsor submits an NDA for approval to market the drug product. Before the FDA will permit testing of a drug in humans (clinical trials), the sponsor must provide us information in an IND demonstrating that the drug is reasonably safe to administer to humans. The sponsor must also provide manufacturing and control data, a detailed plan for clinical trials, and the names and qualifications of the investigators who will be performing the clinical trials.

Not all oral vitamins are prescription drugs. If the preparation contains 1mg or more of folic acid, then it is prescription. They are indicated for a variety of reasons but mainly to maintain normal blood levels and, therefore, prevent a variety of clinical conditions associated with vitamin deficiencies. If a patient is already deficient, then they will need more than the RDA to replete body stores of the deficient vitamin(s). Certain inborn errors of metabolism require treatment with specific vitamins.

Thank you Bd100 CDER Drug Information."

The next day, I e-mailed the FDA back:

"Wednesday, March 10, 2004 8:29 AM

To: DRUGINFO@cder.fda.gov

Subject: Re: DrugInfo Comment Form FDA/CDER Site

Thank you for your very detailed answer . Sodium fluoride supplements weren't tested as a decay preventative until the 1950's or 1960's. The sodium fluoride on the market before 1938 was sold as a rat poison. Were there any other medicinal reasons for using sodium fluoride before 1938? Thank you. Sally"

The FDA's final response to me was as follows:

"Subject: RE: DrugInfo Comment Form FDA/ CDER Site

Date: 3/18/2004 1:17:15 PM Eastern Standard Time

From: DRUGINFO@cder.fda.gov

Reply To: To: Suite1oh1@aol.com

We don't have information on the medical uses of fluoride before 1938.

Thank you bd100 CDER Drug Information"

The campaign to fluoridate the nation is in full swing by fluoridation promoters. They are continuing to downplay or ignore the mounting evidence against fluoridation. If they succeed, then it would be a disaster for the nation, both from the standpoint of health and that of health freedoms. It is very important for all of you to send letters to the editor as well as letters to their legislators on all levels: federal, state, and local. It is best if you express your concerns in a polite, sincere way but with conviction, and ask your legislators to take action, to curb and stop the implementation of fluoride programs, implementation and funding of fluoridation, fluoride programs, and at least declare a moratorium on fluoridation and ultimately halt fluoridation wherever it exists. One person can and does make a difference, sometimes THE difference.

"It is not the function of our Government to keep the citizen from falling into error, It is the function of the citizen to keep the Government from falling into error."
– Robert Houghwout Jackson,
Chief Judge at the War-Crimes Tribunal in Nuremberg

CDC Water Fluoridation Stand Long Influenced By Dentists

By Dr.Mercola
Mercola.com, July 23, 2011
Straight to the Source

For related articles and more information, please visit OCA's Health Issues page and our Appetite For a Change page.

Documents released under the Freedom of Information Act show that since the 1970's, the dental health professionals in the Centers for Disease Control (CDC) have completely controlled the agency's stance supporting water fluoridation. No CDC toxicologists, minority health professionals, experts in diabetes, or others outside the Oral Health Division had any input into the agency's position.

The documents have drawn attention once again to the CDC's and EPA's fluoride safety statements, which appear at odds with current scientific knowledge.

According to the Fluoride Action Network:

"Law firms are now reviewing old and new documents believed to highlight a pattern of attempts to curtail discussions on fluoride toxicity and downplay the importance of professionals personally reviewing scientific reports about fluorides."

Sources: Fluoride Action Network June 22, 2011

Dr. Mercola's Comments:
The Centers for Disease Control and Prevention (CDC) is part of a larger administrative structure that provides intra-agency support and resource sharing for health issues that require the input from more than one area of expertise. Other offices that share information and expertise with the CDC include the National Center for Chronic Disease Prevention and Health Promotion, Office of Minority Health and Health Equity, and the Agency for Toxic Substances.

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Proposed HHS Recommendation for Fluoride Concentration in Drinking Water for Prevention of Dental Caries

DISCLAIMER: The Secretary of Health and Human Services (HHS) signed the following notice on January 7, 2011, and HHS is submitting it for publication in the Federal Register. Steps have been taken to ensure the accuracy of the pre-publication version, however, it is not the official version. Please refer to the official version in a forthcoming Federal Register publication, or the U.S. Government Printing Office (GPO) website at http://www.gpoaccess.gov/fr/index.html.

Department of Health and Human Services

Proposed HHS Recommendation for Fluoride Concentration in Drinking Water for Prevention of Dental Caries

AGENCY: Department of Health and Human Services, Office of the Secretary.

ACTION: Notice.

SUMMARY: The Department of Health and Human Services (HHS) seeks public comment on proposed new guidance which will update and replace the 1962 U.S. Public Health Service Drinking Water Standards related to recommendations for fluoride concentrations in drinking water. The U.S. Public Health Service recommendations for optimal fluoride concentrations were based on ambient air temperature of geographic areas and ranged from 0.7 – 1.2 mg/L.

HHS proposes that community water systems adjust the amount of fluoride to 0.7 mg/L to achieve an optimal fluoride level. For the purpose of this guidance, the optimal concentration of fluoride in drinking water is that concentration that provides the best balance of protection from dental caries while limiting the risk of dental fluorosis. Community water fluoridation is the adjusting and monitoring of fluoride in drinking water to reach the optimal concentration (Truman BI, et al, 2002).

This updated guidance is intended to apply to community water systems that are currently fluoridating or will initiate fluoridation.[1] This guidance is based on several considerations that include:

- Scientific evidence related to effectiveness of water fluoridation on caries prevention and control across all age groups
- Fluoride in drinking water as one of several available fluoride sources
- Trends in the prevalence and severity of dental fluorosis
- Current evidence on fluid intake in children across various ambient air temperatures.

DATES: To receive consideration, comments on the proposed recommendations for fluoride concentration in drinking water for the prevention of dental caries should be received no later than **[INSERT DATE 30 DAYS FROM DATE OF PUBLICATION IN THE FEDERAL REGISTER]**.

ADDRESSES: Comments are preferred electronically and may be addressed to CWFcomments@cdc.gov. Written responses should be addressed to the U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, CWF Comments, Division of Oral Health, National Center for Chronic Disease Prevention and Health Promotion (NCCDPHP), 4770 Buford Highway, NE, MS F-10, Atlanta, GA 30341-3717.

FOR FURTHER INFORMATION CONTACT: Barbara F. Gooch, Associate Director for Science (Acting), 770-488-6054, CWFcomments@cdc.gov, Division of Oral Health, National Center for Chronic Disease Prevention and Health Promotion (NCCDPHP), Centers for Disease Control and Prevention, 4770 Buford Highway, NE, MS F-10, Atlanta, GA 30341-3717.

SUPPLEMENTARY INFORMATION:

The U.S. Public Health Service has provided recommendations regarding optimal fluoride concentrations in drinking water from community water systems (CWS)[2] for the prevention of dental caries (US DHEW, 1962). HHS proposes to update and replace these recommendations because of new data that address changes in the prevalence of dental fluorosis, fluid intake among children, and the contribution of fluoride in drinking water to total fluoride exposure in the United States. As of December 31, 2008, the Centers for Disease Control and Prevention (CDC) estimated that 16,977 community water systems provided fluoridated water to 196 million people. 95% of the population receiving fluoridated water was served by community water systems that added fluoride to water, or purchased water with added fluoride from other systems. The remaining 5% were served by systems with naturally occurring fluoride at or above the recommended level. More statistics about water fluoridation in the United States are available at http://www.cdc.gov/fluoridation/statistics/2008stats.htm. Guidance for systems with naturally occurring fluoride levels above the recommended level are beyond the scope of this document. Systems that have fluoride levels greater than the national primary (4.0 mg/L) or secondary (2.0 mg/L) drinking water standards established by EPA can find more information at the following EPA web site:
http://water.epa.gov/drink/contaminants/basicinformation/fluoride.cfm. CDC's Recommendations for Fluoride Use (CDC, 2001b), available at http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5014a1.htm, provides guidance on community water fluoridation and use of other fluoride-containing products.

Recommendation

HHS proposes that community water systems adjust their fluoride content to 0.7 mg/L [parts per million (ppm)].

Rationale

Importance of community water fluoridation:

Community water fluoridation is a major factor responsible for the decline of the prevalence and severity of dental caries (tooth decay) during the second half of the 20th century. From the early 1970's to the present, the prevalence of dental caries in at least one permanent tooth (excluding third molars) among adolescents, aged 12 – 17 years, [3] has decreased from 90% to 60% and the average number of teeth affected by dental caries (i.e., decayed, missing and filled) from 6.2 to 2.6 (Kelly JE, 1975, Dye B, et al, 2007). Adults have also benefited from community water fluoridation. Among adults, aged 35 – 44 years,[4] the average number of affected teeth decreased from 18 in the early 1960's to 10 among adults, aged 35 – 49 years, in 1999-2004 (Kelly JE, et al, 1967; Dye B, et al, 2007). Although there have been notable declines in tooth decay, it remains one of the most common chronic diseases of childhood (USDHHS, 2000; Newacheck PW et al, 2000). Effective population-based interventions to prevent and control dental caries, such as community water fluoridation, are still needed (CDC, 2001a).

Systematic reviews of the scientific evidence related to fluoride have concluded that community water fluoridation is effective in decreasing dental caries prevalence and severity (McDonagh MS, et al, 2000a, McDonagh MS, et al, 2000b, Truman BI, et al, 2002, Griffin SO, et al, 2007). Effects included significant increases in the proportion of children who were caries-free and significant reductions in the number of teeth or tooth surfaces with caries in both children and adults (McDonagh MS, et al, 2000b, Griffin SO, et al, 2007). When analyses were limited to studies conducted after the introduction of other sources of fluoride, especially fluoride toothpaste, beneficial effects across the lifespan from community water fluoridation were still apparent (McDonagh MS, et al, 2000b; Griffin SO, et al, 2007).

Fluoride works primarily to prevent dental caries through topical remineralization of tooth surfaces when small amounts of fluoride, specifically in saliva and accumulated plaque, are present frequently in the mouth (Featherstone JDB, 1999). Consuming fluoridated water and beverages and foods prepared or processed with fluoridated water routinely introduces a low concentration of fluoride into the mouth. Although other fluoride-containing products are available and contribute to the prevention and control of dental caries, community water fluoridation has been identified as the most cost-effective method of delivering fluoride to all members of the community regardless of age, educational attainment, or income level (CDC, 1999, Burt BA, 1989). Studies continue to find that community water fluoridation is cost-saving (Truman B, et al, 2002).

Trends in availability of fluoride sources:

Community water fluoridation and fluoride toothpaste are the most common sources of non-dietary fluoride in the United States (CDC, 2001b). Community water fluoridation began in 1945, reaching almost 50% of the U.S. population by 1975 and 64% by 2008, http://www.cdc.gov/fluoridation/statistics/2008stats.htm; http://www.cdc.gov/fluoridation/pdf/statistics/1975.pdf. Toothpaste containing fluoride was first marketed in the United States in 1955 (USDHEW, 1980) and by the 1990's accounted for more than 90 percent of the toothpaste market (Burt BA and Eklund SA, 2005). Other products that provide fluoride now include mouthrinses, fluoride supplements, and professionally applied

fluoride compounds. More detailed explanations of these products are published elsewhere (CDC, 2001b) (ADA, 2006) (USDHHS, 2010). More information on all sources of fluoride and their relative contribution to total fluoride exposure in the United States is presented in a report by EPA (US EPA 2010a).

Dental fluorosis:

Fluoride ingestion while teeth are developing can result in a range of visually detectable changes in the tooth enamel (Aoba T and Fejerskov O, 2002). Changes range from barely visible lacy white markings in milder cases to pitting of the teeth in the rare, severe form. The period of possible risk for fluorosis in the permanent teeth, excluding the third molars,[5] extends from about birth through 8 years of age when the preeruptive maturation of tooth enamel is complete (CDC, 2001b; Massler M and Schour I, 1958). When communities first began adding fluoride to their public water systems in 1945, drinking water and foods and beverages prepared with fluoridated water were the primary sources of fluoride for most children (McClure FJ, 1943). Since the 1940's, other sources of ingested fluoride, such as fluoride toothpaste (if swallowed) and fluoride supplements, have become available. Fluoride intake from these products, in addition to water and other beverages and infant formula prepared with fluoridated water, have been associated with increased risk of dental fluorosis (Levy SL, et al, 2010, Wong MCM, et al, 2010, Osuji OO et al, 1988, Pendrys DG et al, 1994, Pendrys DG and Katz RV 1989, Pendrys DG, 1995). Both the 1962 USPHS recommendations and the current proposal for fluoride concentrations in community drinking water were set to achieve a reduction in dental caries while minimizing the risk of dental fluorosis.

Results of two national surveys indicate that the prevalence of dental fluorosis has increased since the 1980's, but mostly in the very mild or mild forms. The most recent data on prevalence of dental fluorosis come from the National Health and Nutrition Examination Survey (NHANES), 1999-2004. NHANES assessed the prevalence and severity of dental fluorosis among persons, aged 6 to 49 years. Twenty-three percent had dental fluorosis of which the vast majority was very mild or mild. Approximately 2% of persons had moderate dental fluorosis, and less than 1% had severe. Prevalence was higher among younger persons and ranged from 41% among adolescents aged 12 – 15 years to 9% among adults, aged 40 – 49 years. The higher prevalence of dental fluorosis in the younger persons probably reflects the increase in fluoride exposures across the U.S. population through community water fluoridation and increased use of fluoride toothpaste.

The prevalence and severity of dental fluorosis among 12 – 15 year olds in 1999-2004 were compared to estimates from the Oral Health of United States Children Survey, 1986-87, which was the first national survey to include measures of dental fluorosis. Although these two national surveys differed in sampling and representation (schoolchildren versus household), findings support the hypothesis that there has been an increase in dental fluorosis that was very mild or greater between the two surveys. In 1986-87 and 1999-2004 the prevalence of dental fluorosis was 23% and 41%, respectively, among adolescents aged 12 to 15. (Beltrán-Aguilar ED, et al, 2010a). Similarly, the prevalence of very mild fluorosis (17.2% and 28.5%), mild fluorosis (4.1% and 8.6%) and moderate and severe fluorosis combined (1.3% and 3.6%) have increased.

The estimates for severe fluorosis for adolescents in both surveys were statistically unreliable because of too few cases in the samples.

More information on fluoride concentrations in drinking water and the impact of severe dental fluorosis in children is presented in a report by EPA (US EPA 2010 b).

Relationship between dental caries and fluorosis at varying water fluoridation concentrations:

The 1986-87 Oral Health of United States Children Survey is the only national survey that measured the child's water fluoride exposure and can link that exposure to measures of caries and fluorosis (US DHHS, 1989). An additional analysis of data from this survey examined the relationship between dental caries and fluorosis at varying water fluoride concentrations for children aged 6 to 17 years (Heller KE, et al, 1997). Findings indicate that there was a gradual decline in dental caries as fluoride content in water increased from negligible to 0.7 mg/L. Reductions plateaued at concentrations from 0.7 to 1.2 mg/L. In contrast, the percentage of children with at least very mild dental fluorosis increased with increasing fluoride concentrations in water. The published report did not report standard errors.

In Hong Kong a small change of about 0.2 mg/L[6] in the mean fluoride concentration in drinking water in 1978 was associated with a detectable reduction in fluorosis prevalence by the mid 1980's[7] (Evans R.W, Stamm JW., 1991). Across all age groups more than 90% of fluorosis cases were very mild or mild. (Evans R.W, Stamm JW., 1991). The study did not include measures of fluoride intake. Concurrently, dental caries prevalence did not increase. (Lo ECM et al, 1990). Although not fully generalizable to the current U.S. context, these findings, along with those from the 1986-87 survey of U.S. schoolchildren, suggest that risk of fluorosis can be reduced and caries prevention maintained toward the lower end (i.e., 0.7 mg/L) of the 1962 USPHS recommendations for fluoride concentrations for community water systems.

Relationship of fluid intake and ambient temperature among children and adolescents in the United States:

The 1962 USPHS recommendations stated that community drinking water should contain 0.7-1.2 mg/L [ppm] fluoride, depending on the ambient air temperature of the area. These temperature-related guidelines were based on studies conducted in two communities in California in the early 1950's. Findings indicated that a lower fluoride concentration was appropriate for communities in warmer climates because children drank more tap water on warm days (Galagan DJ, 1953; Galagan DJ and Vermillion JR, 1957; Galagan DJ et al, 1957). Social and environmental changes, including increased use of air conditioning and more sedentary lifestyles, have occurred since the 1950's, and thus, the assumption that children living in warmer regions drink more tap water than children in cooler regions may no longer be valid.

Studies conducted since 2001 suggest that fluid intake in children does not increase with increases in ambient air temperature (Sohn W, et al, 2001; Beltrán-Aguilar ED, et al, 2010b). One study conducted among children using nationally representative data from 1988 to 1994 did not find an association between fluid intake and ambient air temperature (Sohn W, et al, 2001).

A similar study using nationally representative data from 1999 to 2004 also found no association between fluid intake and ambient temperature among children or adolescents (Beltrán-Aguilar ED, et al, 2010b). These recent findings demonstrating a lack of an association between fluid intake among children and adolescents and ambient temperature support use of a single target concentration for community water fluoridation in all temperature zones of the United States.

Conclusions:

HHS recommends an optimal fluoride concentration of 0.7 mg/L for community water systems based on the following information:

- Community water fluoridation is the most cost-effective method of delivering fluoride for the prevention of tooth decay;
- In addition to drinking water, other sources of fluoride exposure have contributed to the prevention of dental caries and an increase in dental fluorosis prevalence;
- Significant caries preventive benefits can be achieved and risk of fluorosis reduced at 0.7 mg/L, the lowest concentration in the range of the USPHS recommendation.
- Recent data do not show a convincing relationship between fluid intake and ambient air temperature. Thus, there is no need for different recommendations for water fluoride concentrations in different temperature zones.

Surveillance activities:

CDC and the National Institute of Dental and Craniofacial Research (NIDCR), in coordination with other federal agencies, will enhance surveillance of dental caries, dental fluorosis, and fluoride intake with a focus on younger populations at higher risk of fluorosis to obtain the best available and most current information to support effective efforts to improve oral health.

Process:

The U.S. Department of Health and Human Services (HHS) convened a federal inter-departmental, inter-agency panel of scientists (Appendix A) to review scientific evidence related to the 1962 USPHS Drinking Water Standards related to recommendations for fluoride concentrations in drinking water in the United States and to update these proposed recommendations. Panelists included representatives from the Centers for Disease Control and Prevention, the National Institutes of Health, the Food and Drug Administration, the Agency for Healthcare Research and Quality, the Office of the Assistant Secretary for Health, the U.S. Environmental Protection Agency, and the U.S. Department of Agriculture. The panelists evaluated existing recommendations for fluoride in drinking water, systematic reviews of the risks and benefits from fluoride in drinking water, the epidemiology of dental caries and fluorosis in the U.S., and current data on fluid intake in children, aged 0 to 10 years, across temperature gradients in the U.S. Conclusions were reached and are summarized along with their rationale in this proposed guidance document. This guidance will be advisory, not regulatory, in nature. Guidance will be submitted to the Federal Register and will undergo public and stakeholder comment for 30 days, after which HHS will review comments and consider changes.

DATED:

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Kathleen Sebelius

Secretary

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Comments regarding the document, Fluoride: Exposure and Relative Source

Contribution Analysis, should be sent to EPA at FluorideScience@epa.gov.

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EPA SCIENTISTS OPPOSING FLUORIDE

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EPA Scientists & Workers Call for an End to Water Fluoridation Because of Cancer Risk

From: Environment News Service <www.ens-newswire.com

FOR IMMEDIATE RELEASE

EPA UNIONS CALL FOR NATIONWIDE MORATORIUM ON FLUORIDATION, CONGRESSIONAL HEARING ON ADVERSE EFFECTS, YOUTH CANCER COVER UP

WASHINGTON, DC, August 30, 2005 --/WORLD-WIRE/-- Eleven EPA employee unions representing over 7000 environmental and public health professionals of the Civil Service have called for a moratorium on drinking water fluoridation programs across the country, and have asked EPA management to recognize fluoride as posing a serious risk of causing cancer in people. The unions acted following revelations of an apparent cover-up of evidence from Harvard School of Dental Medicine linking fluoridation with elevated risk of a fatal bone cancer in young boys.

The unions sent letters to key Congressional committees asking Congress to legislate a moratorium pending a review of all the science on the risks and benefits of fluoridation. The letters cited the weight of evidence supporting a classification of fluoride as a likely human carcinogen, which includes other epidemiology results similar to those in the Harvard study, animal studies, and biological reasons why fluoride can reasonably be expected to cause the bone cancer - osteosarcoma - seen in young boys and test animals.

The unions also pointed out recent work by Richard Maas of the Environmental Quality Institute, University of North Carolina that links increases in lead levels in drinking water systems to use of silicofluoride fluoridating agents with chloramines disinfectant.

The letter to EPA Administrator Stephen Johnson asked him to issue a public warning in the form of an advanced notice of proposed rulemaking setting the health-based drinking water standard for fluoride at zero, as it is for all known or probable human carcinogens, pending a recommendation from a National Academy of Sciences' National Research Council committee. That committee's work is not expected to be done before 2006.

The unions also asked Congress and EPA's enforcement office, or the Department of Justice, to look into reasons why the Harvard study director, Chester Douglass, failed to report the seven-fold increased risk seen in the work he oversaw, and instead wrote to the National Institute of Environmental Health Sciences, the federal agency that funded the Harvard study, saying there was no link between fluoridation and osteosarcoma. Douglass sent the same negative report to the National Research Council committee studying possible changes in EPA's drinking water standards for fluoride.

The unions who signed the letters represent EPA employees from across the nation, including laboratory scientists in Ohio, Oklahoma and Michigan, regulatory support scientists and other workers at EPA headquarters in Washington, D.C. and science and regulatory workers in Boston, New York, Philadelphia, Atlanta, and San Francisco.

They are affiliated with the National Treasury Employees Union, the American Federation of Government Employees, Engineers and Scientists of California/International Federation of Professional and Technical Engineers, and the National Association of Government Employee/Service Employees International Union.

The unions' letter is online at:
http://nteu280.org/Issues/Fluoride/fluoridesummary.htm

FOR INFORMATION CONTACT:

Dr. William Hirzy Vice-President, NTEU Chapter 280
Phone (cell) 202-285-0498 This is complete notice.

USA Citizens can easily back-up the 7,000 EPA 'career employees.' Consider, you and your family need CLEAN & SAFE Drinking Water, every day. This is your opportunity to back those 7000+ EPA union members that care about scientific integrity and exposing the truth about 'corporate hazardous waste fluorosilicates' being metered into our drinking waters. Their common sense demand for a 'moratorium on fluoridation' along with Congressional Investigation incl. 'under oath' hearings is critical!

We, the undersigned, join with members of eleven EPA unions in their call for an immediate Congressional act placing a national moratorium on water fluoridation pending a full Congressional investigation into this public policy, which affects - directly and indirectly - every resident of the United States.

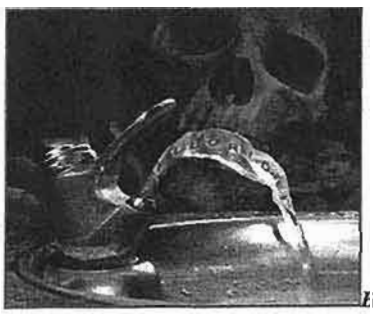
Read & Sign Citizens PETITION at:
http://petition.powalliance.org/index.html

Sunday, October 16, 2011

THE STATESMAN SENTINEL

EPA Scientist: Water Fluoridation Greatest Scientific Fraud of Century

July 21, 2010StatesmanSentinel.com · Comments (5)



By Rebecca Noel

Our Interesting World

The dangers of fluoride have been well known in the alternative health arena for decades but unfortunately the general public en masse has been basically indoctrinated into believing fluoride prevents cavities and is something good for them…

It's been hard for anyone to oppose the propaganda given about fluoride. Many people who speak out against fluoride are losing their jobs and being ostracized by their colleagues. With this in mind, it is no wonder that more people aren't speaking out. Currently, any doctor or dentist who opposes fluoridation would run the risk of losing their license to practice medicine or dentistry.

But more and more people are starting to speak out regardless of job security including the EPA's own scientists! Bravo to these brave men who have listened to their conscience and can no longer participate in the great lie that is poisoning so many innocent people!

For over 60 years the subject of fluoride has been a big controversy. How fluoride got approved started back in the 1940's with an aluminum company called ALCOA (aluminum company of America). This company was facing problems with toxic waste. One of the byproducts of aluminum manufacture is sodium fluoride.

Because of its toxicity, it must be disposed of in the highest rated waste disposal facility. This would be at a tremendous cost. In 1944, a long time ALCOA lawyer named Oscar Ewing was named the company's chief counsel with fees in the then astronomical range of $750,000 a year. In 1947, Ewing was made Federal Security Agency Administrator, with the announcement that he was taking a big cut in salary.

The US Public Health Service, then a division of FSA, came under command of Ewing, and he began to vigorously promote fluoridation nationwide. Ewing's public relations strategist for the fluoride campaign was the nephew of Sigmund Freud, Edward L. Bernays. This was the man who literally wrote the book on propaganda....and I don't mean figuratively. In 1928 Bernays published a book entitled: "Propaganda", in which he describes how the human mind can be manipulated.

This book was recently republished in 2004. It is interesting to note that Bernays was also involved in the promotion of cigarettes. To this day it is not exactly clear how ALCOA garnered the support of the American Medical and Dental Association; however, there may have been other factors involved.

These factors may have involved the fact that mass quantities of fluoride were produced in the production of the atomic bomb. In fact the first litigation over the atomic bomb had nothing to do with radiation....it was fluoride poisoning that caused ill health. It was these factors that may have led to the need to put a friendlier face on fluoride.

It is also noteworthy that fluoridation began at the time of World War II. This was a time when aluminum production was being increased to provide for the production of airplanes and other military needs. It is theorized that this increased need for aluminum production may have paved the way for fluoride's acceptance.

It is also interesting to note that prior to 1940 fluoride was considered a toxic element. In fact in 1939 the U.S. Public Health Service regulations state "the presence of fluorides in excess of 1ppm shall constitute rejection of the water supply"....yet when water fluoridation is instituted these days the levels are set at a minimum of 1ppm.

In fact, an article appearing in the Journal of American Medical Association on September 18, 1943 states, "fluorides are general protoplasmic poisons, changing the permeability of the cell membrane by inhibiting certain enzymes".

In fact, before 1940, fluoride's only approved use was as a rat and cockroach poison. In smaller doses, it was approved for use as a treatment for an overactive thyroid gland but this use was abandoned after many patients suffered destruction of their thyroid gland.

Continue reading

The Frederick Post

Frederick, Maryland Saturday, February 13, 1994

EPA ordered to reinstate Montgomery scientist

Secretary of Labor Robert Reich has ordered the reinstatement of a Montgomery County Scientist who was fired by the Environmental Protection Agency after he opposed the use of fluoride in drinking water.

Dr. William Marcus was awarded back pay for his $87,000 per year job as a senior toxicologist, $50,000 for hardship and other compensation under Mr. Reich's order, issued Monday.

Mr. Reich ratified a Dec. 3, 1992, ruling by administrative law judge, David Clarke that the EPA "retaliated" against Dr. Marcus by firing him in May 1992 for his scientific reports which recommended removing fluoride from drinking water.

Fluoride, Dr. Marcus said in an interview Friday, is "a poison" found to cause liver cancer and other health problems. It also causes lead to leach from plumbing into water supplies, he said.

Dr. Marcus, an EPA scientist for 18 years until he was fired, was protected under federal "whistleblower" laws designed to safeguard employees who report damaging facts about their employer, Mr. Reich said.

The EPA is an independent federal agency, but the Labor Department has jurisdiction in disputes involving its employees.

Before Dr. Marcus was fired, EPA Inspector General John Martin issued a report on him "which contained slanderous, false and derogatory information," said Mr. Reich.

The report's charges were "unsubstantiated," and that "the true reason for the discharge was retaliation", Mr. Reich said.

Dr. Marcus said the report was an attempt to discredit him because he had testified in 38 court cases about the harmful effects of chemical produced by national and international companies.

Dr. Marcus said EPA officials sent out memos calling him "a threat," claiming that he had

carried a gun to work to possibly kill his superior.

Upon learning that Dr. Marcus had filed a lawsuit, EPA officials broke a federal law by shredding documents that would have exonerated him, said Mr. Reich,.

Dr. Marcus was accused of "stealing time" from the government by working as a consultant while on the EPA payroll. His attorneys found that time cards were falsified in the investigation process. Government officials attempted to file charges against him using the fake documents. Dr. Marcus said.

"Fighting the federal government is by no means an easy task," he said describing a two year legal battle with his employer. "Since (EPA Lawyers) have jobs and their job depends not upon doing what is right but on fighting, they will fight forever...It is a vicious circle in which the costs to the taxpayer are never considered."

"There are independent, hard working EPA scientists who are afraid to publish the truth because they're afraid of losing their jobs." Dr. Marcus said. "It has a chilling effect."

EPA officials would not comment on the matter, spokeswoman Denise Graveline said Friday.

Since then our opposition to drinking water fluoridation has grown, based on the scientific literature documenting the increasingly out-of-control exposures to fluoride, the lack of benefit to dental health from ingestion of fluoride and the hazards to human health from such ingestion. These hazards include acute toxic hazard, such as to people with impaired kidney function, as well as chronic toxic hazards of gene mutations, cancer, reproductive effects, neurotoxicity, bone pathology and dental fluorosis. First, a review of recent neurotoxicity research results.

In 1995, Mullenix and co-workers[2] showed that rats given fluoride in drinking water at levels that give rise to plasma fluoride concentrations in the range seen in humans suffer neurotoxic effects that vary according to when the rats were given the fluoride - as adult animals, as young animals, or through the placenta before birth. Those exposed before birth were born hyperactive and remained so throughout their lives. Those exposed as young or adult animals displayed depressed activity. Then in 1998, Guan and co-workers[3] gave doses similar to those used by the Mullenix research group to try to understand the mechanism(s) underlying the effects seen by the Mullenix group. Guan's group found that several key chemicals in the brain - those that form the membrane of brain cells - were substantially depleted in rats given fluoride, as compared to those who did not get fluoride.

Another 1998 publication by Varner, Jensen and others[4] reported on the brain- and kidney damaging effects in rats that were given fluoride in drinking water at the same level deemed "optimal" by pro-fluoridation groups, namely 1 part per million (1 ppm). Even more pronounced damage was seen in animals that got the fluoride in conjunction with aluminum. These results are especially disturbing because of the low dose level of fluoride that shows the toxic effect in rats - rats are more resistant to fluoride than humans. This latter statement is based on Mullenix's finding that it takes substantially more fluoride in the drinking water of rats than of humans to reach the same fluoride level in plasma. It is the level in plasma that determines how much fluoride is "seen" by particular tissues in the body. So when rats get 1 ppm in drinking water, their brains and kidneys are exposed to much less fluoride than humans getting 1 ppm, yet they are experiencing toxic effects. Thus we are compelled to consider the likelihood that humans are experiencing damage to their brains and kidneys at the "optimal" level of 1 ppm.

In support of this concern are results from two epidemiology studies from China[5, 6] that show decreases in I.Q. in children who get more fluoride than the control groups of children in each study. These decreases are about 5 to 10 I.Q. points in children aged 8 to 13 years.

Another troubling brain effect has recently surfaced: fluoride's interference with the function of the brain's pineal gland. The pineal gland produces melatonin which, among other roles, mediates the body's internal clock, doing such things as governing the onset of puberty. Jennifer Luke[7] has shown that fluoride accumulates in the pineal gland and inhibits its production of melatonin. She showed in test animals that this inhibition causes an earlier onset of sexual maturity, an effect reported in humans as well in 1956, as part of the Kingston/Newburgh study, which is discussed below. In fluoridated Newburgh, young girls experienced earlier onset of menstruation (on average, by six months) than girls in non-fluoridated Kingston.[8]

From a risk assessment perspective, all these brain effect data are particularly compelli
because they are convergent.

We looked at the cancer data with alarm as well. There are epidemiology studies that a
whole-animal and single-cell studies (dealing with the cancer hazard), just as the neuro
just mentioned all points in the same direction. EPA fired the Office of Drinking Water
toxicologist, Dr. William Marcus, who also was our local union's treasurer at the time,
remain silent on the cancer risk issue.[9] The judge who heard the lawsuit he brought aga

Dr. William Marcus

see also:

- Statement of Dr. J. William Hirzy, National Treasury Employees Union Chapter 280, before The Subcommittee on Wildlife, Fisheries and Drinking Water, United States Senate, June 29, 2000
- EPA scientists take action against EPA for failing to protect public health -- Important scientific and technical considerations were ignored when the Recommended Maximum Contaminant Level (RMCL) for fluoride was set (1986 Amicus Brief).
- The need for a Code of Ethics at the EPA became critical. Without an enforceable code of ethics with sanctions, the distortion of truth caused by the pressures of politics would continue.
- Environmental Protection Agency Union fights back

CHAPTER 280
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WHY EPA'S HEADQUARTERS UNION OF SCIENTISTS OPPOSES FLUORIDATION

The following documents why our union, formerly National Federation of Federal Employees Local 2050 and since April 1998 Chapter 280 of the National Treasury Employees Union, took the stand it did opposing fluoridation of drinking water supplies. Our union is comprised of and represents the approximately 1500 scientists, lawyers, engineers and other professional employees at EPA Headquarters here in Washington, D.C.

The union first became interested in this issue rather by accident. Like most Americans, including many physicians and dentists, most of our members had thought that fluoride's only effects were beneficial - reductions in tooth decay, etc. We too believed assurances of safety and effectiveness of water fluoridation.

Then, as EPA was engaged in revising its drinking water standard for fluoride in 1985, an employee came to the union with a complaint: he said he was being forced to write into the regulation a statement to the effect that EPA thought it was alright for children to have "funky" teeth. It was OK, EPA said, because it considered that condition to be only a *cosmetic* effect, not an adverse *health* effect. The reason for this EPA position was that it was under political pressure to set its health-based standard for fluoride at 4 mg/liter. At that level, EPA knew that a significant number of children develop moderate to severe dental fluorosis, but since it had deemed the effect as only cosmetic, EPA didn't have to set its health-based standard at a lower level to prevent it.

We tried to settle this ethics issue quietly, within the family, but EPA was unable or unwilling to resist external political pressure, and we took the fight public with a union *amicus curiae* brief in a lawsuit filed against EPA by a public interest group. The union has published on this initial involvement period in detail.[1]

firing made that finding - that EPA fired him over his fluoride work and not for the phony reason put forward by EPA management at his dismissal. Dr. Marcus won his lawsuit and is again at work at EPA. Documentation is available on request.

The type of cancer of particular concern with fluoride, although not the only type, is osteosarcoma, especially in males. The National Toxicology Program conducted a two-year study[10] in which rats and mice were given sodium fluoride in drinking water. The positive result of that study (in which malignancies in tissues other than bone were also observed), particularly in male rats, is convergent with a host of data from tests showing fluoride's ability to cause mutations (a principal "trigger" mechanism for inducing a cell to become cancerous) e.g.[11a, b, c, d] and data showing increases in osteosarcomas in young men in New Jersey[12], Washington and Iowa[13] based on their drinking fluoridated water. It was his analysis, repeated statements about all these and other incriminating cancer data, and his requests for an independent, unbiased evaluation of them that got Dr. Marcus fired.

Bone pathology other than cancer is a concern as well. An excellent review of this issue was published by Diesendorf et al. in 1997.[14] Five epidemiology studies have shown a higher rate of hip fractures in fluoridated vs. non-fluoridated communities.[15a, b, c, d, e] Crippling skeletal fluorosis was the endpoint used by EPA to set its primary drinking water standard in 1986, and the ethical deficiencies in that standard setting process prompted our union to join the Natural Resources Defense Council in opposing the standard in court, as mentioned above.

Regarding the effectiveness of fluoride in reducing dental cavities, there has not been any double-blind study of fluoride's effectiveness as a caries preventative. There have been many, many small scale, selective publications on this issue that proponents cite to justify fluoridation, but the largest and most comprehensive study, one done by dentists trained by the National Institute of Dental Research, on over 39,000 school children aged 5-17 years, shows no significant differences (in terms of decayed, missing and filled teeth) among caries incidences in fluoridated, non-fluoridated and partially fluoridated communities.[16] The latest publication[17] on the fifty-year fluoridation experiment in two New York cities, Newburgh and Kingston, shows the same thing. The only significant difference in dental health between the two communities as a whole is that fluoridated Newburgh, N.Y. shows about twice the incidence of dental fluorosis (the first, visible sign of fluoride chronic toxicity) as seen in non-fluoridated Kingston.

John Colquhoun's publication on this point of efficacy is especially important.[18] Dr. Colquhoun was Principal Dental Officer for Auckland, the largest city in New Zealand, and a staunch supporter of fluoridation - until he was given the task of looking at the world-wide data on fluoridation's effectiveness in preventing cavities. The paper is titled, "Why I changed My Mind About Water Fluoridation." In it Colquhoun provides details on how data were manipulated to support fluoridation in English speaking countries, especially the U.S. and New Zealand. This paper explains why an ethical public health professional was compelled to do a 180 degree turn on fluoridation.

Further on the point of the tide turning against drinking water fluoridation, statements are now coming from other dentists in the pro-fluoride camp who are starting to warn that topical fluoride (e.g. fluoride in tooth paste) is the only significantly beneficial way in which that substance affects dental health.[19, 20, 21] However, if the concentrations of fluoride in the oral cavity are sufficient to inhibit bacterial enzymes and cause other bacteriostatic effects, then those concentrations are also capable of producing adverse effects in mammalian tissue, which likewise relies on enzyme systems. This statement is based not only on common sense, but also on results of mutation studies which show that fluoride can cause gene mutations in mammalian and lower order tissues at fluoride concentrations estimated to be present in the mouth from fluoridated tooth paste.[22] Further, there were tumors of the oral cavity seen in the NTP cancer study

mentioned above, further strengthening concern over the toxicity of topically applied fluoride.

In any event, a person can choose whether to use fluoridated tooth paste or not (although finding non-fluoridated kinds is getting harder and harder), but one cannot avoid fluoride when it is put into the public water supplies.

So, in addition to our concern over the toxicity of fluoride, we note the uncontrolled - and apparently uncontrollable - exposures to fluoride that are occurring nationwide via drinking water, processed foods, fluoride pesticide residues and dental care products. A recent report in the lay media[23] that, according to the Centers for Disease Control, at least 22 percent of America's children now have dental fluorosis, is just one indication of this uncontrolled, excess exposure. The finding of nearly 12 percent incidence of dental fluorosis among children in un-fluoridated Kingston New York[17] is another. For governmental and other organizations to continue to push for *more* exposure in the face of current levels of over-exposure coupled with an increasing crescendo of adverse toxicity findings is irrational and irresponsible at best.

Thus, we took the stand that a policy which makes the public water supply a vehicle for disseminating this toxic and prophylactically useless (via ingestion, at any rate) substance is wrong.

We have also taken a direct step to protect the employees we represent from the risks of drinking fluoridated water. We applied EPA's risk control methodology, the Reference Dose, to the recent neurotoxicity data. The Reference Dose is the daily dose, expressed in milligrams of chemical per kilogram of body weight, that a person can receive over the long term with reasonable assurance of safety from adverse effects. Application of this methodology to the Varner et al.[4] data leads to a Reference Dose for fluoride of 0.000007 mg/kg-day. Persons who drink about one quart of fluoridated water from the public drinking water supply of the District of Columbia while at work receive about 0.01mg/kg-day from that source alone. This amount of fluoride is more than 100 times the Reference Dose. On the basis of these results the union filed a grievance, asking that EPA provide un-fluoridated drinking water to its employees.

The implication for the general public of these calculations is clear. Recent, peer-reviewed toxicity data, when applied to EPA's standard method for controlling risks from toxic chemicals, require an immediate halt to the use of the nation's drinking water reservoirs as disposal sites for the toxic waste of the phosphate fertilizer industry.[24]

This document was prepared on behalf of the National Treasury Employees Union Chapter 280 by Chapter Senior Vice-President J. William Hirzy, Ph.D. For more information please call Dr. Hirzy at 202-260-4683. His E-mail address is hirzy.john@epa.gov

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Fluoride: Protected Pollutant or Panacea?
Are the claimed benefits of ingesting fluoride over-rated
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Abstracts

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Vice President of EPA's Scientist Union Testifies Against Fluoridation

STATEMENT OF

Dr. J. WILLIAM HIRZY
NATIONAL TREASURY EMPLOYEES UNION CHAPTER 280

BEFORE THE SUBCOMMITTEE ON WILDLIFE, FISHERIES AND DRINKING WATER UNITED STATES SENATE

JUNE 29, 2000

(Click here for printer-friendly format)

Good morning Mr. Chairman and Members of the Subcommittee. I appreciate the opportunity to appear before this Subcommittee to present the views of the union, of which I am a Vice-President, on the subject of fluoridation of public water supplies.

Our union is comprised of and represents the professional employees at the headquarters location of the U.S. Environmental Protection Agency in Washington D.C. Our members include toxicologists, biologists, chemists, engineers, lawyers and others defined by law as "professionals." The work we do includes evaluation of toxicity, exposure and economic information for managements use in formulating public health and environmental protection policy.

I am not here as a representative of EPA, but rather as a representative of EPA headquarters professional employees, through their duly elected labor union. The union first got involved in this issue in 1985 as a matter of professional ethics. In 1997 we most recently voted to oppose fluoridation. Our opposition has strengthened since then.

Summary of Recommendations

1) We ask that you order an independent review of a cancer bioassay previously mandated by Congressional committee and subsequently performed by Battelle Memorial Institute with appropriate blinding and instructions that all reviewers independent determinations be reported to this Committee.

2) We ask that you order that the two waste products of the fertilizer industry that are now used in 90% of fluoridation programs, for which EPA states they are not able to identify any chronic studies, be used in any future toxicity studies, rather than a substitute chemical. Further, since federal agencies are actively advocating that each man woman and child drink, eat and bathe in these chemicals, silicofluorides should be placed at the head of the list for establishing a MCL that complies with the Safe Drinking Water Act. This means that the MCL be protective of the most sensitive of our population, including infants, with an appropriate margin of safety for ingestion over an entire lifetime.

3) We ask that you order an epidemiology study comparing children with dental fluorosis to those not displaying overdose during growth and development years for behavioral and other disorders.

4) We ask that you convene a joint Congressional Committee to give the only substance that is being mandated for ingestion throughout this country the full hearing that it deserves.

National Review of Fluoridation

The Subcommittees hearing today can only begin to get at the issues surrounding the policy of water fluoridation in the United States, a massive experiment that has been run on the American public, without informed consent, for over fifty years. The last Congressional hearings on this subject were held in 1977. Much knowledge has been gained in the intervening years. It is high time for a national review of this policy by a Joint Select Committee of Congress. New hearings should explore, at minimum, these points:

1) excessive and un-controlled fluoride exposures;

2) altered findings of a cancer bioassay;

3) the results and implications of recent brain effects research;

4) the "protected pollutant" status of fluoride within EPA;

5) the altered recommendations to EPA of a 1983 Surgeon Generals Panel on fluoride;

6) the results of a fifty-year experiment on fluoridation in two New York communities;

7) the findings of fact in three landmark lawsuits since 1978;

8) the findings and implications of recent research linking the predominant fluoridation chemical with elevated blood-lead levels in children and anti-social behavior; and

9) changing views among dental researchers on the efficacy of water fluoridation

Fluoride Exposures Are Excessive and Un-controlled

According to a study by the National Institute of Dental Research, 66 percent of Americas children in fluoridated communities show the visible sign of over-exposure and fluoride toxicity, dental fluorosis (1). That result is from a survey done in the mid-1980's and the figure today is undoubtedly much higher.

Centers for Disease Control and EPA claim that dental fluorosis is only a "cosmetic" effect. God did not create humans with fluorosed teeth. That effect occurs when children ingest more fluoride than their bodies can handle with the metabolic processes we were born with, and their teeth are damaged as a result. And not only their teeth. Childrens bones and other tissues, as well as their developing teeth are accumulating too much fluoride. We can see the effect on teeth. Few researchers, if any, are looking for the effects of excessive fluoride exposure on bone and other tissues in American children. What has been reported so far in this connection is disturbing. One example is epidemiological evidence (2a, 2b) showing elevated bone cancer in young men related to consumption of fluoridated drinking water.

Without trying to ascribe a cause and effect relationship beforehand, we do know that American children in large numbers are afflicted with hyperactivity-attention deficit disorder, that autism seems to be on the rise, that bone fractures in young athletes and military personnel are on the rise, that earlier onset of puberty in young women is occurring. There are biologically plausible mechanisms described in peer-reviewed research on fluoride that can link some of these effects to fluoride exposures (e.g. 3,4,5,6). Considering the economic and human costs of these conditions, we believe that Congress should order epidemiology studies that use dental fluorosis as an index of exposure to determine if there are links between such effects and fluoride over-exposure.

In the interim, while this epidemiology is conducted, we believe that a national moratorium on water fluoridation should be instituted. There will be a hue and cry from some quarters, predicting increased dental caries, but Europe has about the same rate of dental caries as the U.S. (7) and most European countries do not fluoridate (8). I am submitting letters from European and Asian authorities on this point. There are studies in the U.S. of localities that have interrupted fluoridation with no discernable increase in dental caries rates (e.g., 9). And people who want the freedom of choice to continue to ingest fluoride can do so by other means.

Cancer Bioassay Findings

In 1990, the results of the National Toxicology Program cancer bioassay on sodium fluoride were published (10), the initial findings of which would have ended fluoridation. But a special commission was hastily convened to review the findings, resulting in the salvation of fluoridation through systematic down-grading of the evidence of carcinogenicity. The final, published version of the NTP report says that there is, "equivocal evidence of carcinogenicity in male rats," changed from "clear evidence of carcinogenicity in male rats."

The change prompted Dr. William Marcus, who was then Senior Science Adviser and Toxicologist in the Office of Drinking Water, to blow the whistle about the issue (22), which led to his firing by EPA. Dr. Marcus sued EPA, won his case and was reinstated with back pay, benefits and compensatory damages. I am submitting material from Dr. Marcus to the Subcommittee dealing with the cancer and neurotoxicity risks posed by fluoridation.

We believe the Subcommittee should call for an independent review of the tumor slides from the bioassay, as was called for by Dr. Marcus (22), with the results to be presented in a hearing before a Select Committee of the Congress. The scientists who conducted the original study, the original reviewers of the study, and the "review commission" members should be called, and an explanation given for the changed findings.

Brain Effects Research

Since 1994 there have been six publications that link fluoride exposure to direct adverse effects on the brain. Two epidemiology studies from China indicate depression of I.Q. in children (11,12). Another paper (3) shows a link between prenatal exposure of animals to fluoride and subsequent birth of off-spring which are hyperactive throughout life. A 1998 paper shows brain and kidney damage in animals given the "optimal" dosage of fluoride, viz. one part per million (13). And another (14) shows decreased levels of a key substance in the brain that may explain the results in the other paper from that journal. Another publication (5) links fluoride dosing to adverse effects on the brains pineal gland and pre-mature onset of sexual maturity in animals. Earlier onset of menstruation of girls in fluoridated Newburg, New York has also been reported (6).

Given the national concern over incidence of attention deficit-hyperactivity disorder and autism in our children, we believe that the authors of these studies should be called before a Select Committee, along with those who have critiqued their studies, so the American public and the Congress can understand the implications of this work.

Fluoride as a Protected Pollutant

The classic example of EPAs protective treatment of this substance, recognized the world over and in the U S before the linguistic de-toxification campaign of the 1940's and 1950's as a major environmental pollutant, is the 1983 statement by EPAs then Deputy Assistant Administrator for Water, Rebecca Hanmer (15), that EPA views the use of hydrofluosilicic acid recovered from the waste stream of phosphate fertilizer manufacture as,

> "...an ideal solution to a long standing problem. By recovering by-product fluosilicic acid (sic) from fertilizer manufacturing, water and air pollution are minimized, and water authorities have a low-cost source of fluoride..."

In other words, the solution to pollution is dilution, as long as the pollutant is dumped straight into drinking water systems and not into rivers or the atmosphere. I am submitting a copy of her letter.

Other Federal entities are also protective of fluoride. Congressman Calvert of the House Science Committee has sent letters of inquiry to EPA and other Federal entities on the matter of fluoride, answers to which have not yet been received.

We believe that EPA and other Federal officials should be called to testify on the manner in which fluoride has been protected. The union will be happy to assist the Congress in identifying targets for an inquiry. For instance, hydrofluosilicic acid does not appear on the Toxic Release Inventory list of chemicals, and there is a remarkable discrepancy among the Maximum Contaminant Levels for fluoride, arsenic and lead, given the relative toxicities of these substances.

Surgeon Generals Panel on Fluoride

We believe that EPA staff and managers should be called to testify, along with members of the 1983 Surgeon Generals panel and officials of the Department of Human Services, to explain how the original recommendations of the Surgeon Generals panel (16) were altered to allow EPA to set otherwise unjustifiable drinking water standards for fluoride.

Kingston and Newburg, New York Results

In 1998, the results of a fifty-year fluoridation experiment involving Kingston, New York (un-fluoridated) and Newburg, New York (fluoridated) were published (17). In summary, there is no overall significant difference in rates of dental decay in children in the two cities, but children in the fluoridated city show significantly higher rates of dental fluorosis than children in the un-fluoridated city.

We believe that the authors of this study and representatives of the Centers For Disease Control and EPA should be called before a Select Committee to explain the increase in dental fluorosis among American children and the implications of that increase for skeletal and other effects as the children mature, including bone cancer, stress fractures and arthritis.

Findings of Fact by Judges

In three landmark cases adjudicated since 1978 in Pennsylvania, Illinois and Texas (18), judges with no interest except finding fact and administering justice heard prolonged testimony from proponents and opponents of fluoridation and made dispassionate findings of fact. I cite one such instance here.

In November, 1978, Judge John Flaherty, now Chief Justice of the Supreme Court of Pennsylvania, issued findings in the case, Aitkenhead v. Borough of West View, tried before him in the Allegheny Court of Common Pleas. Testimony in the case filled 2800 transcript pages and fully elucidated the benefits and risks of water fluoridation as understood in 1978. Judge Flaherty issued an injunction against fluoridation in the case, but the injunction was overturned on jurisdictional grounds. His findings of fact were not disturbed by appellate action. Judge Flaherty, in a July, 1979 letter to the Mayor of Aukland New Zealand wrote the following about the case:

"In my view, the evidence is quite convincing that the addition of sodium fluoride to the public water supply at one part per million is extremely deleterious to the human body, and, a review of the evidence will disclose that there was no convincing evidence to the contrary...

"Prior to hearing this case, I gave the matter of fluoridation little, if any, thought, but I received quite an education, and noted that the proponents of fluoridation do nothing more than try to impune (sic) the objectivity of those who oppose fluoridation."

In the Illinois decision, Judge Ronald Niemann concludes: "This record is barren of any credible and reputable scientific epidemiological studies and or analysis of statistical data which would support the Illinois Legislatures determination that fluoridation of the water supplies is both a safe and effective means of promoting public health."

Judge Anthony Farris in Texas found: "[That] the artificial fluoridation of public water supplies, such as contemplated by {Houston} City ordinance No. 80-2530 may cause or contribute to the cause of cancer, genetic damage, intolerant reactions, and chronic toxicity, including dental mottling, in man; that the said artificial fluoridation may aggravate malnutrition and existing illness in man; and that the value of said artificial fluoridation is in some doubt as to reduction of tooth decay in man."

The significance of Judge Flahertys statement and his and the other two judges findings of fact is this: proponents of fluoridation are fond of reciting endorsement statements by authorities, such as those by CDC and the American Dental Association, both of which have long-standing commitments that are hard if not impossible to recant, on the safety and efficacy of fluoridation. Now come three truly independent servants of justice, the judges in these three cases, and they find that fluoridation of water supplies is not justified.

Proponents of fluoridation are absolutely right about one thing: there is no real controversy about fluoridation when the facts are heard by an open mind.

I am submitting a copy of the excerpted letter from Judge Flaherty and another letter referenced in it that was sent to Judge Flaherty by Dr. Peter Sammartino, then Chancellor of Fairleigh Dickenson University. I am also submitting a reprint copy of an article in the Spring 1999 issue of the Florida State University Journal of Land Use and Environmental Law by Jack Graham and Dr. Pierre Morin, titled "Highlights in North American Litigation During the Twentieth Century on Artificial Fluoridation of Public Water. Mr. Graham was

chief litigator in the case before Judge Flaherty and in the other two cases (in Illinois and Texas).

We believe that Mr. Graham should be called before a Select Committee along with, if appropriate, the judges in these three cases who could relate their experience as trial judges in these cases.

Hydrofluosilicic Acid

There are no chronic toxicity data on the predominant chemical, hydrofluosilicic acid and its sodium salt, used to fluoridate American communities. Newly published studies (19) indicate a link between use of these chemicals and elevated level of lead in childrens blood and anti-social behavior. Material from the authors of these studies has been submitted by them independently.

We believe the authors of these papers and their critics should be called before a Select Committee to explain to you and the American people what these papers mean for continuation of the policy of fluoridation.

Changing Views on Efficacy and Risk

In recent years, two prominent dental researchers who were leaders of the pro-fluoridation movement announced reversals of their former positions because they concluded that water fluoridation is not an effective means of reducing dental caries and that it poses serious risks to human health. The late Dr. John Colquhoun was Principal Dental Officer of Aukland, New Zealand, and he published his reasons for changing sides in 1997 (20). In 1999, Dr Hardy Limeback, Head of Preventive Dentistry, University of Toronto, announced his change of views, then published a statement (21) dated April 2000. I am submitting a copy of Dr. Limebacks publications.

We believe that Dr. Limeback, along with fluoridation proponents who have not changed their minds, such as Drs. Ernest Newbrun and Herschel Horowitz, should be called before a Select Committee to testify on the reasons for their respective positions.

Thank you for your consideration, and I will be happy to take questions.

Read EPA Union's 1986 Amicus Brief against EPA Management for the agency's issuance of a new, elevated, Maximum Contaminant Level for fluoride

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LEGALITY OF WATER FLUORIDATION

American Association for Justice
formerly the Association of Trial Lawyers of America

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In This Issue. .

Fluoridegate and Fluoride Litigation: What Law Firms Need to Know About Fluoride Toxic Tort Actions

By Chris Nidel, Rockville, MD & Daniel G. Stockin, Ellijay, GA

Chris Nidel, Nidel Law PC, 1011 Pleasant Dr., Rockville, MD 20850; T: 202-730-5926; chris@nidellaw.com; www.nidellaw.com. Daniel G. Stockin, The Life Center Inc., PO Box 639, Ellijay, GA 30540; T: 706-669-0786; stockin2@yahoo.com; www.thelifecenter.com. The next edition of the STEP newsletter will provide an overview and analysis of recent science developments, potential defendants, causes of action, and the rapidly accelerating interest in legal actions by harmed parties.

You have probably heard the recent news in the media about fluoride risks: a growing "Fluoridegate" scandal; cities dropping their longstanding policy of water fluoridation; and concerns about fluoride harm to kidneys, bones, thyroid glands, and teeth. For decades, Americans have heard of a long-simmering controversy over the whole-body safety of ingested fluorides. Now government agencies and private sector groups are admitting concerns about the impact to the body from fluorides in numerous consumer products, including water, beverages, foods made with fluoridated water or containing fluoride fumigant residues, and oral care products.

Of particular interest is news that infants, diabetics, kidney patients, and seniors are "susceptible subpopulations" that are particularly vulnerable to harm from fluorides. The number of potential plaintiffs in these and other groups foreshadows decades of fluoride-related court cases and investigations. As a result, scientists, health care professionals, businesses, and influential leaders are voicing concerns about fluorides. The Gerber baby products company is now selling an unfluoridated water to be used for making milk formula so that parents and others caring for infants will not use fluoridated water when mixing formula for babies.

A signature condition of excessive fluoride intake is "dental fluorosis," a permanent and often disfiguring staining of teeth. A staggering number of Americans have the white, yellow, or brown staining or pitting of teeth caused by fluorides. Most never know what caused the staining. According to the National Center for Health Statistics, approximately 23 percent of people ages 6– 49 have fluorosis, as do 41 percent of adolescents agres 12–15 years old.

Public and private sector groups as well as individuals are potentially responsible for the financial and health impacts of fluorides provided to consumers without full disclosure of the risks. A partial list of defendants includes manufacturers of fluoridation chemicals, oral care product manufacturers, retailers, water utilities, medical and dental practitioners, and professional associations. Given the complexity of potential litigation, plaintiffs may choose to utilize market-share and other legal theories providing liability to a group of defendants for a single, indivisible injury.

Causes of action may include personal injury, failure to warn, negligent misrepresentation, medical or dental malpractice, and consumer fraud. Because African-Americans and other minority groups are disproportionately harmed by fluorides, there may be civil rights and environmental justice avenues for legal cases.

The curtain is lifting, exposing the degree of deception at the root of the Fluoridegate scandal and highlighting the liability of both municipal water providers and private companies.

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Fluoridation of Public Water Systems: Valid Exercise of State Police Power or Constitutional Violation?

*Douglas A. Balog, Esq.**

I. Introduction

The United States Supreme Court has never decided whether a state, in the proper exercise of its police power, can mandate prophylactic medication for a noncontagious disease when such disease is treatable by reasonable, less intrusive means. More specifically, the Supreme Court has never decided whether fluoridation of public water systems is a valid exercise of state police power or a constitutional violation. It is the scope of state police power and governmental involvement with the noncontagious disease of dental caries that will be explored in this Comment. Police power is the implied constitutional authority allowing states to make laws concerning the health, safety, welfare and morals of its citizens.[1] States exercise their police power when they require that students be medicated against contagious diseases, such as measles, mumps and rubella, by way of inoculations prior to attending public schools. This immunization requirement is virtually uncontested by the public because the risk of spreading communicable diseases to other students is widely recognized.[2]

However, states also medicate the public water systems[3] with fluoride in an attempt to retard tooth decay -- a non-contagious disease,[4] also known as dental caries.[5] This state action has been rigorously contested by members of the public because the addition of fluoride to the public water system by municipal providers[6] may have adverse consequences on the health of the general public which outweigh the benefits allegedly prodded in reducing tooth decay in children.[7]

Fears concerning the purity of public water sources have increased dramatically in recent years, due in part to reports of leaking landfills, corroding pipes and crumbling gasoline storage tanks tainting water supplies.[8] These

fears pertain not only to contaminants such as lead, nitrates, pesticides, radon and other organic chemicals that inadvertently find their way into public drinking water, but also to chlorine and fluorine, which are purposely added to public water systems.[9] A public survey conducted in the late 1980s indicated that "[n]early 70 percent of Americans are worried about the quality of their drinking water . . . [with] their concern center[ing] on how water looks, tastes or smells.[10]

In 1992, "Americans spent more than $700 million on inhome filters" and more than $2 billion on bottled water in efforts to avoid drinking contaminated water.[11] In spite of this, a common misconception is that fluoridated tap water provided by a public water system is completely safe to drink. This assumption is unwarranted because fluoridated tap water has the potential to cause adverse health consequences, including death.[12]

While the general public supports chlorination[13] to ensure that tap water is safer to drink, there is both adamant support and unrelenting opposition to the artificial fluoridation of drinking water.[14] Public debate on the issue of fluoridation began in the 1950s and continues to date, resulting in an abundance of lawsuits opposing fluoridation of public water systems.

The public support for fluoridation exists mainly because of the misconception that fluoride in drinking water and toothpaste benefits the development and overall health of the teeth of both children and adults. However, even proponents of fluoridation admit that fluoride does not provide any health benefits when ingested by an adult, while the potential exists for causing adverse health problems, such as crippling skeletal fluorosis.[15] Other adverse health problems linked to fluoride include a 690% increase in bone cancer in of young males,[16] a doubling of hip fractures for both older men and women,[17] and infertility in women.[18]

Some opponents of fluoridation are of the opinion that there is no correlation between the level of fluoride in water and dental caries.[19] As a matter of fact, the federal government has conceded that the purported benefit of fluoridation is limited, as it applies only to developing enamel in the teeth of children up to the age of nine.[20] The problem with health laws specifically targeted at children is that children constitute only a minority of the general public because "[t]he most recent year in which a majority of families included at least one child among their members was 1982.... [C]hildren are defined as the householder's own children who are under the age of 18, have never been married, and are still living at home.[21] It logically follows that persons under the age of eighteen comprise less than 50% of the U.S. population.

Therefore, state laws authorizing municipal fluoridation of water do not benefit the majority of the public, and thus do not promote the health, safety, and welfare of Americans, the majority of whom are adults.

Fluoridation of public water systems has been attacked in the courts on various constitutional grounds, but has always been upheld as a valid exercise of state police power.[22] This result stems from the application of the "rational basis" test of judicial review to fluoridation laws by all of the appellate courts. The rational basis test is the least demanding form of judicial review, providing broad deference to the legislature. It merely requires that the goals sought be legitimate, and that the means chosen by the legislature be rationally related to the achievement of those goals, so as not to violate the Due Process Clause.[23] To pass constitutional muster under the rational basis test, legislation cannot be arbitrary and must have a reasonable purpose which "bears a rational relationship to a [permissible] state objective."[24]

There is a rebuttable presumption that all legislation is constitutional, and "those challenging a statute must prove unconstitutionality beyond a reasonable doubt."[25] Because of this presumption of validity, courts will generally apply the easily-satisfied rational basis test to a challenged law, unless given a reason to justify a higher standard of review. A constitutional challenge to legislation is one such reason for performing a more demanding judicial review, because the rational basis test does not apply if the statute "interferers with the free exercise of some fundamental personal right or liberty."[26]

The rational basis test is properly applied to legislation dealing with public health protection, such as the prevention or spread of contagious or communicable diseases.[27] However, fluoridation of public water systems cannot logically rise to the level of a public health protection measure, as it is merely an attempt to prevent the disease of tooth decay, which is neither contagious nor communicable. Thus, adding prophylactic medication (fluoride) to drinking water exceeds the scope of state police power, and courts should apply the highest standard of judicial review, called "strict scrutiny" to the legislation authorizing fluoridation.

Strict scrutiny is the highest standard of judicial review that courts use to determine if a law deprived, infringed, or interfered with a fundamental constitutional right or liberty.[28] To pass this test, the legislation must be narrowly tailored and necessary to achieve a legitimate, compelling state interest.[29] It is first necessary to understand what constitutes a "fundamental constitutional right" in order to know when a strict scrutiny review is required. The United States Supreme Court has recently held that "[t]he forcible injection of medication into a nonconsenting person's body represents a substantial interference with that person's liberty," [30] which involves a fundamental constitutional right.

This Comment shows why, under strict scrutiny review, state laws authorizing fluoridation of public water systems should be struck down as unconstitutional since they impinge a fundamental constitutional right, the means of accomplishing fluoridation is not narrowly tailored, and there is no compelling government interest involved.[31] To fully understand the detrimental effects of fluoride on the human body, one must be familiar with its chemical properties and how it has become regulated by the federal government. To accomplish this goal, Part I of this Comment examines the adverse health effects caused by fluoride ingestion and how fluoride has come to be labeled as a contaminant, poison, and most importantly, as a drug for treating a noncontagious disease.

Part II traces the regulatory attempts aimed at ensuring the provision of safe drinking water by municipalities, and explains the role of the Environmental Protection Agency in establishing contaminant levels for fluoride under the Safe Drinking Water Act. Part III examines the legal challenges to fluoridation of public water systems, noting that courts have upheld that it is a valid exercise of state police power. Part IV addresses the exercise of state police power as a basis for enacting fluoridation laws.

Further, Part IV analyzes the constitutional protection afforded to an individual in the context of fluoridation laws. Finally, Part V concludes that courts have used the wrong standard in their judicial review of statutes authorizing fluoridation, and that fluoridation laws will not pass constitutional muster under a strict scrutiny standard of review. This is because fluoridation statutes violate the constitutionally protected liberty interest to be free from unwanted medical treatment recognized by the U.S. Supreme Court in the 1990 cases, Cruzan v. Director, Missouri Dept of Health[32] and Washington v. Harper.[33] These two decisions have not yet been relied upon by a lower court in the constitutional analysis of fluoridation laws.

Fluoridating public water in an attempt to target children whose permanent teeth are still developing is like using a shotgun to shoot an apple off someone's head; sure, you hit the apple, but the side effects are undesirable.

I. Health Effects on the Human Body From Fluoride Ingestion

To understand why most, if not all, states have laws providing for the addition of fluoride to the public water systems and why the public opinion is split as to the benefits and harms of fluoridation, some background information on fluoride is useful. Fluoride is a binary compound,[34] consisting of the element fluorine[35] combined with another element, such as copper, magnesium, iron, sodium, or zinc.[36] Fluorine has been estimated to be the thirteenth most abundant element in the earth's crust and is usually found only in combination with other elements, producing compounds called fluorides.[37]

It is crucial to note that fluorine is not an essential nutrient needed by the human body.[38] Virtually all foods contain trace amounts of fluoride, but the quantity is negligible and not considered for purposes of regulating maximum fluoride levels in drinking water.[39] The use of fluoride for medicinal purposes originated because it was discovered in communities where the water supply naturally contained a small percentage of dissolved fluorides that the tendency toward tooth decay in children was notably reduced.[40] Accordingly, the United States Public Health Service endorsed the artificial fluoridation of public drinking water in 1950.[41] Because the government is now actively involved in preventing tooth decay (a periodontal disease), it is actually practicing medicine, which is defined as "the science and art of . . . preventing disease," and fluoride may be considered a 'medicine' which is defined as "any drug or other substance used in treating disease ...."[42]

The decision to add fluoride to public water systems sparked controversy because "the fluoride encountered in 'natural' drinking water is calcium fluoride,"[43] but artificial fluoridation is accomplished by using either sodium fluoride,[44] sodium fluorosilicate,[45] or hydrofluorosilicic acid.[46] Sodium fluoride was the first compound used in public water systems to artificially fluoridate public water, which caused an uproar because sodium fluoride is a known poison used commercially as an insecticide, rodenticide, wood preservative and fungicide, in ceramics production, and in light metal production.[47] The federal government recognizes the toxicity of sodium fluoride because it is regulated as an active ingredient in pesticides under the Federal Insecticide, Fungicide, and Rodenticide

Act (FIFRA).[48] Pesticides with highly toxic quantities of sodium fluoride in them must have the skull and crossbones symbol as well as the word poison prominently displayed on the container.[49]

The government promoters seeking to add fluoride to the public water systems back in the 1950s held a conference and tried to diffuse this issue by instructing those in attendance not to use the word "artificial" in conjunction with fluoridation and not to tell the public that sodium fluoride is being used because "that is rat poison."[50] Instead, the public should only be told that "fluorides" are added to the water.[51]

This is hardly comforting because in a table of water-borne contaminants, the Environmental Protection Agency (EPA) lists fluoride between cyanide and mercury, two toxic substances that the public would certainly never tolerate to be purposely added to their water supply.[52]

The scientific community is sharply divided as to the detrimental effects of fluoride on the human body.[53] The EPA solicited and received over 400 written public comments and held two full days of public hearings in Washington, D.C. pertaining to the issue of whether fluoride in public drinking water posed adverse health effects.[54] Many professional health organizations and state officials believed that fluoride in drinking water causes no adverse health effects, but other commentators believed that it can cause serious adverse health effects, such as crippling skeletal fluorosis,[55] mutagenicity,[56] and oncogenicity.[57] Dr. John Yiamouyiannis, an expert biochemist witness who has testified in several lawsuits challenging fluoridation statutes, authored a book entitled Fluoride - The Aging Factor.[58] In this book, Dr. Yiamouyiannis describes, among other adverse health effects, how fluoride damages enzymes and interferes with collagen formation in the human body, resulting in premature aging.[59] This book, along with eighty-eight other technical reports and studies, were considered by the EPA in its determination of the Recommended Maximum Contaminant Level (RMCL) for fluoride to be published in the Code of Federal Regulations (CFR).[60] The EPA concluded that there was an inadequate basis to say that fluoride is oncogenic, mutagenic, or results in allergic or idiosyncratic sensitivity.[61] However, the EPA acknowledged that the conclusions of the studies conflicted and that there are ongoing chronic rat and mouse bioassays designed to measure the oncogenic[62] potential of fluoride, which they will reconsider when the results become available.[63]

The EPA did acknowledge that dental fluorosis, a condition manifested by staining and/or pitting of the teeth, can result from ingesting fluoride, but labeled it as a cosmetic effect rather than an adverse health effect within the meaning of the Safe Drinking Water Act.[64] The EPA also concluded that crippling skeletal fluorosis has been thoroughly documented to be associated with the consumption of fluoridated drinking water in the U.S., and accordingly set the RMCL to protect against this adverse health effect.[65] In response to public comments opposing fluoridation, the EPA emphasized that the Safe Drinking Water Act "prohibits [the] EPA from requiring the addition of any substance for preventative health care purposes unrelated to [the] contamination of drinking water," and that just because it issued final regulations does not mean that it endorses the fluoridation of public water systems.[66] The federal government also regulates the ingestion of fluoride by humans in the Food, Drug, and Cosmetic Act, chapter 1, subchapter D, part 355 Anticaries Drug Products for Over-the-Counter Human Use,[67] and also in chapter 1, subchapter B. part 165 Beverages.[68] Fluoride used in a toothpaste, dentifrice, mouthwash, gel, or rinse is considered to be an anticaries drug, which is "[a] drug that aids in the prevention and prophylactic treatment of dental cavities (decay, caries)."[69] Three sources of fluoride are used for topical application in the mouth: sodium fluoride, [70] sodium monofluorophosphate,[71] and stannous fluoride.[72] While the maximum permissible fluoride concentration in water is 4mg/ L, it is much higher in topical applications, depending on its form. For example, dentifrices[73] contain a theoretical total fluorine concentration of 850 to 1150 parts per million (ppm)[74] in a paste dosage form.[75] This concentration is aimed at obtaining at least 650 ppm[76] of fluoride ions,[77] whereas treatment rinses[78] target a fluoride ion concentration of 0.01 0.05 percent in an aqueous solution.[79]

The government has recognized the adverse health effects that may result from swallowing either fluoridated toothpaste or mouth rinse, and requires that warning labels be affixed to the anticaries drug products. [80] All fluoride dentifrices (tooth pastes and tooth powders) must be labeled "Warning: Keep out of the reach of children under 6 years of age. [81] The labels for rinse and gel products emphasize the importance of spitting out the solution and not swallowing it.[82] This is because fluoride is very toxic, and at least one child has been killed from swallowing a fluoride jell applied by a dental hygienist.[83] In summary, fluoride is neither a vitamin nor a mineral necessary for human health.[84] Rather, fluorine is a highly reactive element[85] used as a prophylactic drug to help prevent tooth decay in developing permanent teeth.[86] Fluoride provides no benefits to adults, and ingestion of it will only result in the health problems previously mentioned.[87]

II. Legislative History of Safe Drinking Water

One of the first published cases attempting to ensure the safety of drinking water was Commonwealth v. Towanda Water-Works.[88] In Towanda, the Pennsylvania Attorney General alleged that the public water in the borough of Towanda was impure, unwholesome, polluted, unfit for use by the public, and dangerous to their lives and health.[89] The evidence as to the purity of the water was conflicting, but the jury found that the water was wholesome, [90] even though it was not pure.[91] The court took judicial notice that the only possible way to obtain pure water was by distillation.[92] Thus, the court held that the statute requiring "pure" water to be furnished for public consumption was to be construed to mean wholesome water, not pure in the abstract or chemical sense.[93] This case laid the foundation for the first drinking water standard set by the U.S. Public Health Service.

Promulgated in 1914, this standard was designed to protect the public from acute bacterial diseases,[94] and eventually led to the enactment of federal legislation in 1974 known as the Safe Drinking Water Act (SDWA).[95] The Safe Drinking Water Act requires the EPA Administrator to identify waterborne contaminants and publish maximum contaminant levels (enforceable standards) and recommended maximum contaminant levels (nonenforceable health goals) for municipal providers.[96] The SDWA was amended in 1986, changing the Recommended Maximum Contaminant Level (RMCL) terminology to Maximum Contaminant Level Goals (MCLG).[97] The EPA Administrator must promulgate national primary drinking water regulations for contaminants which may have an adverse effect on the health of persons and which are "known or anticipated to occur in public water systems."[98] The EPA issued national primary drinking water regulations for fluoride because it concluded that crippling skeletal fluorosis[99] is an adverse health effect, and has been thoroughly documented to be associated with consumption of fluoridated drinking water.[100]

The EPA promulgated a Maximum Contaminant Level[101] (MCL) for fluoride in the 1985 National Interim Primary Drinking Water Regulations, pursuant to section 1412 of the Safe Drinking Water Act.[102] This interim MCL varied from 1.4 milligrams per liter (mg/L) to 2.4 mg/L, depending upon the annual average ambient air temperatures.[103] This interim amount was twice the optimum fluoride concentration, and was determined to strike an appropriate balance between the occurrence of dental fluorosis[104] and the prevention of dental caries.[105]

In response to this proposed MCL, the EPA received over 400 written public comments and held two full days of public hearings as to whether fluoride in drinking water posed adverse health consequences.[106] Based upon all the information it received, the EPA set the Recommended Maximum Contaminant Level (RMCL) of fluoride at 4 mg/L,[107] and subsequently set the Maximum Contaminant Level (MCL) at 4 mg/L also.[108] The EPA promulgated an RMCL for fluoride because it agreed with Surgeon Generals Shapiro and Koop that adverse health effects stemming from the ingestion of fluoride include "death, gastrointestinal hemorrhage or irritation, arthralgias, and crippling fluorosis." [109] RMCLs are non-enforceable health goals which are set at levels for which there are "no known or anticipated adverse health effects" and which leave a margin of safety to protect against crippling skeletal fluorosis.[110] The difference between an MCL and an RMCL is that the RMCL is supposed "to be based only on health and safety considerations while an MCL takes feasibility and cost into consideration.[111]

The Administrator must also promulgate the national secondary drinking water regulations, which are designed to protect the public health by controlling contaminants that "may adversely affect the odor or appearance of [drinking] water.[112] These secondary regulations specify the Secondary Maximum Contaminant Levels (SMCLs) which limit those contaminants that "may adversely affect the aesthetic quality of drinking water such as taste, odor, color and appearance ...."[113] Fluoride was not included in the original list of contaminants, but in 1985 the EPA Administrator proposed a SMCL of 2.0 mg/L for fluoride.[114] This limit was eventually approved and incorporated into the Code of Federal Regulations (CFR).[115]

If the EPA's SMCL for fluoride is exceeded, but the MCL is not, the CFRs require that the municipal water provider send the following notice to all paying users, as well as to the state public health officer:

> Public Notice
>
> Dear User:
>
> The U.S. Environmental Protection Agency requires that we send you this notice on the level of fluoride in your drinking water. The drinking water in your community has a fluoride concentration of [fill in amount] milligrams per liter (mg/L).

Federal regulations require that fluoride, which occurs naturally in your water supply, not exceed a concentration of 4.0 mg/L in drinking water. This is an enforceable standard called a Maximum Contaminant Level (MCL), and it has been established to protect the public health. Exposure to drinking water levels above 4.0 mg/L for many years may result in some cases of crippling skeletal fluorosis, which is a serious bone disorder.

Federal law also requires that we notify you when monitoring indicates that the fluoride in your drinking water exceeds 2.0 mg/L. This is intended to alert families about dental problems that might affect children under nine years of age. The fluoride concentration of your water exceeds this federal guideline.

Fluoride in children's drinking water at levels of approximately 1 mg/L reduces the number of dental cavities. However, some children exposed to levels of fluoride greater than about 2.0 mg/1 may develop dental fluorosis. Dental fluorosis, in its moderate and severe forms, is a brown staining and/or pitting of the permanent teeth.

Because dental fluorosis occurs only when developing teeth (before they erupt from the gums) are exposed to elevated fluoride levels, households without children are not expected to be affected by this level of fluoride. Families with children under the age of nine are encouraged to seek other sources of drinking water for their children to avoid the possibility of staining and pitting.

Your water supplier can lower the concentration of fluoride in your water so that you will still receive the benefits of cavity prevention while the possibility of stained and pitted teeth is minimized. Removal of fluoride may increase your water costs. Treatment systems are also commercially available for home use. Information on such systems is available at the address given below. Low fluoride bottled drinking water that would meet all standards is also commercially available.

For further information, contact [Public Water System employee's name, address, and phone number] at your water system[116]

The EPA has published a disclaimer in the national primary drinking water regulation for fluoride stating that: (1) they do not endorse fluoridation, even though they established an RMCL, (2) public water systems are not required to meet the RMCL, (3) states are not required to adopt the RMCL, and (4) fluoridation is a matter for state and local authorities.[117]

The RMCL for fluoride promulgated by the EPA is based upon the presumption that a child will drink 1.4 liters of tap water a day and that an adult will drink 2.0 liters a day.1[118] However, the EPA does not admit that suppliers of water[119] frequently violate the RMCL as well as the MCL for fluoride. In fiscal year 1986, 740 Public Water Systems (PWS) were in violation of fluoride levels.[120] Moreover, fluoride was the second most frequently violated contaminant by Significant Non-Compliers (SNCs).[121]

III. Legal Challenges Against Public Water System Fluoridation

The United States Supreme Court has never decided whether a state can compel individuals to ingest fluoride through public drinking water, when fluoride can be administered by other reasonable, less intrusive means. Accordingly, some state courts have limited their constitutional analysis of fluoridation statutes, based on reasoning that since the United States Supreme Court has declined to hear any fluoridation cases, there must not be any substantial constitutional issues.[122] The trial court in Paduano v. City of New York[123] held that "[w]hile denials of certiorari do not constitute decisions on the merits, it is clear that the Supreme Court has repeatedly held that no substantial Federal questions are presented by objections to fluoridation."[124] The court assumed, however, that the United States Supreme Court is, in effect, affirming the lower court when it dismisses the appeal.[125] However, this is

not true, and courts should be compelled to analyze all constitutional issues in light of the most recent Supreme Court decisions.[126]

A. Challenges Based on States' Lack of Authority to Fluoridate Public Water Systems

The first reported challenge to the addition of fluoride to a public water supply by a state was in De Aryan v. Butler.[127] In De Aryan, a taxpayer brought suit seeking to enjoin the addition of fluoride to water furnished to San Diego.[128] The basis for the challenge was that the city council exceeded its authority in enacting the fluoridation resolution.[129] The De Aryan court found that the State Board of Public Health was part of the Department of Public Health and thus had the "power to formulate policies affecting public health, and to adopt, promulgate, repeal, and amend rules and regulations consistent with law for the protection of the public health.[130] The State Legislature had "delegated to the State Board of Public Health the duty and powers necessary to control and regulate the purity, potability, and wholesomeness of public waters in this state."[131]

The De Aryan court further found that the entire police power of the state is vested in the legislature, and is only limited by the State Constitution and other applicable statutes.[132] Furthermore, even though the enforcement of police power may seem harsh at times, it is an indispensable part of state sovereignty and may not be legally limited unless its use is unreasonable and arbitrarily invoked.[133] Accordingly, applying the rational basis test, the De Aryan court concluded that the addition of fluoride to the public water system was a valid exercise of police power as long as it was not unreasonable or an abuse of discretion.[134] Although the petitioner taxpayer proffered exhibits, reports, and expert witnesses at the trial, there was no allegation in the petition that the City Council abused its discretion or made an unreasonable decision to endorse fluoridation.[135] Therefore, the District Court of Appeal could not consider the evidence.[136]

A somewhat different challenge against the authority of a city to fluoridate the public water system was made in Wilson v. City of Mountlake Terrace.[137] In Wilson, the appellants, representing a class of 300 persons in an unincorporated area adjacent to Mountlake Terrace, objected to the introduction of fluoride into their water by the Alderwood Water District.[138] Alderwood was under contract to provide fluoridated water to Mountlake Terrace, and also provided water to the appellants through a common distribution line.[139] The appellants argued that a city cannot exercise its police power outside its boundaries, and, therefore, Mountlake Terrace was without authority to impose fluoridation on persons living outside the city.[140] The appellants, however, did not claim to be harmed by the fluoridation, and stipulated that fluoridation did not render the water unfit for human consumption according to health department standards.[141] The Supreme Court of Washington agreed with the trial court's finding that "the fluoridation of appellant's water is the incidental, although inevitable, result of the city's exercise of its police power ...."[142] Applying the rational basis test, the court affirmed the judgment for Mountlake Terrace allowing continued fluoridation because of the finding that appellants were not harmed.[143]

B. Scope of Police Power Exceeded by the States

In 1954, one year after the De Aryan challenge, residents of Tulsa sought to enjoin enforcement of an ordinance authorizing fluoridation of the city water supply, alleging that it constituted an unwarranted exercise of state police power.[144] These plaintiffs, in Dowell v. City of Tulsa, argued that Oklahoma had "never . . . attempt[ed] to regulate or control any disease except those that are 'contagious, infectious, or dangerous'".[145] The Dowell court, however, did not believe that the Oklahoma Legislature "intended to restrict its enactment of measures designed to promote the public health and welfare to those designed to prevent the spread of [such] diseases."[146] The court found support for its position by noting the many statutes regulating food, lodging, and other subjects "that have no direct connection with or relation to [such] diseases."[147] Thus, using the rational basis test, the court upheld the fluoridation statute as a valid exercise of state police power "relating to eugenics and the maintenance of a healthy, normal, and socially sound populace."[148]

That same year, the appellant in McGurren v. City of Fargo[149] challenged the police power of the state, alleging that an implied contract exists between the public water system supplier and the consumer.[150] The rationale was that the city furnished and sold water in a proprietary capacity, and since the appellant had performed his contractual requirements by making the necessary service arrangements and paying the city for the water, then the city was mutually obliged to furnish water that was as reasonably pure and wholesome as possible.[151] The appellant further argued that the city breached the implied contract by adding fluoride to the water because the water was no longer free from any contamination, rendering the water "unfit for domestic use and unsafe and dangerous to individuals."[152] The appellant then argued that this breach endangered the health of the community, thereby exceeding the police power, which is intended to protect the public's health.[153] Accordingly, the McGurren court felt

that an injunction preventing the addition of fluoride to the public water supply was proper and overruled the appellee's demurrer,[154] remanding the case to the district court to allow the City of Fargo answer the complaint.[155]

C. Constitutional Challenges

The constitutional challenges brought against the fluoridation of public water systems have covered the entire spectrum of colorable arguments. These arguments include First Amendment freedom of religion, Fourteenth Amendment denial of liberty, abridgement of privileges and immunities, and denial of equal protection, and finally, Ninth Amendment invasion of personal privacy.

1. Fourteenth Amendment Challenges

The Fourteenth Amendment to the United States Constitution provides, in pertinent part, that

> no State shall make or enforce any law which shall abridge the privileges or immunities of citizens of the United States; nor shall any State deprive any person of life, liberty, or property, without due process of law; nor deny to any person within its jurisdiction the equal protection of the laws.[156]

Challenges to public water system fluoridation statutes typically include allegations of: (1) deprivation of personal liberty; (2) abridgement of one's privileges and immunities; or (3) denial of equal protection of the law.

(a) Deprivation of Personal Liberty

Two years after a federal court in California rendered the decision in De Aryan,[157] the seminal case of Kraus v. City of Cleveland[158] was decided in Ohio Supreme Court. The plaintiff in Kraus attacked legislation authorizing fluoridation of the public water supply as an infringement of fundamental liberties.[159] Urging that every individual has a personal liberty right "to protect his health as he deems best to insure a long and happy life," the plaintiff argued that fluoridation of the city water supply deprives him of this right in violation of the Fourteenth Amendment.[160] The Kraus court recognized this personal liberty right, but noted that it is not absolute because it is subject to limits stemming from the police powers of the state.[161] The plaintiff argued, however, that individual rights are subordinate to state police powers only when there is an overriding public purpose, such as a present danger, necessity, or emergency, and suggested that no such purpose existed to justify fluoridation of the water supply.[162] The Kraus court then examined the scope of the police power under Ohio law and found that if it satisfied the following four prongs, it was a valid exercise of police power: (1) it must be reasonable and necessary to achieve the legislature's objectives; (2) it must not violate the U.S. Constitution; (3) it must not be in direct conflict with any provision of the State Constitution; and (4) it must not be used in an arbitrary and oppressive manner.[163]
The public health measure at issue in Kraus pertained to the prevention of dental caries by increasing resistance in children to tooth decay, which the court felt was a "serious and widespread disease."[164] Accordingly, the Kraus court found that "any reasonable measure designed to decrease or retard the incidence of dental caries is in the interest and welfare of the public," and that this exercise of police power was not arbitrary or oppressive.[165] Thus, although the Kraus court did not disagree that fluoridation is an invasion of a person's constitutional right to protect his health as he deems best, it applied the rational basis test and held that this right must yield to police power exercised to prevent caries in Children.[166]

(b) Abridgment of Privileges and Immunities

In Teeter v. Municipal City of La Porte,[167] an action was brought to enjoin fluoridation of the municipal water supply, based on allegations that the local ordinance abridged the Privileges and Immunities Clause[168] of the Fourteenth Amendment.[169] The appellants argued that fluoridation of the public water system was "an enforced method of taking drugs and giving [the] same to their children . . . [and that] each individual should have the right to determine what to drink and eat without dictation from others ...." [170] The court determined, however, that it was "not necessary to decide the constitutional issues at this stage of the proceeding" and merely indicated that it was not "in a position to hold conclusively as a matter of law [that] fluoridation will not have cumulative toxic effects."[171] Thus, the Teeter decision did not advance the legal analysis of constitutional issues raised by forced fluoridation.
Almost ten years after Teeter, plaintiffs in Paduano v. City of New York[172] sought to enjoin the proposed fluoridation of the public water system arguing that, among other things, it violated the Privileges and Immunities Clause of the Fourteenth Amendment to the U.S. Constitution as well as Article I, Section 1 of the New York State Constitution.[173] The Paduano court held that fluoridation of the water supply "may be the only practical method of insuring the administration of this drug to the very young," and is probably the most efficient and cheapest method of doing so.[174] The court also held that "[i]t is not shocking to realize that the State, acting in the interest of children, too young to be sui juris,[175] may intervene in the parental area."[176] The plaintiffs' motion for an injunction was

denied because the Paduano court felt bound by the principles of stare decisis[177] to follow other cases, where the Supreme Court has denied certiorari to all fluoridation challenges.[178]

(c) Denial of Equal Protection of the Laws

It is undisputed even by the proponents of fluoridation that any benefit provided by fluoridation affects only tooth enamel that is still developing in children. Therefore, challenges have been made alleging that fluoridation of public water systems violates the Equal Protection Clause of the Fourteenth Amendment[179] because it affects only a limited class, namely, children.[180] These challenges have been dismissed because the United States Supreme Court has held that police power may be applied to a reasonable classification, and that just because the class is not all-embracing does not mean the legislation violates the Equal Protection Clause.[181] Several courts have held that fluoridation does not affect only a limited class because "[c]hildren of today are adult citizens of tomorrow"[182] and "it is apparent that children become adults."[183] Thus, the characterization of fluoridation laws as "class legislation" has passed the rational basis test applied by the courts.

2. First Amendment Right to Freedom of Religion

Another constitutional challenge asserted against fluoridation is based upon religious beliefs held by many citizens which forbid them to take medication for the prevention or treatment of any disease. The First Amendment to the United States Constitution provides, in pertinent part, that "Congress shall make no law respecting an establishment of religion, or prohibiting the free exercise thereof."[184] Thus, many cases have been brought, based on the theory that fluoridation violates the First Amendment right to freedom of religion.[185]

A First Amendment challenge was raised in Kraus v. City of Cleveland,[186] where the plaintiff contended that fluoridation of the public water system "compels people to take a form of medication contrary to their religious beliefs" in violation of the First Amendment.[187] The Kraus court noted that the United States Supreme Court has held that "freedom of religion has a dual aspect, freedom to believe, and freedom to act exercising such beliefs. The first is an absolute right, [but] the second is not," and therefore may be regulated in order to protect society.[188] Accordingly, the Kraus court held that the constitutional guaranty to freedom of religion must yield to regulations imposed "in the interests of the public welfare."[189]

3. Ninth Amendment Implied Right of Personal Privacy

In addition to the explicit constitutional rights protected by the First and Fourteenth Amendments, the Ninth Amendment provides that "[t]he enumeration in the Constitution, of certain rights, shall not be construed to deny or disparage others retained by the people."[190] This amendment was enacted so as to ensure that the government cannot violate fundamental rights of the public merely because those rights were not explicitly protected by the Constitution. Under the auspices of the Ninth Amendment, the City of Brainerd, Minnesota alleged that people have a prerogative to refuse fluoridation which is derived from the implicit constitutional right of privacy.[191] While there is no explicit mention of the right of privacy in the Constitution, the United States Supreme Court has held that "the right of privacy protects an individual's right 'to be free from unwarranted governmental intrusion into matters so fundamentally affecting a person,'" such as the decision whether or not to terminate pregnancy.[192]

The Brainerd court recognized that while the United States Supreme Court has never ruled on the issue, "the right of personal privacy could also extend to protect an individual's decision regarding what he will or will not ingest into his body."[193] The Supreme Court, however, has held that "the constitution does not protect an individual 'against all intrusions' but only 'against intrusions which are not justified in the circumstances, or which are made in an improper manner'."[194] The Brainerd court found that while forced fluoridation does infringe upon an individual's freedom, such infringement cannot be given substantial weight if there are no significant adverse consequences to the individual.[195] If any weight were given to this infringement, the court felt that people could interfere with governmental enactment of similar public health measures, such as chlorinating the water.[196] Accordingly, the Brainerd court held "that fluoridation is a justified intrusion into an individual's bodily integrity."[197]

The most recent reported challenge to fluoridation is the 1994 case Safe Water Association, Inc. v. City of Fond Du Lac.[198] In Safe Water, the appellant asserted, inter alia, that the city's adoption of an fluoridation ordinance violated the constitutional right to privacy.[199] Plaintiffs had to first overcome the hurdle presented by Froncek v. City of Milwaukee, [200] a 1955 Wisconsin Supreme Court decision upholding fluoridation. In order to dispose of its adverse impact, Safe Water pointed out that the precedential weight of this decision was sufficiently limited by the more recent decisions of the United States Supreme Court in Griswold v. Connecticut[201] and Roe v. Wade,[202] which drastically enlarged the scope of the right to privacy.[203] The Safe Water Court noted that the U.S. Supreme Court recognized the implicit guarantee of "zones of privacy" in Carey v. Population Services, Int'l.,[204] but that this right

of privacy is narrow and is subject to some limitations.[205] The court then granted summary judgment against Safe Water Association, reasoning that it failed to see any relationship between cases concerning the freedom to make reproductive choices and the issue of the right to be free from fluoridated water.[206]

IV. Analysis

A proper analysis of the statutes authorizing the fluoridation of public water systems must begin with a review of the limitations on state police power. Laws relating to "minimum wages for women and minors, maximum hours for women and minors, . . . control of venereal disease, blood tests for marriage licenses, sterilization, pasteurization of milk, chlorination of water, and vaccination have all been held valid as based on police power exercised in regard to public health."[207] However, the United States Supreme Court has never decided whether fluoridation of public water involves constitutional issues, and thus the lower courts must make their own determination if fluoridation is a valid exercise of state police power or a constitutional violation.[208]

A. Lack of Authority Challenges

The first category of challenges to fluoridation of public water systems involves the lack of authority for states to require fluoridation of public water systems.[209] The first reported challenge to fluoridation was De Aryan v. Butler,[210] where the issue was whether the city council of San Diego and the State Board of Health had the authority to require that fluorides be added to the public water supply.[211] The District Court of Appeal held that the State Board of Health was delegated the police power "necessary to control and regulate the purity, potability and wholesomeness of public waters in the state."[212] Because there was no allegation that the city council of San Diego abused its discretion or acted unreasonably in enacting the fluoridation statute, the De Aryan court correctly concluded that the statute was a valid exercise of police power.[213]

In the case Wilson v. City of Mountlake Terrace,[214] plaintiffs challenged the authority of the city to fluoridate their water, arguing that they lived outside the city limits and, therefore, are not citizens, residents, nor taxpayers of the city.[215] The Supreme Court of Washington correctly concluded that the valid exercise of police power may not be challenged by someone experiencing an incidental effect, if such individual does not allege that any harm resulted therefrom.[216] The challenges to fluoridation in these two cases did not involve constitutional issues, and, therefore, the rational basis test was properly employed as the basis for judicial review.

B. Scope of Police Power Exceeded Challenges

The second category of legal challenges to fluoridation of public water systems involves allegations that the state exceeded the scope of their police powers.[217] The appellant in Dowell v. City of Tulsa[218] contended that police power was limited to the regulation of contagious, infectious, or dangerous diseases, but the court felt this distinction was immaterial, ridiculous, and of no consequence to the promotion of public health.[219] In the case McGurren v. City of Fargo, [220] the appellant convinced the court that he had a breach of contract issue, which was then remanded to the lower court for resolution.[221] The Dowell court properly used the rational basis test to find that the state did not exceed the scope of its police power, because the appellant failed to sufficiently identify a fundamental constitutional right which was infringed. The McGurren court never reached the constitutional issues raised, and thus did not further the constitutional analysis of fluoridation laws.

C. Constitutional Challenges

The third category of legal challenges to fluoridation of public water systems involves allegations that the state deprived an individual of their personal liberty, abridged their privileges and immunities, or denied them equal protection of the law in contravention of the Fourteenth Amendment to the United States Constitution.[222]

1. Deprivation of Personal Liberty Challenges

The problem in making a challenge that fluoridation of public water systems deprives a person of personal liberty is that this constitutional right is not absolute; the Constitution provides that no state may deprive a person of liberty without due process of law.[223] "Liberty" may be subdivided "into three headings involving governmental restraints on (1) physical freedom, (2) the exercise of fundamental constitutional rights, and (3) other forms of freedom of choice or action."[224] The liberty guaranteed and protected by the Fourteenth Amendment includes the freedom to marry, establish a home, bring up children, live and work where one chooses, get a job, acquire useful knowledge, use and enjoy one's faculties, freedom from unauthorized physical restraint, and those privileges "essential to the orderly pursuit of happiness by free people."[225]

A state may enact legislation and deprive citizens of that state of any of these rights, but the citizens must first be afforded due process of law.[226] Due process is a course of legal proceedings according to the rules and principles

which have been established in our legal system for the protection and enforcement of private rights.[227] "Substantive" due process provides protection from arbitrary and unreasonable actions, while "procedural" due process requires that a party whose rights are to be affected be given notice and an opportunity to be heard before a court or other appropriate decision-making body.[228]

The appellant in Kraus v. City of Cleveland[229] alleged that he was deprived of his liberty to protect his health as he deems best, but did not claim that he was denied due process.[230] The Kraus trial court had applied the rational basis test to the fluoridation statute and found it to be a valid exercise of police power, which was affirmed by the Supreme Court of Ohio.[231] The problem in this and every other case is that the rational basis test does not apply if the statute being analyzed interferes with a fundamental right.[232] This is where the ambiguity arises, because the "law" is constantly changing, and what is unconstitutional today may be legal tomorrow.[233] Therefore, modern courts should reanalyze the limitations on personal liberty in light of recent Supreme Court cases. When it is finally decided that fluoridation of the public water systems impinges on a fundamental right, the rational basis test will be inappropriate, and strict judicial scrutiny by the courts will be required.

2. Abridgement of Privileges and Immunities Challenges

The problem in making a challenge that fluoridation of public water systems abridges a person's privileges and immunities is that the purpose of this clause is to protect "those rights peculiar to being a citizen of the federal government; it does not protect those rights which relate only to state citizenship."[234] The privileges and immunities challenges made in Teeter v. Municipal City of La Porte[235] and Paduano v. City of New York[236] have nothing to do with citizenship privileges in different states and thus are unfounded in the Constitution.[237] Accordingly, these cases were properly decided using the rational basis test of judicial review.

3. Denial of Equal Protection of the Laws Challenges

The last Fourteenth Amendment challenge to statutes authorizing the fluoridation of public water systems is that they violate equal protection of the laws.[238] In Chapman v. City of Shreveport,[239] the appellees argued that it was unreasonable to fluoridate the water when it affected only a limited class.[240] This argument, however, goes towards proving that fluoridation statutes are not narrowly tailored, not that they have been denied equal protection of the laws. Equal protection of the laws requires that individuals be treated in a similar manner.[241] No claim was made in Chapman that the appellee was not receiving the same protection being provided to other persons in similar circumstances; instead, he was trying to remove himself from the prophylactic medication being provided to children through fluoridated water.[242]

The Supreme Court of Louisiana stated that the exercise of police power is not objectionable solely because it does not apply to all classes, and held that the legislature is allowed to subject adults to fluoridation because the statute was not arbitrary, oppressive, or unreasonable.[243] This holding unduly expands the purpose of police power, which is supposed to promote the general welfare of the majority of the public. American households are composed mainly of adults, not children, and thus, fluoridating public water systems is an overbroad use of police power which can cause adverse health effects for the majority of the public.[244]

There are many reasonable alternatives available to parents who want their children to receive fluoride treatment, such as topical gels, mouth rinses, children's toothpaste, fluoride tablets, and drops.[245] A study found that "fluoride administered in tablet form or in vitamin preparations was more than twice as effective as fluoridated water in preventing cavities."[246] Children ages 7 to 12 had no tooth decay in 54% of those who took fluoride tablets or drops since infancy, while only 23.9% of adults with lifetime exposure to fluoridated water were cavity-free.[247] An additional argument supporting fluoride supplementation from sources other than public water systems is that fluoride tablets or drops can be administered in exact doses, but the amount of fluoride ingested from drinking tap water cannot be controlled.[248] These tablets or drops can then be discontinued after the permanent teeth have finished developing around age 10-12, or even sooner if deleterious side effects occur.[249]

4. Freedom of Religion Challenges

In addition to the Fourteenth Amendment challenges, many plaintiffs alleged that the state had violated their First Amendment right to freedom of religion.[250] The appellant in Kraus v. City of Cleveland[251] claimed that fluoridation of the water supply compels people to take a form of medication contrary to their religious beliefs in contravention of the First Amendment to the U.S. Constitution and the State Constitution.[252] The United States Supreme Court has ruled on similar challenges, and has held that the freedom to believe is absolute and is a fundamental right, but the freedom to act according to such beliefs is not fundamental and may be regulated under police powers.[253]

The Kraus court then tried to distinguish fluoridation from other infringements on freedom of religion.[254] The court recognized that although the City of Cleveland is the sole supplier of public drinking water within that city, there is no absolute duty on the part of the city to supply water.[255] Furthermore, courts have held that citizens opposed to drinking fluoridated water are free to buy non-fluoridated water because there is no direct compulsion to drink tap water.[256] The Kraus court recognized that obtaining non-fluoridated water may pose a problem of inconvenience for some and possibly of economics for others, but felt it was not a wholly impossible situation.[257]

The argument that there is no compulsion to drink fluoridated water is without merit. Public water systems were established to serve the public.[258] Since the majority of the public are adults, then fluoridating tap water to provide a drug to a minority of the public (children) defeats the whole purpose of the public water system. Besides the inconvenience and expense of having to buy bottled water, it is virtually impossible to escape eating processed foods that have been prepared using fluoridated water. Therefore, there is a compulsion to ingest fluoridated water, whether it comes from the faucet or from the foods we eat. Although the Kraus court felt there was no compulsion to drink fluoridated tap water, the ease at which it reached that conclusion suggests the worthlessness of the achievement. Despite the erroneous conclusion by the Kraus court, it correctly applied the rational basis test to the infringement of religion challenge, because the Supreme Court has not recognized the exercise of religion as an absolute constitutional right.[259]

5. Right of Privacy Challenges

The very last category of legal challenges to fluoridation of public water systems involves allegations that the state deprived someone of their implied Ninth Amendment constitutional right of personal privacy to be free from unwanted government intrusions.[260] The Minnesota Supreme Court recognized this implicit constitutional right in Minnesota State Board of Health v. City of Brainerd,[261] but felt that it only protected against intrusions that were unjustified or made improperly.[262] The Brainerd court felt that if much weight was given to the right of privacy argument, then people could start refusing to let the government make similar intrusions.[263] Other courts have acknowledged the "zone of privacy" espoused by the U.S. Supreme Court in Carey v. Population Services, Int'l.,[264] but have held that this zone is narrow and subject to limitations.[265]

Some states, however, have determined that fluoridation of public water systems falls within this zone of privacy. The trial court in Chapman v. City of Shreveport[266] concluded that fluoridation of the city water supply was not reasonably related to the public health, and that tooth decay is not a matter of public health.[267] Furthermore, it held that the choice to ingest fluoride is strictly "within the realm of private dental health and hygiene," and that every person should be free to choose his medical treatment for himself and his family.[268]

Along these same lines, the dissent in Minnesota State Board of Health v. City of Brainerd[269] realized that although the majority had used a balancing test to weigh the state's intrusion into the citizen's right of privacy, they failed to look at other alternative means that minimized intrusion.[270] Given that Minnesota's purpose was to make publicly-funded fluoride treatment readily available, the dissent felt that the city could have been compelled to provide fluorine tablets or dental applications to whomever wanted treatment, without infringing on the right of privacy of the majority.[271] Justice Yetka concluded by noting that there was not a compelling state interest to fluoridate the water, especially since it could possibly be carcinogenic, and that reasonable alternatives existed, which tipped the balance in favor of an individual's rights.[272]

The dissent in Kaul v. City of Chehalis[273] recognized that measures directly affecting the bodily integrity of a person represent the most penetrating exercise of police power.[274] Only the emergency of a present danger justifies quarantine, isolation, or compulsory treatment, and it is doubtful whether compulsory vaccination can be made without such danger.[275] Justice Hill, in his dissent, pointed out that any proposed health regulation must not impair essential rights and principles, and anyone who wants or needs fluorine can get a prescription for topical application, or ingest it by other ways.[276] Additionally, he noted that the United States Supreme Court has held that health regulations must not restrain personal liberty "under conditions essential to the equal enjoyment of the same right by others" or unless there is "pressure of great dangers" to the public's safety.[277]

Justice Hill reiterated the well known fact that "[w]hile dental caries may be termed a 'disease' which is prevalent in the teeth of almost everyone, it is not contagious or communicable in any way."[278] In addition, "[d]ental caries in no way endangers the public health in the sense that its existence in the teeth of one individual might adversely affect the personal health of any other individual."[279] Furthermore, Justice Hill felt that allowing the state to fluoridate public water systems would open the door to compulsory mass medication or preventative treatment for any disease without regard to a person's right to decide such matters for himself.[280] Justice Hill concluded that the "prevention of

dental caries by compulsory treatment of the teeth does not fall within the scope of protection of the public dental health for which the police power may be invoked."[281] He believed that education and persuasion, not compulsion, should be the government's goal if fluorine is actually the key to dental health.[282]

D. Supreme Court Stance on Unwanted Medical Treatment

In Cruzan v. Director, Missouri Department of Health,[283] the United States Supreme Court stated that although many state courts have analyzed the right to refuse medical treatment under the implied constitutional right of privacy, it "is more properly analyzed in terms of a Fourteenth Amendment liberty interest."[284] In Cruzan, the Supreme Court acknowledged that "[t]he principle that a competent person has a constitutionally protected liberty interest in refusing unwanted medical treatment may be inferred from our prior decisions."[285] Additionally, the Supreme Court assumed that the Constitution would grant a person "a constitutionally protected right to refuse lifesaving hydration and nutrition."[286] In a prior case, the Supreme Court held that "[t]he forcible injection of medication into a nonconsenting person's body represents a substantial interference with that person's liberty."[287] However, the court also recognized that while a person has a liberty interest under the Fourteenth Amendment Due Process Clause, whether the person's "constitutional rights have been violated must be determined by balancing his liberty interests against the relevant state interests."[288] This "relevant" state interest, also referred to as a "compelling" state interest,[289] is one which the state is forced or obliged to protect.[290] While all states have a compelling interest to prevent contagious diseases, such as the spread of smallpox in Jacobson v. Massachusetts, [291] tooth decay is not contagious, poses no risk of an outbreak, and thus is not a compelling interest such as would require state intervention. Accordingly, courts should apply a strict scrutiny standard of review when balancing a substantial liberty interest against fluoridation, which is, in effect, merely a state-mandated prophylactic measure for a noncontagious disease. A strict scrutiny standard requires that a state have a compelling interest to enact legislation, and that such legislation be narrowly tailored to achieve its purpose so as not to infringe on personal liberty interests protected by the Constitution.[292]

There is clearly no right or compelling interest for the federal government to mandate fluoridation of drinking water because it is known that fluoride is a contaminant which may have an adverse affect on the health of persons.[293] If states were bound by the Safe Water Drinking Act, then they would be prohibited from requiring fluoridation of the public water systems, despite their police power. This state police power is supposed to be used to promote the general health and welfare of the public, and should not be used as authority to purposely add contaminants into public drinking water. While reasonable minds may differ about whether the state's interest in health encompasses non-contagious diseases and whether this interest is compelling, fluoridation of public water systems does not pass constitutional muster because it fails the second prong of the strict scrutiny test: it is not narrowly tailored to achieve the legislature's purpose, and reasonable alternatives exist.

V. Conclusion

It is incumbent upon the United States Supreme Court to grant certiorari to the next fluoridation challenge brought based upon a due process violation of an individual's liberty interest. Whereas the Supreme Court has yet to resolve the issue of whether fluoridation invades a constitutionally protected interest when the state mandates the ingestion of a prophylactic drug to prevent a noncontagious disease, the Court has held, however, that a state may exercise its police power to protect the public from the spread of contagious disease. This distinction between contagious and noncontagious disease is critical because it determines the extent of the state interest when balancing the right of an individual to be free from compulsory medication against the state interest in attempting to prevent tooth decay by fluoridating public water systems.

The holding in Washington v. Harper[294] reflects the modern Supreme Court position, whereby "[t]he forcible injection of medication into a nonconsenting person's body represents a substantial interference with that person's liberty."[295] However, this holding is qualified by the caveat that whether this constitutionally protected liberty interest has been violated "must be determined by balancing that liberty interest against the relevant state interests."[296] The balancing is accomplished by subjecting fluoridation statutes to a strict scrutiny review in order to determine if they pass constitutional muster.

Because there is no compelling state interest to mandate prophylactic drugs for a noncontagious disease, the means of accomplishing the legislature's goals is not narrowly tailored, and reasonable alternatives exist, fluoridation statutes will fail the strict scrutiny test. Pursuant to the holdings in Harper and Cruzan, it is reasonably certain that fluoridation of public water systems will eventually be deemed a substantial invasion of personal liberty in violation of the Constitution of the United States of America.

Fluoridating public water in an attempt to target children whose permanent teeth are still developing is like using a shotgun to shoot an apple off someone's head; sure, you hit the apple, but the side effects are undesirable.

References

1. The Tenth Amendment, considered to be the source of state police power, provides that "[t]he powers not delegated to the United States by the Constitution, nor prohibited by it to the States, are reserved to the States respectively, or to the people." U.S. CONST. amend. X. See infra note 22.back 1back 1
2. See generally Michael S. Morgenstern, The Role of the Federal Government in Protecting Citizens from Communicable Diseases, 47 U. CIN. L. REV. 537 (1978).
3. "The term 'public water system' means a system for the provision to the public of water for human consumption through pipes or other constructed conveyances, if such system has at least fifteen service connections or regularly serves at least twenty-five individuals." Public Health Service (Safe Drinking Water) Act § 1401(4), 42 U.S.C. § 300f(4) (1994), as amended by Act of Aug. 6, 1996, Pub. L. No. 104-128, 1996 U.S.C.C.A.N. (110 Stat.) 1613, 1616 (1996) [hereinafter Safe Drinking Water Act].
4. A disease is a "destructive process in the body, with a specific cause and characteristic symptoms. WEBSTER's NEW UNIVERSAL UNABRIDGED DICTIONARY 523 (2d ed. 1983). Furthermore, tooth decay, technically called dental caries, is the destruction or necrosis of teeth. See DORLAND's ILLUSTRATED MEDICAL DICTIONARY 250 (27th ed. 1988). Therefore, since tooth decay is a destructive process in the body, it is a disease. See also infra note 5.
5. Dental caries is "[a] disease of calcified tissues of teeth characterized by demineralization of the inorganic portion and destruction of the organic matrix." 21 C.F.R. § 355.3(d) (1996).back 5back 5
6. Municipal providers include "a city, town, or other public body created by or pursuant to State law, or an Indian Tribe." Safe Drinking Water Act § 1401(10), 42 U.S.C. § 300f(10) (defining "municipality").
7. Fluoridation is an issue which has caused great controversy since the 1950s. Abundant opposition still exists today, as can be evidenced by surfing the Internet. See, e.g., Preventative Dental Health Association, The Dangers of Fluoridation (last modified Dec. 15, 1995) ; Gerard F. Judd, Ph.D & Chemist, Keep Your Teeth, We Now Know How!! Fluoridation Not the Answer (visited May 10, 1997) ; Michael Schachter, M.D., P.A., The Dangers of Fluoride and Fluoridation (visited May 10, 1997) ; Health Action Network Society, Is Fluoride Good For You? (visited May 10, 1997); Fluoride Issues (visited May 10, 1997).
8. See BUREAU OF CONSUMER PROTECTION - OFFICE OF CONSUMER/BUSINESS EDUCATION, FEDERAL TRADE COMMISSION, FACTS FOR CONSUMERS: WATER TESTING SCAMS. See also Exclusive Interview with Carol Browner, Administrator of the U.S. Environmental Protection Agency, POPULAR SCIENCE (visited May 10, 1997); Mark D. Uehling, How Safe is Your Water?, POPULAR SCIENCE, Oct. 1996, at 63.
9. See Fit to Drink?, CONSUMER REPORTS, Jan. 1990, at 30 (pertaining to chlorine, lead, nitrates, pesticides, radon, and other organic chemicals). See also National Primary Drinking Water Regulations, Fluoride, 50 Fed. Reg. 47,142, 47,146 (1985) (codified at 40 C.F.R. § 141.51) (pertaining to fluoride).
10. Fit to Drink?, CONSUMER REPORTS, Jan. 1990, at 30. See also Is Your Water Safe?, POPULAR SCIENCE (visited May 10, 1997) .back 10back 10
11. Fran Donegan, Water Treatment Basics, POPULAR MECHANICS, May 1993, at 61.
12. See, e.g., Smith v. State, 921 P.2d 632 (Alaska 1996). In Smith, excessive fluoride in the public water system caused widespread illness in the town of Hooper Bay, Alaska, and even resulted in the death of one resident. *See id.* at 633.
13. Only one case has challenged chlorination of the public water supply. See Commonwealth v. Town of Hudson, 52 N.E.2d 566 (Mass. 1943). The town of Hudson, Massachusetts rejected an order by the State Department of Public Health to chlorinate its water supply because they believed it would adulterate their otherwise pure water. *See id.* at 569, 572. The state had ordered chlorination of the water supply during the war to guard against sabotage by enemy agents or sympathizers.

See id. at 570. The town objected on grounds that its water supply was naturally pure. *See id.* at 572. The Supreme Judicial Court of Massachusetts upheld the order to chlorinate as a valid exercise of state police power to protect against disease-producing organisms. *See id.* at 571, 572.

14. See, e.g., De Aryan v. Butler, 260 P.2d 98 (Cal. 1953), cert. denied, 347 U.S. 1012 (1954) (earliest reported court challenge against fluoridation); Safe Water Association, Inc. v. City of Fond Du Lac, 516 N.W.2d 13 (Wis. Ct. App. 1994), review dismissed, 520 N.W.2d 91 (Wis. 1994) (most recently reported court challenge against fluoridation) and other cases cited infra. In addition to private citizen suits opposing fluoridation, many organizations are also fighting fluoridation. Some of these include: Global Alliance Against Fluoridation [New York, NY], The Anti-Fluoridation Association of Victoria [Melbourne, Australia], Citizens for Safe Drinking Water [San Diego, CA], Safe Water Coalition, Inc. [Orinda, CA], New York State Coalition Opposed to Fluoridation, Inc. [Old Bethpage, NY], New Zealand Pure Water Association Inc. [Bay of Plenty, New Zealand], Support Coalition [Eugene, OR], PA Mandatory Fluoridation Alert [Oakmont, PA], Safe Water Coalition of Washington State [Spokane, WA], and the National Pure Water Association [Wakefield, United Kingdom]. See Organizations Which Oppose Fluoridation (visited May 10, 1997) . Also, both the Environmental Defense Fund, Inc. and the Natural Resources Defense Council, Inc. (NRDC) have challenged the maximum fluoride levels in water set by the Environmental Protection Agency. See Environmental Defense Fund, Inc. v. Costle, 578 F.2d 337 (D.C. Cir. 1978); Natural Resources Defense Council, Inc. v. Environmental Protection Agency, 812 F.2d 721 (D.C. Cir. 1987).
15. See generally National Primary Drinking Water Regulations; Fluoride, 50 Fed. Reg. 47,142 - 47,155 (codified at 40 C.F.R. § 141.51).back 15back 15
16. See Preventative Dental Health Association, Adverse Health Effects Linked To Fluoride (last modified Jan. 11, 1996) (citing Perry D. Cohn, Ph.D., An Epidemiological Report on Drinking Water Fluoridation and Osteosarcoma in Young Males, New Jersey Department of Health, Environmental Health Service (Nov. 8, 1992).
17. *See id.* (citing Hip Fracture Rates Related to Fluoridated Water, Journal of the American Medical Association Vol. 264(4), 500-02 (1990), J.C. Robins & J.L. Ambrus, Studies on Osteoporosis IX. Effect of Fluoride on Steroid Induced Osteoporosis, Research Communications in Chemical Pathology and Pharmacology, Vol. 37, No. 3, 453-61 (1982).
18. *See id.* (citing S.C. Freni, Journal of Toxicology and Environmental Health, Vol. 42, 109-21 (1994).
19. *See id.* (citing M. Diesendorf, Tooth Decay Not Related to Fluoride Intake From Water, NATURE, Vol. 322, July 10, 1986; J. Colquhoun, Tooth Decay Related to Economics of Family, AMERICAN LABORATORY, Vol. 17, 1985, at 98-109; J. Colquhoun, COMMUNITY DENTISTRY AND ORAL EPIDEMIOLOGY, Vol. 13, 1985 at 37-41; JOHN YIAMOUYIANNIS, PH.D., FLUORIDE - THE AGING FACTOR (2d ed. 1986); D. Ziegelbecker, FLUORIDE, Vol. 14, 1981, at 123-28).
20. See National Primary Drinking Water Regulations; Fluoride, 50 Fed. Reg. 47,156, 47,171 (Appendix A - proposal for a new warning notice to the public, which reads: "Fluoride, at the appropriate levels in the drinking water of children up to the age of nine, reduces cavities."). See also AM. JUR. 3D Proof of Facts Taber's Cyclopedic Medical Dictionary 682 (16th ed. 1989), which states that "fluoride taken after the age of 8 to 10 will have little effect on the prevention of dental caries." back 20back 20
21. U.S. DEPARTMENT OF COMMERCE - BUREAU OF THE CENSUS, HOUSEHOLD AND FAMILY CHARACTERISTICS: MAR. 1994 viii (Sept.1995).
22. Police power is not explicitly provided for in the Constitution. It is recognized as a power inherent in state sovereignty to protect the health, safety, general welfare, and morals of the public, secured by the Tenth Amendment. See Lochner v. New York, 198 U.S. 45, 53 (1905). "Police power" is a general term containing many ramifications and has never been pinpointed as to its exact meaning." City Comm'n of the City of Fort Pierce v. State ex rel. Altenhoff, 143 So. 2d 879, 889 (Fla. 1962).

23. See Weinberger v. Salfi, 422 U.S. 749, 769 (1975) (citing Richardson v. Belcher, 404 U.S. 78, 84 (1971).
24. Village of Belle Terre v. Boraas, 416 U.S. 1, 8 (1974) (citing Reed v. Reed, 404 U.S. 71, 76 (1971).
25. Safe Water Ass'n Inc. v. City of Fond Du Lac, 516 N.W.2d 13, 17 (Wis. Ct. App. 1994), review dismissed, 520 N.W.2d 91 (Wis. 1994).back 25back 25
26. See San Antonio Independent School District v. Rodriguez, 411 U.S. 1, 38 (1973). See also BLACK'S LAW DICTIONARY 872 (6th ed. 1991) (defining rational basis test).
27. See Doe v. Bolton, 410 U.S. 179, 215 (1973) (Douglas, J., concurring) (citing Jacobson v. Commonwealth of Massachusetts, 197 U.S. 11 (1905), which upheld state imposed vaccinations to prevent epidemics).
28. See San Antonio Independent School District v. Rodriguez, 411 U.S. 1, 37-38 (1973). See also Doe v. Bolton, 410 U.S. at 216 (Douglas, J., concurring) (stating that "[u]nless regulatory measures are so confined and are addressed to the specific areas of compelling legislative concern, the police power would become the great leveler of constitutional rights and liberties").
29. See Roe v. Wade, 410 U.S. 113, 155 (1973).
30. Washington v. Harper, 494 U.S. 210, 229 (1990).back 30back 30
31. There is no compelling state interest to impose regulations for noncontagious or nonhazardous health concerns. See, e.g. Carey v. Population Services International, 431 U.S. 678, 690 (1977) (holding that a statute controlling the distribution of nonhazardous contraceptives "bears no relation to the State's interest in protecting [public] health"). See also Michael S. Morgenstern, The Role of the Federal Government in Protecting Citizens from Communicable Diseases, 47 U. CIN. L. REV. 537, 538 n.2 (1978), which recognizes the governmental interest only in communicable diseases, which are "disease[s] the causative agent of which may pass or be carried from one person to another directly or indirectly." (citing DORLAND's ILLUSTRATED MEDICAL DICTIONARY 455 (25th ed. 1974).
32. 497 U.S. 261 (1990).
33. 494 U.S. 210 (1990).
34. A binary compound is composed of two elements. See WEBSTER'S NEW UNIVERSAL UNABRIDGED DICTIONARY 183 (2d ed. 1983).
35. See DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 643 (27th ed. 1988).back 35back 35
36. See John J. Miller, Ph.D., The Fluoride Ion, PREVENTION, July 1964, at 56.
37. See GEORGE L. WALDBOTT, M.D., FLUORIDATION: THE GREAT DILEMMA 20 (1978).
38. See Jonathan Forman, M.D., What Looks Like a Neurosis May Be a Fluorosis, PREVENTION, June 1964, at 92. See also John J. Miller, Ph.D., The Fluoride Ion, PREVENTION, July 1964, at 57-58.
39. See National Primary Drinking Water Regulations; Fluoride, 50 Fed. Reg. 47,142, 47,145 (Table 1) (codified at 40 C.F.R. § 141.51). See also Kraus v. City of Cleveland, 116 N.E.2d 779, 792 (Ohio 1953).
40. See 10 COLLIER'S ENCYCLOPEDIA 109 (1992). This observation was limited to only those children whose teeth were still developing when they drank the fluoridated water. See id.back 40back 40
41. See Michael Wollan, Controlling the Potential Hazards of Government-Sponsored Technology, 36 GEO. WASH. L. REV. 1105, 1128 (1968).
42. WEBSTER's NEW UNIVERSAL UNABRIDGED DICTIONARY 1118 (2d ed. 1983) (defining "medicine").
43. ANNE-LISE GOTZSCHE, THE FLUORIDE QUESTION 68 (1975).
44. For the Material Safety Data Sheet (MSDS) detailing the characteristics and health hazards of sodium fluoride, see (visited May 10, 1997).
45. The chemical symbol of sodium fluorosilicate, a salt of hydrofluorosilicic acid, is Na2SiF6. See GEORGE L. WALDBOTT, M.D., FLUORIDATION: THE GREAT DILEMMA 24 (1978).back 45back 45
46. Also called hydrofluosilicic acid, its chemical symbol is H2SiF6. *See id.* Hydrofluorosilicic acid and sodium fluorosilicate are used commercially for electroplating, water fluoridation, wood preservation, concrete hardening, a flux for metal casting, production of synthetic mica, extraction of zirconium, and for making acid resistant cements. *See id.* at 24, 25.
47. *See id.* at 25.

48. FIFRA requires all new pesticides to be registered, as well as the reregistration of pesticides first registered before November 1, 1984. See Federal Insecticide, Fungicide, and Rodenticide Act, 7 U.S.C. §§ 136a, 136a-1 (exceptions omitted). The Special Review and Reregistration Division in EPA's Office of Pesticide Programs publishes a document called the "Rainbow Report" (Status of Pesticides in Reregistration and Special Review) which lists sodium fluoride as an active ingredient in pesticides. See Environmental Protection Agency, Pesticide Active Ingredients Index, (visited May 10, 1997). Sodium Fluoride was originally labeled as an "economic poison" under FIFRA. See 7 U.S.C. § 135a(a)(4) (1981) (omitted). "The term 'economic poison' means (1) any substance or mixture of substances intended for preventing, destroying, repelling, or mitigating any insects, rodents, nematodes, fungi, weeds, and other forms of plant or animal life or viruses, except viruses on or in living man or other animals, which the Administrator shall declare to be a pest ...." 7 U.S.C. § 135(a) (omitted).
49. See 7 U.S.C. § 136(q)(2)(D) (1991) (emphasis added).
50. Promotion and Application of Water Fluoridation: Hearings Before the Dept. of Labor and Health, Education and Welfare Appropriations, 89th Cong., Vol. 5, (1967) (statement of Dr. John W. Knutson, Chief, Division of Dental Public Health, Public Health Service, at the proceedings of the Fourth Annual Conference of State Dental Directors with the Public Health Service and the Children's Bureau, held at the Federal Security Building in Washington, D.C., on June 6-8, 1951).back 50back 50
51. *See id.*
52. See 40 C.F.R. § 141.23(k)(3)(ii) (1994). See also 40 C.F.R. § 141.51 (1996) (contaminant table listing fluoride between cyanide and lead). Additionally, "[f]rom today's perspective, health professionals were reckless to promote mass fluoridation as early as 1951" because fluoride is an acute poison and only crude risk data was available then. Allan Mazur, Why Do We Worry About Trace Poisons?, RISK: HEALTH, SAFETY AND ENVIRONMENT 35, 41 (Winter 1996). Mr. Mazur believes that in 1951 health professionals were not as concerned about chronic exposure to trace poisons as we are today, and that if fluoridation of public water systems were proposed today, supported only by the risk data available in 1951, it would not be approved. *See id.* at 41-42.
53. See generally National Primary Drinking Water Regulations, Fluoride, 50 Fed. Reg. 47,142 - 47,155 (codified at 40 C.F.R. § 141.51).
54. *See id.* at 47,145.
55. Fluorosis is "a condition due to exposure to excessive amounts of fluorine or its compounds," resulting in combined osteosclerosis and osteomalacia (alternating brittle and soft areas of bone). DORLAND's ILLUSTRATED MEDICAL DICTIONARY 643 (27th ed. 1988).back 55back 55
56. A mutation is "a sudden variation in some inheritable characteristic . . . as distinguished from a variation resulting from generations of gradual change." WEBSTER's NEW UNIVERSAL UNABRIDGED DICTIONARY 1186 (2d ed. 1983).
57. See National Primary Drinking Water Regulations, Fluoride, 50 Fed. Reg. 47,142, 47,145 (codified at 40 C.F.R. § 141.51).
58. See JOHN YIAMOUYIANNIS, PH.D., FLUORIDE - THE AGING FACTOR (1983).
59. *See id.* passim.
60. See National Primary Drinking Water Regulations, Fluoride, 50 Fed. Reg. 47,142, 47,153 (codified at 40 C.F.R. § 141.51).back 60back 60
61. *See id.* at 47,151.
62. Oncogenicity is a factor that causes the development of cancer. See CONCISE SCIENCE DICTIONARY 484 (2d ed. 1991).
63. See National Primary Drinking Water Regulations Fluoride, 50 Fed. Reg. 47,142, 47,147 (codified at 40 C.F.R. § 141.51). In 1993, the EPA announced that it would not revise the maximum contaminant level goal (MCLG) for fluoride after it had considered recent reports concerning the health effects from ingesting fluoride. See Drinking Water Maximum Contaminant Level Goal; Fluoride, 58 Fed. Reg. 68,826 (1993). The decision was based on the results of a study performed by the National Academy of Sciences (NAS), which saw no immediate need to change the MCLG, but felt that further research was needed in the areas of dental fluorosis, bone strength and fractures, and carcinogenicity. *See id.* at 68,827. Accordingly, the EPA

announced that it will continue to solicit public comments on this issue, and hopes to issue a final decision after the NAS research is concluded, which it anticipates will be around the year 2001. *See id.*

64. See National Primary Drinking Water Regulations; Fluoride, 50 Fed. Reg. 47,142 (summary) (codified at 40 C.F.R. § 141.51).

65. *See id.* back 65back 65

66. *Id.* at 47,153.

67. Codified at 21 C.F.R. Part 355 (1996).

68. The Food and Drug Administration (FDA) is now allowing bottled water to have fluoride added, but requires that it have a label listing fluoride as an ingredient. See 60 Fed. Reg. 57,076, 57,079 (1995) (codified at 21 C.F.R. § 165.110(b)(4)(ii) (1997).

69. 21 C.F.R. § 355.3(c) (1996).

70. 70. See 21 C.F.R. § 355.10(a) (1996).back 70 back 70

71. *See id.* § 355.10(b) (1996).

72. *See id.* § 355.10(c) (1996).

73. A dentifrice is "[a]n abrasive-containing dosage form for delivering an anticaries drug to the teeth." 21 C.F.R. § 355.3(e) (1996).

74. One part per million is the same concentration as one milligram per liter (mg/L). See I. TAXEL, CONVERSION FACTORS (1964).

75. See 21 C.F.R. § 355.10(a)(1) (1996).

76. *See id.* But see § 355.10(c)(i) (1996) (requiring stannous fluoride dentifrices to have a fluoride ion concentration of at least 700 ppm). Stannous fluoride is a compound of fluorine and tin. See WEBSTER's NEW UNIVERSAL UNABRIDGED DICTIONARY 1773 (2D ed. 1983) Stannous fluoride has a chemical symbol of SnF2. See GEORGE L. WALDBOTT, M.D., FLUORIDATION: THE GREAT DILEMMA 25 (1978).

77. A fluoride ion is "[t]he negatively charged atom of the chemical element fluorine." 21 C.F.R. § 355.3(g) (1996).

78. A treatment rinse is "[a] liquid dosage form for delivering an anticaries drug to the teeth." *Id.* § 355.3(j).

79. *See id.* § 355.10(a)(3).

80. *See generally id.* § 355.50 (1996) (Labeling of anticaries drug products).back 80back 80

81. *Id.* § 355.50(c)(1).

82. *See id.* § 355.50(d)(2), (d)(4). In fact, many toothpastes contain a warning stating that children under 6 years old should use only a pea-sized amount and should be supervised to prevent swallowing. Such a warning is needed because swallowing too much fluoride can be fatal. See infra note 83.

83. See Robert D. McFadden, $750,000 Given in Child's Death in Fluoride Case, N.Y. TIMES, Jan. 20, 1979, at 23 (a 3 year old boy died after receiving an overdose of fluoride at a New York City Dental Clinic).

84. See supra note 38 and accompanying text.

85. See GEORGE L. WALDBOTT, M.D., FLUORIDATION: THE GREAT DILEMMA 20 (1978).back 85back 85

86. See supra note 69 and accompanying text.

87. See generally National Primary Drinking Water Regulations; Fluoride, 50 Fed. Reg. 47,142 - 47,155 (codified at 40 C.F.R. § 141.51).

88. 15 A. 440 (Pa. 1888).

89. *See id.* at 441.

90. Wholesome water must "not be injurious to the health of those using it." *Id.* at 442.back 90 back 90

91. *See id.*

92. See id at 443.

93. See 15 A. at 443.

94. See EDWARD J. CALABRESE ET AL., SAFE DRINKING WATER ACT 1 (1989).

95. Originally promulgated as the Public Health Service Act, ch. 373, title XIV (1944). The Safe Drinking Water Act is now codified at 42 U.S.C. §§ 300f to 300j-26, as amended by Act of Aug. 6, 1996, Pub. L. No. 104-128, 1996 U.S.C.C.A.N. (110 Stat.) 1613-93 (1996).

96. See National Primary Drinking Water Regulations; Fluoride, 50 Fed. Reg. 47,142 (summary) (codified at 40 C.F.R. § 141.51). See also EDWARD J. CALABRESE ET AL., SAFE DRINKING WATER ACT 17-18.

97. See Safe Drinking Water Act § 1412(a)(2), 42 U.S.C. § 300g-l(a)(2). See also EDWARD J. CALALBRESE ET AL., SAFE DRINKING WATER ACT 19.

98. Safe Drinking Water Act § 1412(b)(3)(A), 42 U.S.C. § 300g-l(b)(3)(A).

99. "The most clearly established fluoride-induced bone complaint is crippling skeletal fluorosis, also called chronic fluoride toxicity ...." ANNE-LISE GOTZSCHE, THE FLUORIDE QUESTION 86 (1975).

100. See National Primary Drinking Water Regulations; Fluoride, 50 Fed. Reg. 47,142 (summary) (codified at 40 C.F.R. § 141.51).back 100back 100

101. The maximum contaminant level is the maximum permissible level of a contaminant in the public water system which may adversely affect the public welfare. See Safe Drinking Water Act § 1401(2), (3), 42 U.S.C. § 300f(2), (3).
102. See National Primary Drinking Water Regulations, Fluoride, 50 Fed. Reg. 47,142, 47,143 (codified at 40 C.F.R. § 141.51).
103. *See id.*
104. Fluorosis is "[a] condition caused by an excessive intake of fluorides (2 or more p.p.m. in drinking water), characterized mainly by mottling, staining, or hypoplasia of the enamel of the teeth, although skeletal bones are also affected. STEDMAN's MEDICAL DICTIONARY 599 (25th ed. 1990).
105. See National Primary Drinking Water Regulations, Fluoride, 50 Fed. Reg. 47,142, 47,143 (codified at 40 C.F.R. § 141.51).
106. See id at 47,145.
107. *See id.* at 47,143.
108. See 40 C.F.R. § 141.62 (1996).
109. National Primary Drinking Water Regulations; Fluoride, 50 Fed. Reg. 47,142, 47,143 (codified at 40 C.F.R. § 141.51)

(emphasis added).
110. *Id.* at 47,142. back 110back 110
111. *Id.* at 47,155.
112. See Safe Drinking Water Act §§ 1401(2), 1412(c), 42 U.S.C. §§ 300f(2) 300g-l(c).
113. National Secondary Drinking Water Regulations, 44 Fed. Reg. 42,195 (1979) (codified at 40 C.F.R. § 143.3).
114. See National Primary Drinking Water Regulations, Fluoride, 50 Fed. Reg. 47,156 (1985).
115. See 40 C.F.R. §143.3 (1996).
116. *Id.* § 143.5. Despite the requirement for this notice, the EPA "has been criticized for failure to enforce required water quality standards and for not reporting to the public the failure of municipal water companies to meet standards." A. DAN TARL0CK ET AL., WATER RESOURCE MANAGEMENT 16 (4th ed. 1993).
117. See National Primary Drinking Water Regulations; Fluoride, 50 Fed. Reg. 47,142, 47,153 (codified at 40 C.F.R. § 141.51).
118. *See id.* at 47,145. If an adult drinks more than two liters of water per day, it logically follows that they are ingesting more fluoride than is calculated to be safe by the EPA.
119. "The term 'supplier of water' means any person who owns or operates a public water system." Safe Drinking Water Act § 1401(5), 42 U.S.C. § 300f(5).
120. See EDWARD J. CALABRESE ET AL., SAFE DRINKING WATER ACT 112 tbl. 5.back 120back 120
121. *See id.* at 108. SNCs "are those systems which have the most serious and more frequent violations." *Id.* at 106.
122. Cases that the Supreme Court has declined to hear include: Birnel v. Town of Fircrest, 335 P.2d 819 (Wash. 1959), appeal dismissed, 361 U.S. 10 (1959), reh'g. denied, 361 U.S. 904 (1959); Chapman v. City of Shreveport, 74 So. 2d 142 (La. 1954), appeal dismissed, 348 U.S. 892 (1954), City of Canton v. Whitman, 337 N.E.2d 766 (Ohio 1975), appeal dismissed, 425 U.S. 956 (1976); De Aryan v. Butler, 260 P.2d 98 (D. Cal. 1953), cert. denied, 347 U.S. 1012 (1954); Dowell v. City of Tulsa, 273 P.2d 859 (Okla. 1954), cert. denied, 348 U.S. 912 (1955); Kraus v. City of Cleveland, 116 N.E.2d 779 (Ohio Com. Pl. 1953), aff'd, 127 N.E.2d 609 (Ohio 1955), appeal dismissed, 351 U.S. 935 (1956); Minnesota State Bd. of Health v. City of Brainerd, 241 N.W.2d 624 (Minn. 1976), appeal dismissed, 429 U.S. 803 (1976); Paduano v. City of New York, 257 N.Y.S.2d 531 (Sup. Ct. 1965), aff'd, 24 A.D.2d 437 (N.Y.A.D. 1965), aff'd, 218 N.E.2d 339 (N.Y. 1966), cert. denied, 385 U.S. 1026 (1967); Readey v. St. Louis County Water Co., 352 S.W.2d 622 (Mo. 1961), appeal dismissed and cert. denied, 371 U.S. 8 (1962), reh'g denied, 371 U.S. 906 (1962); Safe Water Foundation of Texas v. City of Houston, 661 S.W.2d 190 (Tex. Ct. App. 1983), appeal dismissed, 469 U.S. 801 (1984); Schuringa v. City of Chicago, 198 N.E.2d 326 (Ill. 1964), cert. denied, 379 U.S. 964 (1965).
123. 257 N.Y.S.2d 531 (Sup. Ct. 1965).
124. Id at 542. The Paduano court recognized that the judiciary did not have power to impose fluoridation on anyone, but if some proof was proffered that fluoridation has harmful side effects and is not in the interests of the community, they might be able to overrule the

legislation authorizing it. *See id.*
125. *See id.* at 538 n.*.back 125back 125
126. The Supreme Court has noted "that all a denial of a petition for a writ of certiorari

means is that fewer than four members of the Court thought it should be granted." Maryland v. Baltimore Radio Show, 338 U.S. 912, 919 (1950). The Court also stated that they have "rigorously insisted that such a denial carries with it no implication whatever regarding the Court's views on the merits of a case which it has declined to review." *Id.* This has been said again and again, and the Court repeatedly has to reiterate this admonition. *See id.* Therefore, all courts that declined to rule on the constitutional challenges to fluoridation because they interpreted a denial of certiorari as the equivalent to an affirmation of the lower court's decision were wrong! For a detailed analysis of what certiorari denial constitutes, see Peter Linzer, The Meaning of Certiorari Denials, 79 COLUM. L. REV. 1227 (1979).

127. 260 P.2d 98 (D. Cal. 1953).
128. *See id.* at 99.
129. *See id.* at 101. See also Young v. Board of Health of Borough of Somerville, 293 A.2d 164 (N.J. 1972); Rogowski v. City of Detroit, 132 N.W.2d 16 (Mich. 1965). An act performed without any authority to do so is known as "ultra vires." An "[u]ltra vires act of [a] municipality is one which is beyond powers conferred upon it by law. BLACK'S LAW DICTIONARY 1057 (6th ed. 1991).
130. 260 P.2d at 101.back 130back 130
131. *Id.*
132. See id
133. *See id.* at 101,102.
134. *See id.* at 102.
135. See De Aryan v. Butler, 260 P.2d at 99, 103.
136. *See id.* at 102.
137. 417 P.2d 632 (1966).
138. *See id.* at 632,633.
139. *See id.*
140. *See id.* at 634.back 140back 140
141. *See id.* at 635.
142. 417 P.2d at 635.
143. Wilson v. City of Mountlake Terrace, 417 P.2d 632 635 (Wash. 1966).
144. See Dowell v. City of Tulsa, 273 P.2d 859, 860 (Okla. 1954).
145. *Id.* at 861.
146. *Id.*
147. *Id.*
148. *Id.* at 862 (quoting 11 AM. JUR. Constitutional Law § 271, 1023).
149. 66 N.W.2d 207 (N.D. 1954).
150. *See id.* at 209.back 150back 150
151. *See id.*
152. *Id.* at 211 (citing 56 AM. JUR. Waterworks § 75 at 76, § 79 at 983).
153. *See id.* at 209, 212.
154. A "demurrer" is "[a]n assertion that the complaint does not set forth a cause of action upon which relief can be granted." BLACK'S LAW DICTIONARY 298 (6th ed. 1991).
155. See 66 N.W.2d at 212. The decision of the district court on remand is unreported.
156. U.S. CONST. amend. XIV, § 1.
157. 260 P.2d 98 (D. Cal. 1953).
158. 127 N.E.2d 609 (Ohio 1955), appeal dismissed, 351 U.S. 935 (1956).
159. See id at 610.
160. *Id.* See also Readey v. St. Louis County Water Co., 352 S.W.2d 622 628-32 (Mo. 1961), cert. denied, 371 U.S. 8 (1962).back 160back 160
161. See 116 N.E.2d at 794.
162. *See id.* at 794-95.
163. *See id.* at 795.
164. *Id.*
165. *Id.* at 795-96.
166. See 116 N.E.2d at 794-95.
167. 139 N.E.2d 158 (Inc. 1956).
168. The Privileges and Immunities Clause provides that "[n]o State shall make or enforce any law which shall abridge the privileges or immunities of citizens of the United States." U.S. CONST. amend XIV, § 1.
169. See 139 N.E.2d at 160.
170. *Id.* at n.2.back 170back 170
171. *Id.* at 161.
172. 257 N.Y.S.2d 531 (Sup. Ct. 1965).
173. *See id.* at 538.
174. *Id.* at 539.
175. "Sui juris" means "possessing full social and civil rights; not under any legal disability, or the power of another, or guardianship." BLACK'S LAW DICTIONARY 1000 (6th ed. 1991).
176. 257 N.Y.S.2d at 541.
177. Stare decisis is a policy of courts to abide by decided cases and not disturb a settled principle of law. See BLACK'S LAW DICTIONARY 978 (6th ed. 1991).
178. See 257 N.Y.S.2d at 542.
179. The Equal Protection Clause provides that [n]o State shall . . . deny to any person within its jurisdiction the equal protection of the laws." U.S. CONST. amend. XIV, § 1.
180. See, e.g. Chapman v. City of Shreveport, 74 So. 2d 142, 146 (La. 1954) appeal dismissed,

348 U.S. 892 (1954). See also Hall v. Bates, 148 S.E.2d 345 (S.C. 1966), Kraus v. City of Cleveland, 116 N.E.2d 779 (Ohio Com. Pl. 1953) aff'd, 127 N.E.2d 609 (Ohio 1955), appeal dismissed, 351 U.S. 935 (1956). For a classification involving age, see City Comm'n of Fort Pierce v. State ex ref. Altenhoff, 143 So. 2d 879 (D.C. Fla. 1962). In this case, plaintiffs alleged that fluoridation of the water system "is violative of the constitutional guarantee against Class Legislation in that its proponents only claim it is beneficial to children of the age group of one to fourteen years." *Id.* at 881.back 180back 180

181. See Zucht v. King, 260 U.S. 174, 177 (1922). See also West Coast Hotel Co. v. Parrish, 300 U.S. 379, 400 (1937); Sturges & Burn Mfg. Co. v. Beauchamp, 231 U.S. 320, 326 (1913).
182. Chapman v. City of Shreveport, 74 So. 2d 142, 145 (La. 1954).
183. Readey v. St. Louis County Water Co., 352 S.W.2d 622, 632 (Mo. 1961).
184. U.S. CONST. amend. I.
185. See, e.g., Teeter v. Municipal City of La Porte, 139 N.E.2d 158 (Inc. 1956) (challenging a municipal ordinance on the grounds that it violated the rights of those whose religious beliefs were opposed to medication).
186. 116 N.E.2d 779 (Ohio Com. Pl. 1953).
187. *Id.* at 805.
188. Id at 805, 806, (citing Cantwell v. State of Connecticut, 310 U.S. 296 (1940).

189. *Id.* at 808.
190. U.S. CONST. amend. IX.back 190back 190
191. See Minnesota State Bd. of Health v. City of Brainerd, 241 N.W.2d 624, 630 (Minn. 1976), appeal dismissed, 429 U.S. 803 (1976).
192. *Id.* at 631 (quoting Eisenstadt v. Baird, 405 U.S. 438, 453 (1972).
193. *Id.*
194. *Id.* (quoting Schmerber v. California, 384 U.S. 757 (1966).
195. See id at 632.
196. See 241 N.W.2d at 632.
197. *Id.* at 633.
198. 516 N.W.2d 13 (Wis. Ct. App. 1994), review dismissed, 520 N.W.2d 91 (Wis. 1994).
199. *See id.* at 17.

200. 69 N.W.2d 242 (Wis. 1955).back 200back 200
201. 381 U.S. 479 (1965).
202. 410 U.S. 113 (1973).
203. See 516 N.W.2d at 17, 18.
204. 431 U.S. 678 (1977).
205. See 516 N.W.2d at 18.
206. *See id.*
207. Kraus v. City of Cleveland, 127 N.E.2d 609, 611 (Ohio 1955).
208. See supra note 126 and accompanying text.
209. See supra Part III.A.

210. 260 P.2d 98 (D. Cal. 1953).back 210back 210
211. *See id.* at 101.
212. *Id.*
213. *See id.* at 102.
214. 417 P.2d 632 (Wash. 1966).
215. *See id.* at 633.
216. *See id.* at 635.
217. See supra Part III.B.
218. 273 P.2d 859 (Okla.1954).
219. *See id.* at 861,863.

220. 66 N.W.2d 207 (N.D. 1954).back 220back 220
221. See id at 212.
222. See supra Part III.C.1.
223. See U.S. CONST. amend. XIV, § 1 (emphasis added).
224. JOHN E. NOWAK & RONALD D. ROTUNDA, CONSTITUTIONAL LAW, § 13.4, at 519 (5th ed. 1995). See also BLACK'S LAW DICTIONARY 633 (6th ed. 1991) (liberty amounts to freedom from all restraints except those justly imposed by law which are not arbitrary and are "reasonable regulations and prohibitions imposed in the interests of the community.")

225. BLACK'S LAW DICTIONARY 633 (6th ed. 1991).back 225back 225
226. See U.S. CONST. amend. XIV, § 1.
227. See JOHN E. NOWAK & RONALD D. ROTUNDA, CONSTITUTIONAL LAW, § 13.8, at 548 (noting that "[t]he adversary process is best designed to safeguard individual rights against arbitrary action by the government."). See also BLACK'S LAW DICTIONARY 346 (6th ed. 1991) (defining due process of law).
228. See, e.g., Washington v. Harper, 494 U.S. 210 (1990) cited in JOHN E. NOWAK & RONALD D. ROTUNDA, CONSTITUTIONAL LAW, § 10.6, at 358. The Supreme Court's analysis of the substantive due process issue found that the state interest in protecting persons from a psychotic prisoner with whom he might come in contact with was Reasonably related to a legitimate penological interest. JOHN E. NOWAK & RONALD D. ROTUNDA,

CONSTITUTIONAL LAW, § 10.6, at 358 (quoting Turner v. Safley, 428 U.S. 78 (1987). There was a separate procedural due process issue in Washington, which was whether twenty-four hour notice prior to a hearing before a medical board complied with due process principles. *See id.* at 358-59. See also BLACK'S LAW DICTIONARY 836, 977 (6th ed. 1991) (defining procedural due process and substntive due process, respectively).

229. 127 N.E.2d 609 (Ohio 1955).

230. *See id.* at 610.back 230back 230

231. *See id.* at 613-14.

232. See supra note 28 and accompanying text.

233. See, e.g., Roe v. Wade, 410 U.S. 113 (1973).

234. JOHN E. NOWAK & RONALD D. ROTUNDA, CONSTITUTIONAL LAW, § 10.3, at 343 (citing the Slaughter-House Cases, 83 U.S. (16 Wall.) 36 (1873). This case held that "the clause only refers to uniquely federal rights such as the right to petition Congress, the right to vote in federal elections, the right to interstate travel or commerce, the right to enter federal lands, or the rights of a citizen while in custody of federal officers." *Id.* (citing Slaughter-House Cases at 79- 81).

235. 139 N.E.2d 158 (Inc. 1956).

236. 257 N.Y.S.2d 531 (Sup. Ct. 1965).

237. See supra Part III.C.1.(b).

238. See supra Part III.C.1.(c).

239. 74 So. 2d 142 (La. 1954).

240. *See id.* at 146.back 240back 240

241. Fluoridation opponents raise the equal protection issue because fluoridation is purported to benefit only children under the age of nine, thus creating a special class. However "[t]he Supreme Court has not found that any form of heightened judicial scrutiny should be used when reviewing classifications that are based on age." JOHN E. NOWAK & RONALD D. ROTUNDA, CONSTITUTIONAL LAW, § 14.3, at 609 n.38. The Supreme Court uses the rational basis test when the classification does not relate to a fundamental right. *See id.*

242. See 74 So. 2d at 146.

243. *See id.* at 146, 147.

244. See supra Part I.

245. See generally 21 C.F.R. Part 355, Anticaries Drug Products for Over- the-Counter Human Use. There are also many fluoridated products available by prescription.back 245back 245

246. GEORGE L. WALDBOTT, M.D., FLUORIDATION: THE GREAT DILEMMA 307 (1978).

247. *See id.*

248. *See id.* A further argument against fluoridated water can be made in that many children do not drink tap water at all. We are a society of processed food and beverages. Infants drink canned formula, and children drink bottled milk, juice, and soft drinks. Rare is the child who will reach for a glass of tap water when thirsty. However, some childrens' drinks, such as Kool-Aid®, do require tap water to make. But even these "mix it yourself" drinks will not contain artificial fluoridation if the water is drawn from a private well. Thus, the entire purpose of fluoridation is being unintentionally circumvented by the advent of ready-to-drink beverages, unless of course, the beverage manufacturer uses fluoridated water.

249. *See id.*

250. See supra Part III.C.2.back 250back 250

251. 116 N.E.2d 779 (Ohio 1953), aff'd, 127 N.E.2d 609 (Ohio 1955), appeal dismissed, 351 U.S. 935 (1956).

252. *See id.* at 805.

253. See Cantwell v. State of Connecticut, 310 U.S. 296 (1940).

254. See 116 N.E.2d at 806-08.

255. *See id.* at 807.

256. See Chapman v. City of Shreveport, 74 So. 2d 142, 146 (La. 1954).

257. See 116 N.E.2d at 807.

258. The public does not have a choice of providers from whom it may receive tap water, however. One of the leading experts in water law has correctly categorized those receiving water from a public water system as "captive consumers." See A. Dan Tarlock, Safe Drinking Water: A Federalism Perspective, 21 WM & MARY L. REV. 233, 239 (1997). The argument has been made that since water is an article of commerce under Sporhase v. Nebraska ex rel. Douglas, 458 U.S. 941 (1982), then consumers have a "freedom of choice" to seek an alternate source for this "product," such as bottled spring water. The problem with this argument is that it fails to recognize that the municipality has a monopoly over the public water system; consumers cannot switch to a different provider, as is possible with telephone service.

The United States Supreme Court has held that police power measures which impose an

undue cost and inconvenience on commerce are an unconstitutional burden. See, e.g., Bibb v. Navajo Freight Lines, Inc., 359 U.S. 520 (1959). Purchasing bottled water to avoid drinking fluoridated tap water is both expensive and inconveient, and thus is an impermissible burden to be placed on the public. Further, the highly touted charcoal filters, such as the Brita® water pitcher, are not able to filter out fluoride. Expensive reverse osmosis systems or distillation is required to extract fluoride out of the water. Besides this undue burden, it would be virtually impossible to exercise your "freedom of choice" if you are at a restaurant, school, library, shopping mall, the movies, or anywhere away from your supply of bottled water. Therefore, the "freedom of choice" argument is untenable and merely an exercise in obfuscation.back 258back 258

259.*See id.* at 808.

260.See supra Part III.C.3.back 260back 260

261.241 N.W.2d 624 (Minn. 1976).

262.*See id.* at 631.

263.*See id.* at 632.

264.431 U.S. 678 (1977).

265.See Safe Water Association, Inc. v. City of Fond Du Lac, 516 N.W.2d 13, 18 (Wis. Ct. App. 1994).

266.74 So. 2d 142.

267.*See id.* at 143.

268.*Id.*

269.241 N.W.2d 624 (Minn. 1976).

270.*See id.* at 634 (Yetka, J., dissenting).back 270 back 270

271.See id

272.*See id.* at 634-35.

273.277 P.2d 352 (Wash. 1954)(en banc).

274.*See id.* at 359 (Hill, J., dissenting, citing Freund on Police Power 116, § 123).

275.*See id.*

276.*See id.*

277.Id (citing Jacobson v. Massachusetts, 197 U.S. 11 (1905).

278.277 P.2d at 359.

279.*Id.* at 359-60.

280.*See id.* at 360.back 280back 280

281.*Id.* at 361.

282.*See id.* (emphasis added).

283.497 U.S. 261 (1990).

284.*Id.* at 279 n.7.

285.*Id.* at 278.

286.*Id.* at 279.

287.*Id.* at 278 (citing Washington v. Harper, 494 U.S. 210, 229 (1990).

288.497 U.S. at 279, (citing Youngberg v. Romeo, 457 U.S. 307 (1982).

289.See Roe v. Wade, 410 U.S. at 155.

290.See, e.g. Carey v. Population Services International, 431 U.S. 678, 690 (1977) (holding that a statute restricting access to nonhazardous contraceptives had no relation to any compelling state interest in protecting public health).back 290back 290

291.197 U.S. 11 (1905).

292.See supra note 29 and accompanying text.

293.See generally National Primary Drinking Water Regulations; Fluoride, 50 Fed. Reg. 47,142 - 47,155 (codified at 40 C.F.R. § 141.51).

294.494 U.S. 210 (1990).

295.*Id.* at 229.

296.Cruzan v. Director, Missouri Dep't of Health, 497 U.S. at 279.back 296back 296

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*This Comment is dedicated to my dad, Ralph F. Balog, who has opposed fluoridation of public water systems since the early 1960s. The author received a B.S. from Parks College of St. Louis University in 1985, an M.S. from Embry- Riddle Aeronautical University in 1990, and a J.D. from Pace University School of Law in 1997. Thanks to Andrea Herbst and her group for their excellent editing job.

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Black, Tobie

From: Mara Brooks [mbrooks@wda.org]
Sent: Tuesday, May 15, 2012 12:57 PM
To: Bohl, James; Mark Paget; william.lobb@marquette.edu
Cc: Peterson, Todd; Owczarski, Jim; Black, Tobie
Subject: RE: Apprising you of legislation coming forward

Alderman Bohl:
On behalf of the Wisconsin Dental Association, I'd like to thank you for the advance notice of the hearing you have requested on the issue of community water fluoridation in Milwaukee. It is probably no surprise to you when I say that the WDA and its member dentists remain supportive of oral health benefits that are achieved due to community water fluoridation and we look forward to the opportunity to have someone testify on our behalf. Thanks again for the courtesy of the advance notification and please let me know if you would like to meet with the WDA prior to the hearing to discuss this further.
Sincerely,
Mara Brooks

Mara Brooks
Director of Government Services
Wisconsin Dental Association
10 East Doty Street, Suite 509
Madison, WI 53703
(608)250-3442 (p)
(608)282-7716 (f)

From: Bohl, James [mailto:jbohl@milwaukee.gov]
Sent: Monday, May 14, 2012 4:13 PM
To: Mark Paget; Mara Brooks; william.lobb@marquette.edu
Cc: Peterson, Todd; Owczarski, Jim; Black, Tobie
Subject: Apprising you of legislation coming forward

Dean Lobb, Mr. Paget and Ms. Brooks,

I'd like to take this opportunity to inform you of legislation I am introducing before the Milwaukee Common Council which seeks the elimination of forced fluoridation of the city of Milwaukee's municipal water. A public hearing on this matter will take place at the Common Council's Steering and Rules Committee on Thursday, May 31, at 1:30pm, at Milwaukee City Hall, 200 E. Wells St. You and representatives of your organization are welcome to attend this hearing. In addition to this courtesy notice, I will request both the Wisconsin Dental Association and Marquette Dental School be added to the formal agenda notice on this matter by the City Clerk's Office.

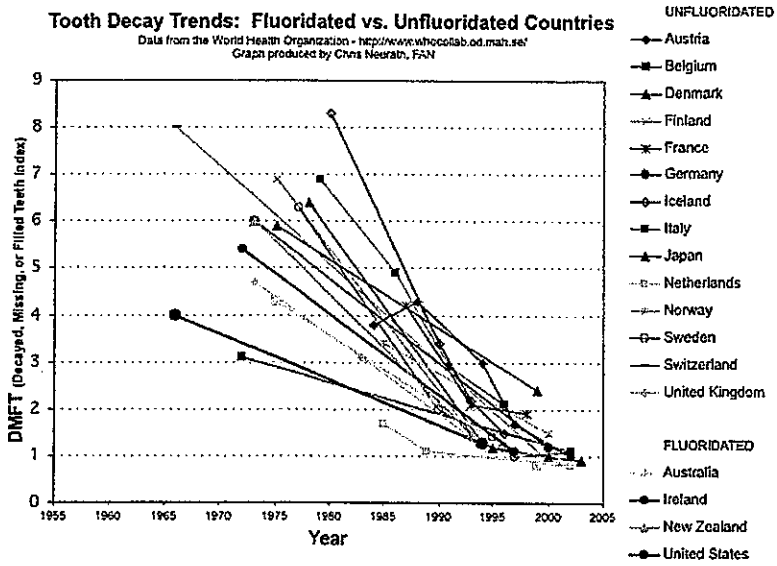
If you are aware of other persons or organizations that should be notified of the hearing, I would ask that you provide information for these individuals/groups to City Clerk Staff Assistant Tobie Black. Ms. Black can be reached at tblack@milwaukee.gov or phone number 414-286-2231.

If you have any further questions or wish to discuss the matter further please do not hesitate to

FLUORIDE ACTION NETWORK GET THE REAL FACTS ON FLUORIDE.

Tooth Decay Trends in Fluoridated VS Unfluoridated Countries

World Health Organization Data (2004) -
Tooth Decay Trends (12 year olds) in Fluoridated vs. Unfluoridated Countries: (back to top)



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DMFT (Decayed, Missing & Filled teeth) Status for 12 year olds by Country - World Health Organization Data (2004) -			
Country	**DMFTs**	**Year**	**Status***
Germany	0.7	2005	No water fluoridation, but salt fluoridation is common
Australia	0.8	1999	More than 50% of water is fluoridated; no salt fluoridation
Denmark	0.8	2006	No water fluoridation or salt fluoridation
Netherlands	0.8	2002	No water fluoridation or salt fluoridation
Zurich, Switzerland	0.9	2000	No water fluoridation, but salt fluoridation is common
UK *(England & Wales)*	0.9	2000	11% of water supplies are fluoridated; no salt fluoridation
Austria	1.0	2002	No water fluoridation, but salt fluoridation is available to a limited extent.
Sweden	1.0	2005	No water fluoridation or salt fluoridation
Italy	1.1	2004	No water fluoridation or salt fluoridation
Belgium	1.1	2002	No water fluoridation or salt fluoridation
Ireland	1.1	1997	More than 50% of water is fluoridated; no salt fluoridation
Finland	1.2	2006	No water fluoridation or salt fluoridation
US	1.28	1992-1994	More than 50% of water is fluoridated; no salt fluoridation
Iceland	1.4	2005	No water fluoridation or salt fluoridation
New Zealand	1.7	2005	More than 50% of water is fluoridated; no salt fluoridation
Norway	1.7	2004	No water fluoridation or salt fluoridation
France	1.9	1998	No water fluoridation, but salt fluoridation is common
Data from WHO Oral Health Country/Area Profile Programme Department of Noncommunicable Diseases Surveillance/Oral Health WHO Collaborating Centre, Malmö University, Sweden http://www.whocollab.od.mah.se/euro.html			

Excerpts from the Scientific Literature -
"Universal Decline in Tooth Decay" in Western World Irrespective of Water Fluoridation: (back to top)

"Although the prevalence of caries varies between countries, levels everywhere have fallen greatly in the past three decades, and national rates of caries are now universally low. This trend has occurred regardless of the concentration of fluoride in water or the use of fluoridated salt, and it probably reflects use of fluoridated toothpastes and other factors, including perhaps aspects of nutrition."
SOURCE: Cheng KK, et al. (2007). Adding fluoride to water supplies. *British Medical Journal* 335(7622):699-702.

"In most European countries, where community water fluoridation has never been adopted, a substantial decline in caries prevalence has been reported in the last decades, with reductions in lifetime caries experience exceeding 75%."
SOURCE: Pizzo G, et al. (2007). Community water fluoridation and caries prevention: a critical review. *Clinical Oral Investigations* 11(3):189-93.

"Graphs of tooth decay trends for 12 year olds in 24 countries, prepared using the most recent World Health Organization data, show that the decline in dental decay in recent decades has been comparable in 16 nonfluoridated countries and 8 fluoridated countries which met the inclusion criteria of having (i) a mean annual per capita income in the year 2000 of US$10,000 or more, (ii) a population in the year 2000 of greater than 3 million, and (iii) suitable WHO caries data available. The WHO data do not support fluoridation as being a reason for the decline in dental decay in 12 year olds that has been occurring in recent decades."
SOURCE: Neurath C. (2005). Tooth decay trends for 12 year olds in nonfluoridated and fluoridated countries. *Fluoride* 38:324-325.

"It is remarkable... that the dramatic decline in dental caries which we have witnessed in many different parts of the world has occurred without the dental profession being fully able to explain the relative role of fluoride in this intriguing process. It is a common belief that the wide distribution of fluoride from toothpastes may be a major explanation, but serious attempts to assess the role of fluoridated toothpastes have been able to attribute, at best, about 40-50% of the caries reduction to these fluoride products. This is not surprising, if one takes into account the fact that dental caries is not the result of fluoride deficiency."
SOURCE: Aoba T, Fejerskov O. (2002). Dental fluorosis: chemistry and biology. *Critical Review of Oral Biology and Medicine* 13: 155-70.

"A very marked decline in caries prevalence [in Europe] was seen in children and adolescents...The number of edentulous adults in Europe has also been declining considerably."
SOURCE: Reich E. (2001). Trends in caries and periodontal health epidemiology in Europe. *International Dentistry Journal* 51(6 Suppl 1):392-8.

"The caries attack rate in industrialized countries, including the United States and Canada, has decreased dramatically over the past 40 years."
SOURCE: Fomon SJ, Ekstrand J, Ziegler EE. (2000). Fluoride intake and prevalence of dental fluorosis: trends in fluoride intake with special attention to infants. *Journal of Public Health Dentistry* 60: 131-9.

"Since the 1960s and 70s, however, a continuous reduction (in tooth decay) has taken place in most 'westernized' countries, it is no longer unusual to be caries-free... During the decades of caries decline, a number of actions have been taken to control the disease, and the literature describes numerous studies where one or several factors have been evaluated for their impact. Still, it is difficult to get a full picture of what has happened, as the background is so complex and because so many factors may have been involved both directly and indirectly. In fact, no single experimental study has addressed the issue of the relative impact of all possible factors, and it is unlikely that such a study can ever be performed."
SOURCE: Bratthall D, Hansel-Petersson G, Sundberg H. (1996). Reasons for the caries decline: what do the experts believe? *European Journal of Oral Science* 104:416-22.

"Caries prevalence data from recent studies in all European countries showed a general trend towards a further decline for children and adolescents...The available data on the use of toothbrushes, fluorides and other pertinent items provided few clues as to the causes of the decline in caries prevalence."
SOURCE: Marthaler TM, O'Mullane DM, Vrbic V. (1996). The prevalence of dental caries in Europe 1990-1995. ORCA Saturday afternoon symposium 1995. *Caries Research* 30: 237-55

"The aim of this paper is to review publications discussing the declining prevalence of dental caries in the industrialized countries during the past decades...[T]here is a general agreement that a marked reduction in caries prevalence has occurred among children in most of the developed countries in recent decades."
SOURCE: Petersson GH, Bratthall D. (1996). The caries decline: a review of reviews. *European Journal of Oral Science* 104: 436-43.

"The regular use of fluoridated toothpastes has been ascribed a major role in the observed decline in caries prevalence in industrialized countries during the last 20 to 25 years, but only indirect evidence supports this claim."
SOURCE: Haugejorden O. (1996). Using the DMF gender difference to assess the "major" role of fluoride toothpastes in the caries decline in industrialized countries: a meta-analysis. *Community Dentistry and Oral Epidemiology* 24: 369-75.

"The marked caries reduction in many countries over the last two decades is thought to be mainly the result of the widespread and frequent use of fluoride-containing toothpaste... There seem to be no other factors which can explain the decline in dental caries, which has occurred worldwide during the same period, in geographic regions as far apart as the Scandinavian countries and Australia/New Zealand."
SOURCE: Rolla G, Ekstrand J. (1996). *Fluoride in Oral Fluids and Dental Plaque.* In: Fejerskov O, Ekstrand J, Burt B, Eds. Fluoride in Dentistry, 2nd Edition. Munksgaard, Denmark. p 215.

"Although difficult to prove, it is reasonable to assume that a good part of the decline in dental caries over recent years in most industrialized countries, notably those Northern European countries without water fluoridation, can be explained by the widespread use of fluoride toothpastes. This reduction in caries has not been paralleled by a reduction in sugar intake..."

SOURCE: Clarkson BH, Fejerskov O, Ekstrand J, Burt BA. (1996). *Rational Use of Fluoride in Caries Control.* In: Fejerskov O, Ekstrand J, Burt B, Eds. Fluoride in Dentistry, 2nd Edition. Munksgaard, Denmark. p 354.

"During the past 40 years dental caries h as been declining in the US, as well as in most other developed nations of the world... The decline in dental caries has occurred both in fluoride and in fluoride-deficient communities, lending further credence to the notion that modes other than water fluoridation, especially dentrifices, have made a major contribution."
SOURCE: Leverett DH. (1991). Appropriate uses of systemic fluoride: considerations for the '90s. *Journal of Public Health Dentistry* 51: 42-7.

"In most European countries, the 12-year-old DMFT index is now relatively low as compared with figures from 1970-1974. WHO (World Health Organization) data relating to availability of fluoride in water and toothpaste appear reliable. However, these data did not explain differences between countries with respect to the DMFT index of 12-year-olds."
SOURCE: Kalsbeek H, Verrips GH. (1990). Dental caries prevalence and the use of fluorides in different European countries. *Journal of Dental Research* 69(Spec Iss): 728-32.

"The most striking feature of some industrialized countries is a dramatic reduction of the prevalence of dental caries among school-aged children."
SOURCE: Binus W, Lowinger K, Walther G. (1989). [Caries decline and changing pattern of dental therapy] [Article in German] *Stomatol DDR* 39: 322-6.

"The current reported decline in caries tooth decay in the US and other Western industrialized countries has been observed in both fluoridated and nonfluoridated communities, with percentage reductions in each community apparently about the same."
SOURCE: Heifetz SB, et al. (1988). Prevalence of dental caries and dental fluorosis in areas with optimal and above-optimal water-fluoride concentrations: a 5-year follow-up survey. *Journal of the American Dental Association* 116: 490-5.

"[D]uring the period 1979-81, especially in western Europe where there is little fluoridation, a number of dental examinations were made and compared with surveys carried out a decade or so before. It soon became clear that large reductions in caries had been occurring in unfluoridated areas. The magnitudes of these reductions are generally comparable with those observed in fluoridated areas over similar periods of time."
SOURCE: Diesendorf, D. (1986). The Mystery of Declining Tooth Decay. *Nature* 322: 125-129.

"Even the most cursory review of the dental literature since 1978 reveals a wealth of data documenting a secular, or long term, generalized decline in dental caries throughout the Western, industrialized world. Reports indicate that this decline has occurred in both fluoridated and fluoride-deficient areas, and in the presence and absence of organized preventive programs."
SOURCE: Bohannan HM, et al. (1985). Effect of secular decline on the evaluation of preventive dentistry demonstrations. *Journal of Public Health Dentistry* 45: 83-89.

"The decline in caries prevalence in communities without fluoridated water in various countries is well documented. The cause or causes are, at this time, a matter of speculation."
SOURCE: Leverett DH. (1982). Fluorides and the changing prevalence of dental caries. *Science* 217: 26-30.

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Excerpts from the Scientific Literature - Tooth Decay Trends in Western European Countries: (back to top)

BELGIUM - Unfluoridated Water, Fluoridated Salt: (back to top)

"Caries-free children increased from 4% to 50%...A remarkable decline in dental caries was observed during the 15-yr period."
SOURCEL Carvalho JC, Van Nieuwenhuysen JP, D'Hoore W. (2001). The decline in dental caries among Belgian children between 1983 and 1998. *Community Dentistry and Oral Epidemiology* 29: 55-61.

DENMARK - Unfluoridated Water, Unfluoridated Salt:

"The paper presents an overview of the oral health situation in Denmark...[N]ational oral epidemiological data have been provided since 1972. Partly due to the preventive approach, a general decrease over-time in the prevalence of dental caries has been documented for children and adolescents. For example, in 1972 children in first class had a mean caries experience of 12.4 def-s against 3.9 def-s in 1990."
SOURCE: Petersen PE. (1992). Effectiveness of oral health care--some Danish experiences. *Proceedings of the Finnish Dental Society* 88: 13-23.

FINLAND - Unfluoridated Water, Unfluoridated Salt:

"During the 10 years, substantial decreases were seen in the mean numbers of dental visits (from 4.0 to 2.4) and fillings (from 2.9 to 1.2). The greatest decrease was seen in the number of fillings made in incisors."
SOURCE: Vehkalahti M, Rytomaa I, Helminen S. (1991). Decline in dental caries and public oral health care of adolescents. *Acta Odontologica Scandinavica* 49: 323-8.

FRANCE - Unfluoridated Water, Fluoridated Salt:

"Epidemiological surveys showed a marked decrease of caries prevalence in French children during the last 20 years."
SOURCE: Obry-Musset AM. (1998). [Epidemiology of dental caries in children] [Article in French] *Arch Pediatr* 5: 1145-8.

GERMANY - Unfluoridated Water, Fluoridated Salt:

"Caries rates are on the decline in the Federal Republic of Germany, too. And, in some cases considerable, increase in the number of children with caries-free teeth and a clear reduction in the average number of carious teeth has been recorded, above all in kindergartens with preventive dentistry programmes."
SOURCE: Gulzow HJ. (1990). [Preventive dentistry in the Federal Republic of Germany] [Article in German] *Oralprophylaxe* 12: 53-60.

GREECE - Unfluoridated Water, Unfluoridated Salt:

"The percentage of caries-free children for the total examined population increased by 94% while the reduction in DMFT index ranged between 38 and 70%. Treatment need was significantly lower in 1991 compared to 1982 in both dentitions."
SOURCE: Athanassouli I, et al. (1994). Dental caries changes between 1982 and 1991 in children aged 6-12 in Athens, Greece. *Caries Research* 28(5):378-82.

ICELAND - Unfluoridated Water, Unfluoridated Salt:

"During the last decade, a continuous decrease in dental caries has been observed among schoolchildren in Iceland...There does not seem to be any single factor responsible for the onset of the caries decline."
SOURCE: Einarsdottir KG, Bratthall D. (1996). Restoring oral health: On the rise and fall of dental caries in Iceland. *European Journal of Oral Science* 104: 459-69.

THE NETHERLANDS - Unfluoridated Water, Unfluoridated Salt:

"According to WHO criteria, 12-year-old children in The Netherlands now have a very low caries experience."
SOURCE: Truin GJ, Konig KG, Bronkhorst EM. (1994). Caries prevalence in Belgium and The Netherlands. *International Dentistry Journal* 44: 379-8.

NORWAY & all SCANDINAVIAN COUNTRIES - Unfluoridated Water, Unfluoridated Salt:

"Denmark, Iceland, Norway, and Sweden have all had a similar decline in dental caries during the last 20 years, although the decline has come later in Iceland. Despite the differences in choice of preventive methods, the dental health of children varies little across the frontiers."
SOURCE: Kallestal C, et al. (1999). Caries-preventive methods used for children and adolescents in Denmark, Iceland, Norway and Sweden. *Community Dentistry and Oral Epidemiology* 27: 144-51.

"Despite differences in the dental health care services and the recording and reporting systems, a consistent and similar decline in dental caries is evident for Denmark, Finland, Norway and Sweden during the last two decades."
SOURCE: von der Fehr FR. (1994). Caries prevalence in the Nordic countries. *International Dentistry Journal* 44: 371-8.

SWEDEN - Unfluoridated Water, Unfluoridated Salt:

"Between 1967 and 1992 the mean dmfs values declined from 7.8 to 1.8. The decline was greatest between 1967 and 1980 and then levelled off."
SOURCE: Stecksen-Blicks C, Holm AK. (1995). Dental caries, tooth trauma, malocclusion, fluoride usage, toothbrushing and dietary habits in 4-year-old Swedish children: changes between 1967 and 1992. *International Journal of Paediatric Dentistry* 5: 143-8

SWITZERLAND - Unfluoridated Water, Fluoridated Salt:

"Caries prevalence has declined by 70-84 percent since the late sixties."
SOURCE: Marthaler TM. (1991). [School dentistry in Zurich Canton: changes as a result of caries reduction of 80 to 85 percent] [Article in German] *Oralprophylaxe* 13: 115-22.

"Surveys of dental caries prevalence were carried out from 1970-1993 in schoolchildren of the city of Zurich. DMFT experience declined by 68 to 80%, while the average dmft decreased by 48-53% (ages 7 to 9)."
SOURCE: Steiner M, Menghini G, Curilovic Z, Marthaler T. (1994). [The caries occurrence in schoolchildren of the city of

Zurich in 1970-1993. A view of prevention in new immigrants] [Article in German]. *Schweiz Monatsschr Zahnmed* 104: 1210-8.

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Fluoride Dangers

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EXCITOTOXICITY: A POSSIBLE CENTRAL MECHANISM IN FLUORIDE NEUROTOXICITY

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SUMMARY: Recent evidence indicates that fluoride produces neuronal destruction and synaptic injury by a mechanism that involves free radical production and lipid peroxidation. For a number of pathological disorders of the central nervous system (CNS), excitotoxiclty plays a critical role. Various studies have shown that many of the neurotoxic metals, such as mercury, lead, aluminum, and iron also injure neural elements in the CNS by an excitotoxic mechanism. Free radical generation and lipid peroxidation, especially in the face of hypomagnesemia and low neuronal energy production, also magnify excitotoxic sensitivity of neurons and their elements. This paper reviews briefly some of the studies that point to a common mechanism for the CNS neurotoxic effects of fluoride and calls for research directed toward further elucidation of this mechanism.

Keywords: Aspartate; Excitotoxicity; Fluoride neurotoxicity; Fluoroaluminum complexes; Glutamate; 4-Hydroxynonenal; Melatonin; Neurodegeneration; Peroxynitrite; Reactive nitrogen species; Reactive oxygen species.

INTRODUCTION

Compelling evidence indicates that fluoride produces injury to the central nervous system (CNS) by several mechanisms. Of particular interest is the ability of fluoride to induce free radical generation and lipid peroxidation in the brain, especially in the hippocampus. In addition, fluoride enhances aluminum absorption from the gastrointestinal mucosa and across the blood-brain barrier. Of particular concern is the recent demonstration that fluoride readily forms a chemical complex with aluminum, similar to the phosphate ion, which is toxic to neurons at low concentrations and can act as an activator of G-proteins, a membrane link to second messenger activation.

While it appears that the toxicity of fluoride is secondary to many widely divergent and unrelated processes, there is compelling evidence that a central mechanism may be involved called excitotoxicity (Figure and Table).

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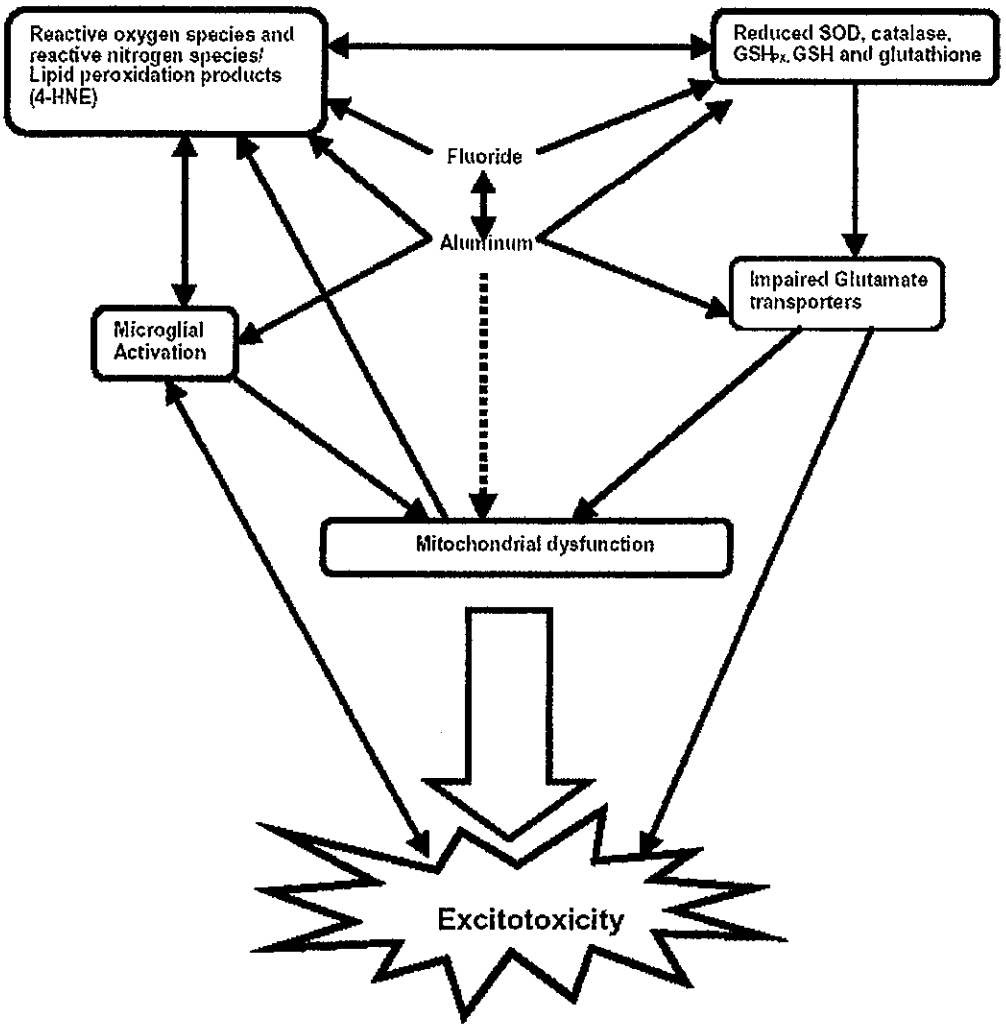

Figure. Possible mechanisms for neurodegenerative effects of fluoride and aluminum as related to excitotoxicity. The broken arrow represents the effects of both elements.

WHAT IS EXCITOTOXICITY?

Excitotoxicity is a common mechanism seen in many neurological disorders, including strokes, brain trauma, CNS infections, autoimmune disorders, multiple sclerosis, heavy metal toxicity, brain tumors, and the majority of neurodegenerative diseases, such as Alzheimer's dementia, Parkinson's disease, and Lou Gehrig's disease (amyotrophic lateral sclerosis, ALS).[1] In a recent series of papers, I argue that excitotoxicity is also the central mechanism of autism and the Gulf War Syndrome.[2-4]

Table. Comparison of the effects of fluoride/aluminium and excitotoxicity

	Fluoride/Aluminium	Excitotoxicity
Increased brain reactive oxygen species (ROS) and reactive nitrogen species (RNS)	yes	yes
Increased lipid peroxidation (LPO)	yes	yes
Decreased glutathione	yes	yes
Decreased superoxide dismutase (SOD)	yes	yes
Elevated brain ascorbate	yes	yes
Hippocampal apoptosis necrosis	yes	yes
G-protein activation	yes	yes
Synaptic injury	yes	yes
Impaired glutamate uptake	yes	yes
Microglial activation	? for fluoride yes for aluminium	yes
ROS in other tissues	? for fluoride yes for aluminium	yes
DNA injury	yes	yes

The process involves accumulation of acidic amino acids in the synaptic cleft for a prolonged period. These special amino acids include cysteine, cysteine sulfinic acid, cysteic acid, and homocysteine, as well as the neurotransmitters glutamate and aspartate. The neurotransmitters glutamate and aspartate normally activate a series of glutamate receptors on the postsynaptic membrane that leads to neuronal excitation. In fact, glutamate is the most abundant neurotransmitter in the CNS and is responsible for attention, alertness, and learning. It is also the most neurotoxic.

If the excitatory amino acids are not removed quickly from the synaptic cleft, the postsynaptic neurons become overstimulated, leading to either synaptic destruction and dendritic retraction or, should the stimulation be prolonged and intense, neuronal destruction by both apoptosis and necrosis.[5] It is for these reasons that extracellular glutamate levels are carefully regulated by a series of glutamate transporters, which remove the glutamate for storage, either in the presynaptic neuron terminal or surrounding astrocytes (glia).[6]

This excitotoxic process was originally discovered by two ophthalmologists, Lucas and Newhouse in 1957[7] and given the name excitotoxicity by Dr John Olney in 1969.[8] Since its discovery, a great deal has been learned about the mechanism of excitotoxicity, the receptors involved, and the glutamate uptake system. In addition, much has been discovered about other toxins that can activate this destructive process. Recently, glutamate receptors have been found in numerous peripheral tissues, including the testes, lungs, pancreatic islet cells, cardiac nerves, ovaries, endothelial cells, immune cells, and bone osteoblasts.[9]

COMMON MECHANISMS

1.Free radical generation

Glutamate receptors are found in numerous types of neurons, including those that utilize other neurotransmitters, such as GABA (gamma-aminobutyric acid), dopamine, norepinephrine, and serotonin.[10] There are two basic types of glutamate receptors, ion-gated channels (ionotrophic) and metabotropic receptors.[11] Three ionotrophic receptor types have been identified, based on their affinity for selective agonists. These include N-methyl-D-aspartate (NMDA), alpha-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid (AMPA), and kainate receptors. Neurons frequently contain more than one of these receptors types on the synaptic membranes.

The ionotrophic receptors control the passage of sodium, potassium, and calcium through membrane channels, which in turn initiates neuronal depolarization (excitation). Most important to the excitotoxic process is calcium accumulation within the cytosol following glutamate receptor activation. Intracellular calcium triggers numerous cellular reactions including the activation of nitric oxide synthase and protein kinase C.[12] These in turn can activate free radical generation and lipid peroxidation as well as eicosanoid activation, should glutamate persist too long in its receptor.[13] These processes play a major role in excitotoxic injury and neuronal death.

Three types of metabotropic receptors and eight subtypes of these receptors have been identified through cloning techniques. They operate mainly by GTP (guanine triphosphate) binding proteins or G-proteins. [14] When these receptors are stimulated by glutamate, the G-protein within the cell membrane is activated, which in turn activates several second messengers within the neuron, including IP3 (inositol 1,4,5-trisphosphate), cAMP (cyclic adenine monophosphate), or cGMP (cyclic guanine monophosphate). There is also evidence that they regulate intracellular calcium.[16] Two of the metabotropic receptors are thought to be neuroprotective and one is capable of triggering excitotoxicity.

Free radicals and lipid peroxidation products generated by excitotoxicity have been shown to damage dendrites and synaptic connections, and, if unrelieved, lead to neuronal destruction.[16] Likewise, free radicals caused by other processes have been shown to trigger excitotoxicity by impairing glutamate removal and by activating microglia, which contain abundant stores of glutamate.[17]

It has also been shown that one of the lipid peroxidation products, 4-hydroxynonenal (4-HNE), specifically impairs synaptic function and inhibits glutamate removal by the glutamate transport proteins.[18] This lipid peroxidation product, though less abundant than malondialdehyde, is significantly more neurotoxic. Any process that precipitates lipid peroxidation also precipitates the production of 4-HNE. Therefore, even if fluoride does not directly trigger excitotoxicity, it will do so indirectly by impairing glutamate removal and by generating reactive oxygen intermediates and lipid peroxidation products.

A study from China found that sodium fluoride significantly increased nitric oxide synthase (NOS) activity.[19] Interestingly, excitotoxins also stimulated NOS activity, which increases intracellular nitric oxide (NO) content. This is of particular importance because NO combines readily with superoxide forming the very powerfully toxic peroxynitrite radical, which plays a major role in all neurodegenerative diseases, primarily by damaging mitochondrial energy production, inhibiting glutamate re-uptake, and stimulating lipid peroxidation.[20-21] Fluoride has also been shown to inhibit superoxide dismutase, which would increase intracellular levels of the superoxide radical, the substrate for peroxynitrite formation.[22]

Another related neurotoxin, aluminum, is known to produce a dramatic increase in brain free radical generation and lipid peroxidation both directly and by increasing neuronal and glial iron levels.[23] In addition, melanin has a high affinity for aluminum, making neuromelanin-containing neurons in the *substantia nigra pars compacta* significantly more vulnerable to free radical and lipid peroxidation injury.[24] Aluminum accumulation and focal increases in reactive oxygen species and lipid peroxidation in this nucleus have been demonstrated in Parkinson's disease.[25]

Another mechanism by which fluoride might increase brain free radical generation and lipid peroxidation would be through activation of protein kinase C by a fluoroaluminum complex. It is known that a major mechanism by which glutamate induces excitotoxicity is activation of protein kinase C. Blocking this enzyme affords significant protection against excitotoxicity. Lead dramatically increases protein kinase C activity in a manner similar to glutamate, thereby triggering excitotoxicity.[26] Fluoride, in the form of silicofluorides in drinking water has been found to increase blood lead levels significantly, indicating an indirect connection between fluoride, free radical generation, and excitotoxicity.[27]

Because of the intimate connection between excitotoxicity, free radical generation, and lipid peroxidation, one can safely assume that fluoride can at least initiate the process indirectly and because of chronic exposure seen with water fluoridation, one would expect an eventual increase in neurodegeneration-associate disorders such as Alzheimer's dementia, ALS, and Parkinson's disease.

2. Inhibition of antioxidant enzymes

Closely connected with excitotoxicity-precipitated free radical generation and lipid peroxidation is the eventual depletion of antioxidant defenses. Several stud-

ies have demonstrated that fluoride toxicity, as well as excitotoxic injury, is associated with selective antioxidant depletion.[28-30]

Fluoride has been shown to inhibit certain antioxidant enzymes and molecules, such as superoxide dismutase (SOD), glutathione reductase, glutathione peroxidase, catalase, and glutathione.[31] This would not only increase free radical injury but would also enhance excitotoxicity, since reactive oxygen species as well as nitrogen species and lipid peroxidation products can trigger the excitotoxic process.[32] Antioxidant enzyme inhibition would necessarily enhance the toxicity of other neurotoxic elements, pesticides, herbicides, and environmental pollutants.

Another mechanism for magnifying the harmful effects of both fluoride and excitotoxins on the brain would be inhibition of melatonin. Melatonin, a hormone produced by the pineal gland, has been shown to have powerful neutralizing effects on free radicals and lipid peroxidation and to increase the levels of several of the antioxidant enzymes in the brain including SOD, glutathione reductase, glutathione peroxidase, catalase, and glutathione itself.[33]

A recent study has shown that fluoride significantly inhibits the release of melatonin from the pineal gland and that fluoride accumulates in the gland in very large concentrations in individuals drinking fluoridated water.[34] Ironically, glutamate and aspartate also powerfully inhibit melatonin release from the pineal gland and do so by a metabotropic receptor.[35] Conceivably, fluoride inhibits release of pineal melatonin by elevating glutamate levels. Since no research has been reported looking for this connection we do not know.

A recent study revealed that babies with the lowest melatonin production had the most neurobehavioral problems.[36] Melatonin levels are also lower in the cerebrospinal fluid (CSF) of Alzheimer's patients as compared with normal individuals.[37] The fact that fluoride lowers melatonin production would indicate that risk of neurodegeneration in both instances would be elevated.[38]

3. Inhibition of mitochondrial energy enzymes

Another connection between glutamate excitotoxicity and fluoride toxicity is related to inhibition of brain energy production. Several studies have shown that anything which suppresses neuronal energy production, especially mitochondrial energy production, greatly enhances excitotoxic sensitivity.[39-41] In fact, when neuronal energy production is low, even physiological levels of excitotoxins such as glutamate can trigger excitotoxicity.

Fluoride is also known to inhibit cellular energy producing enzymes, including mitochondrial electron transport enzymes. It does this both directly, as in the case of glycolytic and Kreb's cycle enzymes,[42] and indirectly, as in the case of the mitochondrial enzymes by the effect of peroxynitrite.[43] Vani and Reddy demonstrated suppression of both antioxidant enzymes and energy generating enzymes in female mice treated with 20 mg of fluoride/kg bw for 14 days.[22]

The importance of neuronal energy suppression by fluoride lies in the fact that that mitochondrial energy suppression is intimately connected as an early event to neurodegenerative diseases such as Alzheimer's dementia and Parkinson's dis-

ease.[44-46] Since fluoride can inhibit these enzymes, even in low concentrations, there is an increased likelihood that excitotoxicity plays a significant role in this process. Likewise, it should be appreciated that Mullenix *et al* have shown that fluoride accumulates in various brain areas of the rat, particularly the hippocampus, resulting in higher fluoride levels in the brain than are seen in the blood.[47] The hippocampus is one of the most sensitive areas of the brain to a multitude of neurotoxic events.

4. Inhibition of glutamate transporters

One of the most important ways glutamate concentrations are controlled in the nervous system is by a series of glutamate transport proteins. Thus far, five such transporters have been demonstrated by cloning techniques.[48] Of particular importance are GLAST (cloned glutamate/aspartate transporter) and GLT-1 (glutamate transporter-1). These transporters are associated with either the glial cells or the neurons themselves. The glial transporters (GLAST and GLT-1) bind to synaptically released glutamate and transport it to the interior of the glial cells. The neuronal transporters bind the glutamate and transfer it to the interior of the presynaptic terminal.

Considerable evidence points to impairment of these transporters as major players in neurodevelopmental disorders and neurodegenerative diseases.[49] The function of these transporters is altered by a number of commonly encountered toxins including mercury,[50] aluminum,[51] iron,[52] cytokines,[53] eicosanoids (PGE2),[54] and 4-HNE.[55] In fact, mercury has been shown to inhibit the glutamate transporters at concentrations below those that are cytotoxic.[56] Anything that increases free radical generation and lipid peroxidation impairs glutamate transport.

Aluminum inhibition of glutamate transporters is of special interest because of the frequent and ready interaction of aluminum and fluoride to form a biologically reactive complex. Although no one has apparently examined the occurrence of fluoride-aluminum complexes as the common inhibitor involved, the possibility is quite high. This is because of the chemical avidity of fluoride for aluminum and the fact they frequently occur together in nature.

Even without the direct involvement of a fluoroaluminum complex, the fact that fluoride is known to cause a seven-fold increase in the absorption of aluminum past gut barriers is of significant concern.[57] In addition, fluoride enhances the passage across the blood-brain barrier. In several studies, fluoride added to drinking water doubled brain aluminum levels, thus increasing the likelihood of glutamate transporter inhibition.[58,59]

Aluminum glutamate, which is formed in the GI tract, has been shown to alter the blood-brain barrier making it more permeable to normally excluded toxins.[60] In addition, it enhanced both aluminum and glutamate concentrations in the brain, significantly increasing the risk of excitotoxicity.

THE ALUMINUM-FLUORIDE CONNECTION

As mentioned in the introduction, aluminum interacts with fluoride to form a fluoroaluminum complex that mimics phosphate groups in biological systems.[61] By this mechanism, it could also activate the G-proteins in cell membranes. As we have seen, the metabotropic receptors are activated by a G-protein mechanism. In addition, numerous cells in the body utilize the G-protein second messenger receptor system, including endothelial cells, lymphocytes, osteoblasts, other neurotransmitters (dopamine, norepinephrine, acetylcholine, serotonin, neuropeptides, and opioids), and glucagon.

Activation of metabotropic excitatory receptors by an aluminum-fluoride complex could initiate excitotoxicity as shown by Lan and coworkers.[62] Because the aluminum-fluoride complex accumulates in the brain, it would also be expected to cause prolonged neurotoxicity, leading eventually to neurodegeneration and synaptic loss.

The aluminum-fluoride complex has been shown to produce neuronal loss in the CA1 and CA-4 areas of the hippocampus when given to animals as 0.5 ppm in drinking water.[59] The toxic effect may be related to a combination of effects, including impairment of energy-producing enzymes, impaired dephosphorylation of hyperphosphorylated tau-protein, increased neuronal iron concentration, elevated free radical and lipid peroxidation levels, and impaired DNA repair, all of which are related to excitotoxicity.

Another toxic effect of aluminum, and possibly a fluoroaluminum complex, is the activation of microglia. These are resident immune cells within the nervous system, which are normally quiescent, but are easily activated by a number of environmental and biological agents, such as viruses, mycoplasma, bacteria, aluminum, mercury, and several pesticides.[63]

Once activated, microglia generate and secrete a number of neurotoxic compounds, including two powerful excitotoxins: glutamate and quinolinic acid.[64] The combination of excitotoxin secretion and cytokine production greatly increases the concentration of free radicals and lipid peroxidation products in the brain. No one has looked at the possibility of fluoride-induced microglial activation. Yet, one would expect the fluoroaluminum complex to activate microglia, since aluminum alone is a powerful activator.[65]

Chronic microglial activation has been associated with a number of neurodegenerative processes, including strokes, multiple sclerosis, brain trauma, experimental allergic encephalomyelitis (EAE), Alzheimer's dementia, Parkinson's disease, and ALS.[3] Because both aluminum and fluoride accumulate in the brain and have their highest concentrations in the hippocampus and neocortex, one would expect chronic microglial activation as well. At least one study noted reactive gliosis (microglial activation) in association with fluoride brain toxicity.[66]

FLUORIDE: A SPECIAL DANGER TO THE DEVELOPING BRAIN

The brain undergoes one of the fastest growth and development rates of any portion of the human body during embryogenesis. This occurs especially during the last trimester and first two years of life, a period called the brain growth spurt. This involves not only the rapid development of synaptic connections (synaptogenesis) and pathway development, but also refinement of all of the synaptic connection made during this period. One way glutamate does this is by stimulating the growth cones that guide neural pathways to their intended destination. The brain develops far greater synaptic connections than are needed during this "brain growth spurt" and as a result, synaptic connections are removed in a process referred to as pruning.

Connected to this pruning process, as well as to synaptogenesis and pathway development, is the level of glutamate within the brain. The rise and fall of brain glutamate levels during development controls these processes, and is finely tuned throughout brain development.[67] Too much glutamate overprunes the synapses and dendrites, whereas too little results in an excess of un-needed connections.[68] Both can result in severe neurodevelopmental problems.

Recent studies have revealed that the glutamate transport proteins also play a significant role in the development of the brain.[69,70] As shown by these studies, anything that alters transporter function can affect brain development. By interfering with neuronal energy production, neurotransmitter levels (especially glutamate), free radical generation and growth cone function, fluoride can have significant harmful effects on neurodevelopment.

In addition, fluoride has also been found to inhibit thyroid function and thereby alter early neuron migration in the developing fetus.[71] This can result in irreversible changes in the fetal brain.

A CALL FOR FURTHER RESEARCH

It is obvious from this short review that more research needs to be done in this area. We need data on both the effects of fluoride and fluoroaluminum on the glutamate transporter proteins and on the exact mechanism of free radical generation being caused by fluoride. In addition, we need studies to see if fluoride can cause chronic microglial activation and neurodegeneration.

Because of the growing number of studies showing a strong connection between aluminum accumulation in the brain and neurodegenerative diseases, studies need to be done to see if the aluminum in neurofibrillary tangles and senile plaques is in fact fluoroaluminum. Further studies are also needed to see if fluoroaluminum passes along olfactory axons into the entorhinal area as has been demonstrated for aluminum itself.[72] This would not only provide direct access to the area of the brain showing the earliest changes of Alzheimer's dementia, but would allow lower concentrations in the drinking water to produce higher concentrations in the hippocampal area than would be attainable from blood.

In addition, special studies are needed using silicofluorides to see if their toxicity to the nervous system differs from that of sodium fluoride. Along this same line, we need data on the possibility of additive and even synergic toxicities when fluoride is combined with mercury, lead, cadmium, and other known neurotoxins.

Although progress has been made on nutrient-based neuroprotection against fluoride toxicity, more research needs to be pursued.[73-77] Chinoy and Sharma found that both vitamin E and D_3 reversed the toxic effect of fluoride on male reproductive organs and that a combination of the two antioxidants completely reversed the toxicity.[78] In a recent study, Chinoy and Shah found that a combination of vitamin C and E and calcium could reverse the toxic effects of both fluoride and arsenic on multiple biochemical parameters, including suppression of dehydroascorbic acid, glutathione, glutathione peroxidase, and SOD in the brains of mice.[79] If excitotoxicity indeed plays a significant role in fluoride toxicity, we need to apply some of the methods used to protect against excitotoxicity, such as increasing the intake of methylcobalamin, melatonin, selenium, the B vitamins, vitamins C, E, D, and K, along with metabolic stimulants such as pyruvate, malate, CoQ10, acetyl-L-carnitine, R-lipoic acid, and ginkgo biloba. Of special importance is supplementation with magnesium, which has been shown to block the NMDA glutamate receptor and decrease free radical production.

One area of particular interest is the use of flavonoids as neuroprotectants. Plant flavonoids are known to be the most versatile and powerful antioxidants known, and one of the few antioxidants that will neutralize peroxynitrite.[80] In addition, they can chelate metals, reduce inflammation, block eicosanoid production, and inhibit enzymes such as protein kinase C, which is critical to excitotoxicity and lead neurotoxicity.[81] A recent study by Juzyszyn and co-workers found that quercetin sulfonate, a water-soluble form of the flavonoid quercetin, protected liver and kidney cells from ammonium fluoride suppression of mitochondrial energy production.[82]

Finally, we need more data on the concentration and accumulation of fluoride in other calcified areas of the brain beside the pineal gland. For example, calcification of the basal ganglion is seen in a small number of individuals. In the past, this was considered an asymptomatic condition occurring in 0.3% of the population examined.[83] While basal ganglion calcification has been noted in a number of disorders, of particular interest is its appearance in Down's syndrome. One study on autopsied Down's brains found calcification in 45% in the area of the basal ganglion and increased calcification there with increasing age.[84] Newer studies have shown that a significant number of these individuals have symptoms related to basal ganglion dysfunction as well as neuropsychiatric disturbances.[85] In addition, recent studies has shown that excitotoxicity induces calcification deposits in the brain, which also contain aluminosilicates.[86] Should these calcifications accumulate fluoride in high concentrations as found in pineal calcifications, one would expect damage to adjacent neurons and glia. With widespread fluoridation of drinking water, one would also expect higher fluoride concentrations in these calcified structures than in the past.

It is obvious from this review that there is an intimate connection between the neurotoxicity of fluoride, aluminum, and glutamate that needs further attention. It is also obvious that excitotoxicity plays some role in this process, perhaps a central one.

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Building a Database of Developmental Neurotoxicants: Evidence from Human and Animal Studies

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Introduction

EPA's program for the screening and prioritization of chemicals for developmental neurotoxicity makes it essential to assemble a list of chemicals that are toxic to the developing mammalian nervous system. Listed chemicals will be used to evaluate the sensitivity, reliability, and predictive power of alternative developmental neurotoxicity assays. To establish this list, a literature review was conducted for over 400 compounds that have been suggested to be developmental neurotoxicants, neurotoxicants, or developmental toxicants. Compounds were assigned one of three groups based on the strength of the evidence for developmental neurotoxicity:

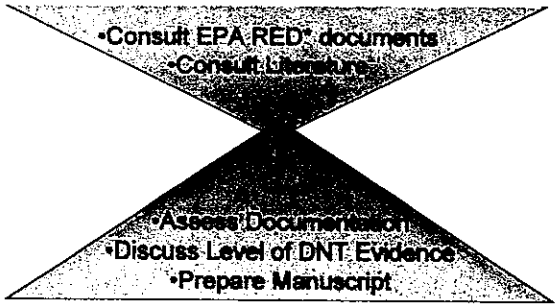
(1) no evidence: either there were no reports that met our criteria for evidence, or there were reports which showed no developmental neurotoxicity;
(2) minimal evidence: one report only or multiple reports from only one laboratory; or
(3) substantial evidence: reports from more than one laboratory.

The chemicals in the latter group will be especially useful for vetting protocols that have been proposed as screens for developmental neurotoxicity.

This presentation has been reviewed by the National Health and Environmental Effects Research Laboratory and approved. Approval does not signify that the contents reflect the views of the Agency.

Approach

Collect lists of putative DNT chemicals (n≈400)



Each chemical was assigned to one of three categories:

1. **No available evidence existed: exclude from manuscript.**
2. **Minimal evidence existed: put in table in manuscript.**
3. **Substantial evidence existed: write a descriptive paragraph for manuscript.**

*Registration Eligibility Decision Documents (available online or via Freedom of Information Act)

Evidence: Criteria for Assessment and Endpoints

a) We included only mammalian studies.
   -no *in vitro* studies were included.
b) We included only studies with the pure chemical (or reasonably so).
   -no mixture studies were included.
   -no human studies were included wherein there was exposure to more than one compound.
   -no formulations were included.
c) We included only studies where the exposure took place during pregnancy or during the period before weaning.
d) We included only studies in which the administered dose was below 5 grams/kg.
e) Where knowledge was available, we considered only studies where the administered dose would not be lethal to the offspring.
f) We did not include any case reports.
g) In studies where the chemical was administered during gestation, to the extent possible, we looked for a litter-based statistical design.
h) If only acute pharmacological effects were reported (either during dosing or shortly thereafter), we did not include that study.

Endpoints assessed included, but were not limited to:

- Head Circumference
- Brain Weight
- Exencephaly
- Brain Morphology
- Motor Activity
- Learning and Memory
- Grip Strength
- Negative Geotaxis
- Startle Response
- Righting Reflex
- Neurochemical Levels
- Receptor Affinity/Number

Chemicals with Minimal Evidence of Developmental Neurotoxicity (n≈100)

1,1,1-Trichloroethane
Abamectin
Acephate
Acetamiprid
ActinomycinD
Amicarbazone (MKH 3586)
Aminozole
Atorvastatin
Atrazine
Azinphos methyl
BAS 510 (Boscalid)
BAS 670H
Bifenthrin
Bismuth Ribromophenate
Brominated veg oil
Busulfan
Carbofuran
Carbon disulfide
Chlordane
Chlordimeform
Chlorfenapyr
Chlorite, sodium
CI-943 (Antipsychotic)
Clodinafop-propargyl
Clothianidin
Coumaphos
Cyfluthrin
Cyhalothrin
Cymoxanil
Denazol
DDT
Dexromoramide
Diaminotoluene (2,6-)
Dichloromethane (methylene chloride)
Dichlorvos (DDVP)
Dicrotophos
Difluoromethylornithine
Dimethoate
Dinoseb
Diphenhydramine
Disulfoton
Emamectin
Endosulphan
Endrin
EPTC (S-Ethyl dipropylthiocarbamate)
Ergotamine
Ethoxythanol (2-)
Ethylene dibromide
Ethylene oxide
Etofenprox
Fenamiphos
Fenitrothion
Fenvalerate
FK 33-824 (Synthetic enkephalin)
Flufenacet (thiaflumide)
Formaldehyde
Glufosinate ammonium
Glyphosate trimesium
Hexachoroplatinate (Na)
Imidacloprid
Ivermectin
Lasofoxifene
Levo-alpha-acetylmethadol
Lidocaine
Malathion
Mancozeb
Maytansine
Methamidaphos
Methyl Ethyl Ketone
MNDA
Molinate
Naled
n-Hexane
Nickel carbonyl
Perchlorate
Phorate (BAS 226 I)
Picrotoxin
Primidone
Profenofos
Prothioconazole
Selenium compounds
Simvastatin
Spirodiclofen
Succamir
Terbufos
tert-Butylhydroquinone, 2-
Tetrachloethylene
Tetracycline
Thiamethoxam
Tributos (DEF)
Triethylene glycol dimethyl ether
Trimethadione
Triphenyl phosphate
VM-26 (Teniposide)
VP-16-213 (Etoposide)

Chemicals with Substantial Evidence of Developmental Neurotoxicity (n≈100)

2-Ethoxyethyl Acetate
Acibenzolar-S -methyl
Acrylamide
Aldicarb
Allethrin
Aluminum (cl or lactate)
Amino-oleolinamide(6-)
Aminopterin
Amphetamine(d-)
Arsenic
Aspartame
Azacytidine(5-)
Benomyl
Benzene
Bisalletrin
Bis(tri-n-butyltin)oxide
Bisphenol A
Bromodeoxyuridine(5-)
Butylated Hydroxy Anisol
Butylated hydroxytoluene
Cadmium
Caffeine
Carbamazepine
Carbaryl
Carbon monoxide
Chlordecone
Chlordiazepoxide
Chlorine dioxide
Chlorpromazine
Chlorpyrifos
Cocaine
Colcemid
Colchicine
Cypermethrin
Dexamethasone
Diamorphine hydrochloride
Diazepam
Cytosine Arabinoside
DEET
Deltamethrin
Diazinon
Dieldrin
Diethylstilbestrol
Diphenylhydantoin
Epidermal Growth Factor
Ethanol
Ethylene thiourea
Flourouracil(5-)
Fluazinam
Fluoride
Griseofulvin
Haloperidol
Halothane
Heptachlor
Hexachlorobenzene
Hexachlorophene
Hydroxyurea
Iminodipropionitrile (IDPN)
Ketamine
Lead
Lindane
LSD
Maneb
Medroxyprogesterone
Mepivacaine
Methadone
Methanol
Methimazole
Methylparathion
Monosodium Glutamate
MPTP
Naloxone
Naltrexone
Nicotine
Methoxyethanol, 2-
Methylazoxymethanol
Methylmercury
Ozone
Paraquat
Parathion (ethyl)
PBDEs
PCBs (generic)
Penicillamine
Permethrin
Phenylacetate
Phenylalanine (d,l)
Phthalate, di-(2-ethylhexyl)
Propylthiouracil
Retinoids/Vit.A/Isotretinoin
Salicylate
Tebuconazole
Tellurium (salts)
Terbutaline
Thalidomide
THC
Toluene
Triamcinolone
Tributyltin chloride
Trichlorfon
Trichloroethylene
Triethylead
Triethyltin
Trimethyltin
Trypan blue
Urethane
Valproate
Vincristine

Sample Paragraph

DEXAMETHASONE
CAS Number: 50-02-2

Formula:

Dexamethasone is synthetic member of the glucocorticoid class of steroid hormones. It is used to treat inflammation and autoimmune conditions (*e.g.*, rheumatoid arthritis), and to counteract side-effects of chemotherapy in cancer patients. Synthetic glucocorticoids, including dexamethasone, are also administered to women at risk for preterm labor to advance fetal maturation and reduce neonatal morbidity and mortality.

Numerous studies in animals have shown neurodevelopmental effects of perinatal dexamethasone treatment in rodents. Doses of 0.2 – 3 mg/kg (which encompasses the therapeutic range in humans) given to the pregnant dam during gestation or to the offspring postnatally alter neurogenesis and differentiation (Bohn, 1984; Carlos et al., 1992), decrease brain size and brain weight (DeKosky et al., 1982; Carlos et al., 1992; Ferguson and Holson, 1999), and alter locomotor activity and learning and memory behavior (DeKosky et al., 1982; Vicedomini et al., 1985; Ferguson et al., 2001; Kreider et al., 2005a). Relatively low doses (0.05 – 0.2 mg/kg) have also been shown to result in long-lasting changes in neurotransmitter systems and intracellular signaling (Kreider et al., 2005b; Kreider et al., 2006; Slotkin et al., 2006). Effects of dexamethasone, including decreased brain weight and hippocampal damage, have also been observed in nonhuman primates (reviewed in Coe and Lubach, 2005).

Human developmental neurotoxicity is associated with perinatal exposure to dexamethasone. Prenatal dexamethasone is routinely administered to mothers at risk for preterm delivery to reduce mortality and the incidence of respiratory distress syndrome and intraventricular hemorrage in premature infants. Postnatal dexamethasone treatment in preterm infants is also used to reduce the risk and severity of chronic lung disease. A preponderance of epidemiologic and clinical evidence, however, indicates that both pre- and post-natal exposure to dexamethasone can result in an increased risk for cerebral palsy, decreased brain size, and long-term effects on cognition and behavior (reviewed in Baud, 2004; Purdy, 2004; Purdy and Wiley, 2004; Sloboda et al., 2005).

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Association of vascular fluoride uptake with vascular calcification and coronary artery disease.

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Abstract

OBJECTIVE: The feasibility of a fluoride positron emission tomography/computed tomography (PET/CT) scan for imaging atherosclerosis has not been well documented. The purpose of this study was to assess fluoride uptake of vascular calcification in various major arteries, including coronary arteries.

METHODS: We retrospectively reviewed the imaging data and cardiovascular history of 61 patients who received whole-body sodium [^{18}F]fluoride PET/CT studies at our institution from 2009 to 2010. Fluoride uptake and calcification in major arteries, including coronary arteries, were analyzed by both visual assessment and standardized uptake value measurement.

RESULTS: Fluoride uptake in vascular walls was demonstrated in 361 sites of 54 (96%) patients, whereas calcification was observed in 317 sites of 49 (88%) patients. Significant correlation between fluoride uptake and calcification was observed in most of the arterial walls, except in those of the abdominal aorta. Fluoride uptake in coronary arteries was demonstrated in 28 (46%) patients and coronary calcifications were observed in 34 (56%) patients. There was significant correlation between history of cardiovascular events and presence of fluoride uptake in coronary arteries. The coronary fluoride uptake value in patients with cardiovascular events was significantly higher than in patients without cardiovascular events.

CONCLUSION: sodium [^{18}F]fluoride PET/CT might be useful in the evaluation of the atherosclerotic process in major arteries, including coronary arteries. An increased fluoride uptake in coronary arteries may be associated with an increased cardiovascular risk.

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Original Paper

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Caries and Fluorosis Prevalence in Communities with Different Concentrations of Fluoride in the Water

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48.4% of children in low fluoride area report no cavities. 46.8% in high fluoride area had no cavities. The fluorsis index was 8x lower, however, in the low-fluoride area. Socio-economic status, sweet consumption + oral habits affect cavities not more fluoride in the water.

Key Words
Dental caries · Fluorosis · Italy · Public health dentistry · Water fluoridation

Abstract
The need to defluoridate and fluoridate the water supplies in areas with drinking water naturally containing above-optimal (≥2.5 mg/l) and suboptimal (≤0.3 mg/l) fluoride concentration and caries and fluorosis prevalence of 12-year-old schoolchildren were assessed in Italy. In the low-fluoride area, 48.4% children were caries-free (DMFT = 0) and the DMFT and DMFS were 1.5 and 2.6; in the high-fluoride area, 46.8% had a DMFT = 0 and the values of the indices were 1.4 and 1.6, respectively. Multiple logistic regression analysis showed a significant association in the caries-free status according to parents' employment status (OR = 1.2, 95% CI = 1.1–1.3) and children's sweets consumption, since children who consumed sweets at least once a day had an adjusted odds ratio of 1.8 (95% CI = 1.4–2.3) compared to those with a lower consumption. Multiple linear regression analysis showed that DMFT and DMFS were significantly higher in children with a lower socioeconomic status and in those who consumed sweets at least once a day, with the DMFS significantly associated also with the area of residence. DT and FT scores were higher in the high- and low-fluoride areas, respectively. No evidence of fluorosis was reported in 94.5 and 55.3% of children in the low- and high-fluoride areas, respectively. The Community Fluorosis Index (CFI) for all permanent teeth was significantly higher in the high-fluoride area, 0.8, than the value, 0.1, found in the low-fluoride community. Our results substantiate the difficulties in defining universal guidelines for the fluoridation or defluoridation of drinking water and the need for an epidemiological approach to the decision as to fluoridate and defluoridate the water supply.

By the 1990s it became apparent that the current range of optimal concentration of fluoride in drinking water (0.7–1.2 mg/l) was not appropriate for all parts of the world. Even in the United States, where this range of concentration was developed [US Public Service, 1962], the advent of air-conditioning, the increased consumption of processed soft drinks and foods, and the widespread availability of fluoride in many forms were rendering obsolete the assumptions upon which the recommended fluoridation range was based. In other parts of the world, in particular the tropical and subtropical parts of Asia and Africa, the recommended fluoridation range had probably never been appropriate, and the unsuitability of international guidelines for fluoride in drinking water had already been established [Manji et al., 1986a, b; Brouwer et al., 1988; Evans, 1989; Warnakula-

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suriya et al., 1992; Irigoyen et al., 1995; Lo and Bagramian, 1996; Villa and Guerrero, 1996]. Moreover, the WHO Expert Committee on Oral Health Status and Fluoride Use [1994] has also recently recommended a more conservative range of 0.5–1 mg/l.

Determining of the most appropriate levels of fluoride in drinking water is crucial both for communities which intend to start water fluoridation and for those with excessive natural fluoride which require partial defluoridation. In these cases, the definitions of explicit criteria appear even more controversial. The European Community Regulations [Direttiva n. 80/778, 1980] do not differentiate between optimal value and maximum allowable concentration and have established a range of 0.7–1.5 mg/l. United States guidelines appear to be more permissive and recommend a water fluoride concentration of 0.7–1.2 mg/l and a maximum allowable value of 4 mg/l [Environmental Protection Agency, 1985, 1986]. Recently, the National Research Council [1993] in the United States has reexamined this limit and concluded that there was no evidence that would necessitate any change, but urged that the situation be constantly reviewed.

The decision to fluoridate or defluoridate the water is complicated, and some have argued that the recommended fluoride concentration should be based on the evaluation of caries and fluorosis prevalence, exposure to other sources of fluoride and the availability of dental care [Ismail, 1995]. The purposes of this study were to determine the need to defluoridate and fluoridate the water supplies of two areas of Italy with drinking water naturally containing above-optimal (≥2.5 mg/l) and suboptimal (≤0.3 mg/l) fluoride concentrations and to report the results of a survey of caries and fluorosis prevalence.

Materials and Methods

The population for this study consisted of schoolchildren 12 years of age who, according to questionnaires, had lived continuously since birth in their respective communities located in the southern part of Italy, and who had always used the community water supply as their primary source of drinking water. The children from low-fluoride (≤0.3 mg/l) communities resided in Catanzaro. The high-fluoride (≥2.5 mg/l) communities were located in the area around Naples.

Random sampling was used to select the primary public schools, from each school, classes were randomly selected and all children in the selected classes were asked to participate in the study, if eligible. Parents of sampled children were notified about the study by the school and invited to participate. The clinical examination took place during the period January to May 1997.

The children were examined in the schools. The 2 examiners had previously been trained and calibrated. Only the permanent teeth were examined. A surface had to be fully erupted to be examined or, if restored, sufficient tooth structure (more than 75%) had to remain. Teeth were not included if they were banded orthodontically or were partially erupted.

The examination of dental caries was carried with portable equipment, using a mirror and probe; no radiographs were taken. The DMFT and DMFS indices were used to record caries experience [World Health Organization, 1987]. The presence of natural teeth was recorded according to the WHO criteria [1987]. The dental information reported included the teeth present and whether these teeth required treatment according to WHO criteria [1987]. Enamel fluorosis was determined for each child using Dean's [1942] index. Each child was classified on the basis of the two teeth in the mouth showing the most advanced signs of fluorosis. Wherever two teeth were not affected to the same degree, the child was assigned the score of the lesser affected tooth. The examiners used criteria described by Russell [1961] for differentiating fluorosis from nonfluoride enamel opacities. According to Dean et al. [1942], only scores of 1–4 were considered definitive for fluorosis and, therefore, for the computation of fluorosis prevalence the category of questionable was not included. The Community Fluorosis Index (CFI), as described by Dean [1942], was also calculated to determine if community differences in the severity of fluorosis existed. The questionable category was included in the calculation of the CFI score.

Before the dental examination, all children completed a pretested, structured questionnaire, under the supervision of a parent/guardian, that sought information on: sociodemographics, consumption of sweets, frequency of oral hygiene habits, use of fluoride vehicles, and utilization of dental services. The sociodemographic variables were sex, age, child's residential history, and parent(s)/guardian(s) employment status. When a child had two working parents/guardians, the highest occupation was considered. The questions concerning frequency of consumption of sweets used the four following categories: (a) never, (b) less than once a day, (c) each day, (d) several times a day. The frequency of toothbrushing was evaluated as following: (a) less than once a day, (b) once a day, (c) more than once a day. The questions concerning the use of fluoride vehicles included information regarding whether or not fluoride dentifrices or fluoride supplement drops or tablets were used during each of the first 6 years of life and at the time of the investigation. The questions on the use of dental services asked whether the child had ever had a dental visit, and the reason for these dental visits.

The data were analyzed using the Stata software program [Stata Corp., 1993]. Multiple logistic regression and multiple linear regression analyses were performed to identify the variables that affect the following dental caries and fluorosis outcomes: caries-free status, DMFT, DMFS, DT, FT, and CFI. In all models the explanatory variables included were the following: area of residence (low fluoride water level = 0, high fluoride water level = 1), sex (male = 0, female = 1), parent(s)/guardian(s) employment status (five categories: high professional and managerial = 1, lower managerial = 2, senior clerical, small commercial operators = 3, skilled artisans, farmers = 4, others = 5), and toothbrushing habits (less than once a day = 0, at least once a day = 1). In caries models, the variables dental visit (never or for a dental problem = 0, routine checkup = 1) and frequency of consumption of sweets (less than once a day = 0, at least once a day = 1) were included. The model building strategy included the following steps: (1) univariate analysis of each variable considered, using the appropriate test statistic (chi-square test, t test or one-way analysis of variance); (2) inclusion of any variable whose univariate test has a p value lower than 0.25; (3) backward elimination of any variable which does not contribute to the model on the ground of the Likeli-

Table 1. Caries experience according to various explanatory variables in children from areas with different levels of fluoride in their water

	Low-fluoride area									
	n	caries-free %	DMFT	DT	MT	FT	DMFS	DS	MS	FS
Sex	(461)									
Men	233	49.8	1.5	0.5	0.1	0.9	2.4	0.9	0.4	1.1
Women	228	46.9	1.6	0.6	0.1	0.9	2.7	2.2	0.3	0.2
Parents' employment status	(455)									
High professional and managerial	127	57.5	1.2	0.2	0.1	0.9	2.1	0.4	0.6	1.1
Lower managerial	171	45.0	1.5	0.5	0.0	1.0	2.2	0.8	0.1	1.3
Senior clerical, small commercial operators	44	47.7	1.4	0.7	0.0	0.7	2.7	1.7	0.0	1.0
Artisans, farmers	90	41.1	1.8	0.8	0.1	0.9	3.4	1.8	0.5	1.1
Others	23	52.2	1.5	0.8	0.2	0.5	3.0	1.5	0.9	0.6
Toothbrushing habits	(458)									
Less than once a day	37	59.5	1.1	0.6	0.0	0.5	2.2	1.4	0.0	0.8
At least once a day	421	47.3	1.5	0.5	0.1	0.9	2.6	1.0	0.4	1.2
Dental visit	(453)									
Never or only when trouble	217	43.3	1.6	0.7	0.1	0.8	2.9	1.5	0.3	1.1
Routine checkup	236	53.8	1.4	0.4	0.1	0.9	2.2	0.7	0.4	1.1
Sweets consumption	(452)									
Less than once a day	276	54.0	1.3	0.4	0.1	0.8	1.9	0.6	0.3	1.0
At least once a day	176	39.2	1.9	0.8	0.1	1.0	3.5	1.6	0.5	1.4

In parentheses the number of children responding to the question is given.

hood Ratio Test (logistic regression) and the F test statistic (linear regression), using a cutoff of 0.05 level significance; variables whose exclusion alters the coefficient of the remaining variables are kept in the model; (4) testing of interaction terms using a cutoff of 0.15 level significance [Kleinbaum et al., 1988; Hosmer and Lemeshow, 1989]. The outcome caries-free status was dichotomized into 'caries-free' (DMFT = 0) and 'caries' (DMFT ≥ 1). Adjusted odds ratio (OR) and 95% confidence intervals (CI) were calculated.

Results

A total of 462 children participated in the low-fluoride group and 553 children participated in the high-fluoride group for response rates of 81.3 and 90.5%, respectively. Almost all children (99.2%) used fluoride toothpastes and the large majority of them (98.7%) were using dentifrices with standard fluoride concentration (<1,000 ppm) since the early years of life. Use of fluoride supplements between birth and 6 years of age was not common, since in the low- and in high-fluoride areas only 3 and 1% of children, respectively, reported a regular use of supplements in that period; a greater proportion of children reported a regular use of supplements at the time of the investigation (23 and 11%, respectively). Therefore, the information collected on the use of fluoride vehicles clearly indicated that this factor is very unlikely to contribute to the differences in fluorosis and caries prevalence in the two areas surveyed.

There were no significant differences between the children living in the low- and high-fluoride areas with regard to sex (chi-square = 0.01, 1 d.f., p = 0.91) and utilization of dental services (chi-square = 1.07, 1 d.f., p = 0.3), while children in the low-fluoride area had higher socioeconomic status (chi-square = 68.31, 4 d.f., $p<0.0001$), better toothbrushing habits (chi-square = 40.06, 1 d.f., $p<0.0001$) and consumed sweets less frequently (chi-square = 12.49, 1 d.f., $p<0.0001$) than those in the high-fluoride area.

In the children living in the area with a low fluoride water concentration the prevalence of caries-free status (DMFT = 0) was 48.4% and the DMFT and DMFS scores were 1.5±1.9 (SD) and 2.6±3.9 (SD), while 46.8% of the children in the area with high fluoride level had a DMFT = 0 and the mean (± SD) values of the indices were 1.4±1.7 and 1.6±1.9, respectively. However, at univariate analysis, only differences in DMFS turned out to be statistically significant (t = 4.46, 1,012 d.f., $p<0.0001$). Children living in the low-fluoride area had a significantly lower DT score

High-fluoride area									
n	caries-free %	DMFT	DT	MT	FT	DMFS	DS	MS	FS
(553)									
277	47.3	1.4	1.2	0.0	0.2	1.5	1.3	0.0	0.2
276	46.0	1.5	1.3	0.0	0.2	1.8	1.4	0.1	0.3
(553)									
52	50.0	1.1	0.9	0.0	0.2	1.2	1.0	0.0	0.2
208	53.9	1.2	0.9	0.0	0.3	1.5	1.1	0.0	0.4
89	44.9	1.4	1.3	0.0	0.1	1.6	1.5	0.0	0.1
153	41.2	1.6	1.5	0.0	0.1	1.9	1.7	0.0	0.2
51	33.3	1.6	1.6	0.0	0.0	2.1	2.0	0.1	0.0
(553)									
126	39.7	1.5	1.4	0.0	0.1	1.7	1.6	0.0	0.1
427	48.7	1.4	1.2	0.0	0.2	1.6	1.3	0.0	0.3
(553)									
283	47.7	1.5	1.3	0.0	0.2	1.8	1.5	0.0	0.3
270	45.6	1.3	1.2	0.0	0.1	1.5	1.3	0.0	0.2
(553)									
276	54.4	1.1	1.0	0.0	0.1	1.2	1.1	0.0	0.1
277	39.0	1.7	1.4	0.0	0.3	2.0	1.7	0.0	0.3

Table 2. Results of the logistic regression model (1) and linear regression models (2–6) for estimates of associations of caries experience and fluorosis with potential risk factors

Variable	OR	SE	95% CI	p value
Model 1: Outcome: Caries-free status				
log-likelihood = –676.94, chi-square = 32.48 (2 d.f.), $p<0.0001$				
Sweets consumption	1.81	0.23	1.40–2.33	<0.001
Parents' employment status	1.17	0.06	1.05–1.30	0.003

Variable	Coeff.	SE	t	p value
Model 2: Outcome: DMFT				
$F(2,994) = 15.46$, $p<0.0001$, $R^2 = 3.02\%$, adjusted $R^2 = 2.88\%$				
Parents' employment status	0.11	0.05	2.24	0.026
Sweets consumption	0.58	0.12	4.85	<0.001
Constant	0.89			
Model 3: Outcome: DMFS				
$F(3,993) = 20.44$, $p<0.0001$, $R^2 = 5.82\%$, adjusted $R^2 = 5.53\%$				
Parents' employment status	0.27	0.08	3.23	0.001
Residence	–1.14	0.21	–5.52	<0.001
Sweets consumption	1.08	0.20	5.36	<0.001
Constant	1.45			

Variable	Coeff.	SE	t	p value
Model 4: Outcome: DT				
$F(3,993) = 35.75$, $p<0.0001$, $R^2 = 9.75\%$, adjusted $R^2 = 9.47\%$				
Parents' employment status	0.19	0.04	5.04	<0.001
Residence	0.55	0.09	5.99	<0.001
Sweets consumption	0.38	0.09	4.27	<0.001
Constant	–0.06			
Model 5: Outcome: FT				
$F(2,1005) = 42.28$, $p<0.0001$, $R^2 = 7.76\%$, adjusted $R^2 = 7.58\%$				
Parents' employment status	–0.07	0.03	–2.05	0.041
Residence	–0.66	0.08	–8.31	<0.001
Constant	1.04			
Model 6: Outcome: CFI				
$F(1,1006) = 256.35$, $p<0.0001$, $R^2 = 20.31\%$, adjusted $R^2 = 20.23\%$				
Residence	0.63	0.03	16.01	<0.001
Constant	0.14			

Table 3. Prevalence of Dean's fluorosis index scores (%) and CFI according to various explanatory variables in children from areas with different levels of fluoride in their water

	Dean's fluorosis score, %							
	Low-fluoride area							
	n	normal	questionable	very mild	mild	moderate	severe	CFI (SD)
Sex	(461)							
Men	233	81.6	13.7	3.9	0.4	–	0.4	0.13(0.36)
Women	228	82.5	11.0	5.7	0.4	–	0.4	0.14(0.37)
Parents' employment status	(455)							
High professional and managerial	127	81.1	14.2	4.7	–	–	–	0.12(0.26)
Lower managerial	171	78.4	13.4	6.4	0.6	1.2	–	0.18(0.48)
Senior clerical, small commercial operators	44	84.1	6.8	6.8	2.3	–	–	0.15(0.40)
Artisans, farmers	90	87.8	11.1	1.1	–	–	–	0.07(0.19)
Others	23	82.6	13.0	4.4	–	–	–	0.11(0.26)
Toothbrushing habits	(458)							
Less than once a day	37	78.4	13.5	8.1	–	–	–	0.15(0.31)
At least once a day	421	82.2	12.3	4.5	0.5	0.5	–	0.13(0.37)
Dental visit	(453)							
Never or only when trouble	217	83.4	14.3	1.8	0.5	–	–	0.11(0.32)
Routine checkup	236	80.1	11.0	7.6	0.9	0.4	–	0.16(0.40)
Sweets consumption	(452)							
Less than once a day	276	81.5	12.7	4.4	0.7	0.7	–	0.15(0.41)
At least once a day	176	82.4	11.9	5.7	–	–	–	0.12(0.27)

In parentheses the number of children responding to the question is given.

(t = –7.52, 1,012 d.f., $p<0.0001$) and higher FT score (t = 9.01, 1,012 d.f., $p<0.0001$) compared to those in the high-fluoride communities. Other factors, besides residence, found significantly associated to caries experience were parents' employment status and sweets consumption. In particular, no caries-free status, DMFT and DMFS indices appeared to be higher in lower socioeconomic classes ($p<0.0001$) and tended to increase with the increase of frequency of sweets consumption ($p<0.0001$).

Table 1 presents the percentage of children who were caries-free and the mean DMFT and DMFS scores according to various explanatory variables and by water fluoride status. The effects on caries experience of parents' employment status and sugar consumption were evident both in low- and high-fluoride areas. Indeed, DMFT increased from 1.2 to 1.8 and from 1.1 to 1.6 as the parents' employment status decreased in the low- and high-fluoride areas, respectively, whereas DMFS increased from 2.1 to 3.4 and from 1.2 to 1.9. DMFT and DMFS were approximately 50 and 80% higher both in low- and high-fluoride areas in children consuming sweets at least once a day. Multivariate analyses substantially confirmed the results of univariate analysis. In particular, the results of multiple logistic regression analysis showed a significant association in the caries-free status according to parents' employment status (OR = 1.2, 95% CI = 1.1–1.3) and children's sweets consumption, since children who consumed sweets at least once a day had an adjusted OR of 1.8 (95% CI = 1.4–2.3) compared to those with a lower consumption (model 1 in table 2). The results of multiple linear regression analysis showed that DMFT and DMFS were significantly higher in children with a lower socioeconomic status and in those who consumed sweets at least once a day, with the DMFS significantly associated also with the area of residence (models 2 and 3 in table 2). The effect of the area of residence was significant also on DT and FT scores, since they were higher in the high- and low-fluoride areas, respectively (models 4 and 5 in table 2). No significant interactions among the variables at 0.15 level were detected, and therefore they were not included in the final models. In all fitted models, graphs of residuals did not show any clear evidence of curvilinearity, heteroscedasticity and outliers.

High-fluoride area							
n	normal	questionable	very mild	mild	moderate	severe	CFI (SD)
(553)							
30.3	30.3	22.0	31.0	14.1	2.2	0.4	0.78(0.75)
34.4	34.4	23.9	24.3	14.5	2.5	0.4	0.74(0.78)
(553)							
52	28.8	23.1	32.7	13.5	1.9	–	0.77(0.72)
208	37.0	23.6	24.0	13.9	1.0	0.5	0.68(0.73)
89	28.1	21.4	39.3	10.1	1.1	–	0.74(0.64)
153	32.0	22.9	24.2	16.3	3.9	0.7	0.82(0.84)
51	25.5	23.5	27.5	17.6	5.9	–	0.92(0.86)
(553)							
126	27.8	29.4	26.9	15.9	–	–	0.73(0.67)
427	33.7	21.1	27.9	13.8	3.0	0.5	0.77(0.79)
(553)							
283	31.8	25.1	26.9	13.8	2.1	0.3	0.75(0.75)
270	33.0	20.7	28.5	14.8	2.6	0.4	0.78(0.78)
(553)							
276	32.2	21.7	28.6	14.9	2.2	0.4	0.77(0.76)
277	32.5	24.2	26.7	13.7	2.5	0.4	0.75(0.77)

Table 3 shows the percentage distribution of Dean's fluorosis score in the examined children according to various explanatory variables and by water fluoride status. The prevalence of dental fluorosis (score of 1 and higher) was related to the water fluoride level of the community. Indeed, 94.5% of the children had no evidence of fluorosis in the area with low fluoride concentration in the water supply as compared to 55.3% of the children in the high-fluoride area. In the remaining, a relatively small proportion of children showed definite signs of fluorosis in the high-fluoride area: only 2.7% of the teeth affected had a score of 3 (moderate) and no evidence of severe degree was observed. The mean (± SD) score using the CFI for all permanent teeth was 0.1±0.3 and 0.8±0.8 in children living in low- and high-fluoride communities, respectively. The results of multiple linear regression analysis indicated that the mean CFI score was significantly higher in children living in the area with high fluoride concentration in the water supply compared to those in the low-fluoride area (model 6 in table 2). Again, no evidence of curvilinearity, heteroscedasticity and outliers was found at the graph of residuals.

Discussion

The debate about the relationship between the concentration of fluoride in drinking water and dental caries and fluorosis began with Dean's [1942] 21-city study in the United States. A resurgence of interest has occurred during the past two decades, with a large body of literature published in several countries; comparisons with these studies must be interpreted cautiously because of different climatic conditions, patterns of water and dentifrice ingestion, dietary habits, fluoride supplement exposition, use of different criteria and indices, and a lack of uniformity of collected information. We agree with Nowjack-Raymer et al. [1995] that limits of any retrospective study exist mainly because of the possible inaccuracy of a parent to recall information about a child's fluoride intake. However, our results suggest that, apart from the possibly misclassified child, all children ingested similar quantities of fluoride. Comparison with the most recent international figures on caries at age 12 shows that children in our study had better dentition than those reported in most other industrialized

countries of comparable life-style supplied with similar water fluoride concentrations. Indeed, in low-fluoride areas our caries-free prevalence, 48.4%, and DMFS index, 2.6, were better than the values reported in the United States by Grobler et al. [1986] (36.4% and 4.9), Ismail et al. [1990] (8.4% and 8.63), and Jackson et al. [1995], who reported a DMFS of 6.65. Treasure and Dever [1994] in New Zealand found that 22% were caries-free and had a DMFS of 6.2. Moreover, our DMFT, 1.5, was lower than the 1.91 observed by one of us in children living in an area with 0.3 ppm fluoride water concentration [Angelillo et al., 1990]. In high-fluoride areas, the percentage of caries-free children, 46.8%, and the DMFS score, 1.6, were lower that the values found in the United States by Driscoll et al. [1983] with a DMFS of 2.59, Grobler et al. [1986] with values of 23.5% and 8.63, and Jackson et al. [1995], who found a DMFS of 4.47. Data from recent studies in European countries showed that declines of DMFT averages to 1.0 seem to be attainable. This is particularly borne out by the averages from England and Wales as well as Finland, with a DMFT of 1.2, and from the Netherlands and Switzerland, where averages as low as 0.8–1.1 have been reported [Marthaler et al., 1996]. The reasons for this very encouraging and continuing decline in caries prevalence among the populations of many areas of the developed world may be attributed to the introduction of fluoride into a number of oral health care products and in particular the increased use of fluoride toothpastes.

The employment status of parents, an indicator of socioeconomic status, and children's sweets consumption were shown in this study to be associated with caries prevalence. Indeed, children from lower socioeconomic status and those who consumed sweets at least once a day had a significantly greater level of dental caries experience in terms of the absolute number of subjects affected and caries indices. These findings have confirmed several previous studies [Serra Majem et al., 1993; Kalsbeek and Verrips, 1994; Grindefjord et al., 1996; Petridou et al., 1996; Angelillo et al., 1998]. Moreover, in the current investigation the beneficial effect of water fluoride on caries experience was observed when comparing children from the low- to those from the high-fluoride community. Indeed, the DMFS value in the low-fluoride area was significantly higher, and our result indicates that water fluoride may continue to provide protection even in the presence of a low prevalence of dental caries. This finding is in accordance with results from previous studies that have shown an additional benefit in caries reduction when comparing a low- to a high-fluoride community [Driscoll et al., 1983, 1986; Angelillo et al., 1990; Ismail et al., 1993; Jackson et al., 1995].

At the low fluoride level, 4.7% of the children had dental fluorosis with a Dean's score of 0.1, and these results offer support for the finding reported in children who were lifetime residents of a low-fluoride community in the United States. Indeed, fluorosis was almost absent with only 2.9% of the children examined showing any definite signs of the condition and a Dean's index of 0.1 [Driscoll et al., 1986]. Moreover, Leverett [1986] found a prevalence of 10.1% and Kumar et al. [1989] a value of 9.4% and a Dean's score of 0.23. Present data on the prevalence and severity of dental fluorosis recorded in the high-fluoride area were lower than in most studies carried out in other countries. Prevalence recorded in the permanent dentition of US schoolchildren with a fluoride concentration in the drinking water of 3.48–4.07 ppm was 87.5% [Driscoll et al., 1983]. More recently, data from children in Mexico, who were exposed to water fluoride concentrations of about 2.8 ppm, showed a prevalence of fluorosis of 97.8%, with more than two thirds of the children classified in the moderate and severe categories, and a Dean's index of 2.9 [Irigoyen et al., 1995]. Jackson et al. [1995], in children living in an area with water containing 4 ppm of fluoride, found that 89.7% had evidence of fluorosis and almost half of the children received a Dean's score of 3 or greater. This extreme heterogeneity in findings, across various countries, makes the definition of universal guidelines on water fluoride concentration especially difficult, and suggests the need for a more pragmatic approach. In our survey, it was obvious that the higher water fluoride concentration led to an increased fluorosis prevalence, but it should be pointed out that the pattern of severity seems to be the same, and almost all subjects have less than moderate fluorosis. According to Dean [1942], the CFI score of 0.1 for the low-fluoride area warrants no public health concern, while the score of 0.8 in the high-fluoride area constituted a slight problem. Both scores, although significantly different, show that the prevalence of fluorosis in the two communities might not be considered a public health problem by Dean's criterion. This finding is consistent with past and present findings [Driscoll et al., 1986; Leverett, 1986; Ismail et al., 1993; Jackson et al., 1995].

The ultimate goal of this survey was to collect epidemiological data that could support the decision-making process about the choice for implementing a water treatment program (fluoridation or defluoridation) in the areas surveyed. In the low-fluoride community, fluorosis and caries prevalence results indicated that a critical level of fluoride exposure in this population had not been reached, and that water fluoridation was very likely to be an effective caries-preventive measure. However, caries prevalence appears to

be very low and it is well known that the cost-effectiveness of water fluoridation programs depends to a great extent on the underlying levels of caries in the target population [White et al., 1989; Birch, 1990]. Therefore, from a cost-effectiveness point of view, water fluoridation needs to be assessed with other alternative programs, in order to efficiently use resources aimed at the promotion of dental health.

In the above-optimal fluoride area a decision to not implement a defluoridation program appears more straightforward. Unfortunately, current Italian regulations mandate that the high-fluoride area that has been studied is required to defluoridate its water supply or to shift to alternative water sources with lower fluoride concentration. These actions are not supported by our results and, although we did not assess the community acceptance of fluorosis, according to the findings of recent epidemiologic studies, fluorosis is perceived as a minor aesthetic problem and is not a concern for the public at large [Ismail et al., 1990, 1993; Williams and Zwemer, 1990; Clark et al., 1993; Riordan, 1993]. The benefits of water fluoride, in terms of protection against dental caries, appears to be substantiated, particularly because the availability of dental care, given the high contribution to the DMFT score of active decay, does not appear satisfactory. Previous studies have shown that discontinuation of water fluoridation or defluoridation of naturally fluoridated water supplies result in a significant increase in dental caries and a doubling of the cost of restorative care [Stephen et al., 1987; Attwood and Blinkhorn, 1989].

In conclusion, the results of our study document the difficulties in defining universal guidelines for the fluoridation or defluoridation of drinking water. Water treatment programs considering fluoridation or defluoridation cannot rely solely upon international standards, but must add epidemiological considerations to the decision as to fluoridate and defluoridate the water supply. Information on caries and fluorosis prevalence, exposure to other fluoride products and availability of dental care are fundamental in order to make rational choices, implement cost-effective interventions and thus to realize the considerable savings inherent in avoiding unnecessary and costly water treatment programs.

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Dental fluorosis decline after changes to supplement and toothpaste regimens

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* See page 238 *
For Major Findings

Riordan PJ. Dental fluorosis decline after changes to supplement and toothpaste regimens. Community Dent Oral Epidemiol. 2002; 30: 233–40. ©Blackwell Munksgaard, 2002

Abstract – In 1989/90, in 659 12-year-olds in Perth (F 0.8 mg/L) and the Bunbury region of Western Australia (WA) (F ~0.25 mg/L), dental fluorosis prevalences were 40.2% and 33.0%. Fluoride supplements (OR 4.63) and extended residence in a fluoridated area (OR 4.06) were significant risk factors; toothpaste ingestion variables had ORs greater than unity; in 1990, DMFT for this age group was 0.84. School Dental Service took steps to discourage supplement and toothpaste ingestion and to promote low fluoride toothpaste for children < 6 years of age. *Objectives:* To evaluate the effect of this campaign on fluorosis and caries. *Methods:* Between May–July 2000, 582 10-year-olds were examined for dental fluorosis (TF index) and dental caries (DMFT) in school dental clinics. *Results:* Fluorosis prevalence was 22.2% in Perth and 10.8% in the Bunbury region. Overall prevalence was 18.0% and of this, 80.2% was TF 1, 17.9% was TF 2 and just 1.9% was TF 3. In 1989/90, 79 children had used supplements before the age of 4 year; in 2000 only 40 had done so ($P < 0.001$). Mean DMFT values in Perth and Bunbury were 0.32 and 0.28 ($P > 0.05$). Low F toothpaste, unavailable in 1989/90, had been used by 24.5%. The only significant risk factor was residence, OR 2.0. *Conclusions:* Fluorosis prevalence seems to have fallen in parallel with a reduction in discretionary intake from supplements and toothpaste. No increase in dental caries experience was recorded. Because the teeth examined in this study were at risk of fluorosis in 1992–95, very soon after policies changed, and because people are slow to change health habits, it seems reasonable to expect a further improvement when teeth mineralised in the late 1990s become visible.

Key words: caries, dentifrice, enamel mottling, fluoride, fluorosis, supplement

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In 1989/90 in Western Australia, a survey of dental fluorosis among children (12 year old, $n = 659$) revealed a prevalence of 0.40 in the fluoridated Perth metropolitan area, and 0.33 in the nonfluoridated region around the south-west towns of Bunbury and Busselton (1). The principal risk factors identified in that study were, in descending order of Odds Ratio (OR) magnitude, use of fluoride supplements (OR 4.63, 95% CI 1.97, 10.90), residence in a fluoridated area (OR 4.06, CI 2.55, 6.44) and variables associated with toothpaste use and ingestion at young age (various ORs, range 1.02–1.35, all *n.s*). The use of fluoride supplements in the nonfluoridated areas was in accordance with professional advice at the time.

A subsequent study indicated that dental fluorosis in these children that exceeded TF score 1 was considered unattractive by dentists, parents and other lay people (2) and since that work, other researchers working in different societies have come to essentially the same conclusion, that as fluorotic lesions are more severe, aesthetic concerns increase (3–6). While there is an understandable tendency to play down the importance of aesthetic concerns as a trade-off for improved dental health (7), fluorotic lesions are undesirable. Arguably, they are unaesthetic manifestations of ingestion of mildly toxic quantities of fluoride and their prevalence and severity should be minimised.

As a result of the earlier studies in Western Aus-

tralia, several options were considered to reduce the aesthetic impact of systemic fluoride ingestion. Reduction of water fluoride concentration was considered. Reduction of water fluoride concentration in Hong Kong led to lower levels of dental fluorosis (8). An interruption to the fluoridation of water supplies in Durham, NC, resulted in reduced fluorosis prevalence and severity in the age cohorts whose teeth were mineralising at the time of the interruption, and these authors concluded that 'dental fluorosis is sensitive to even small changes in fluoride exposure from drinking water' (9). In the WA situation, although fluoridation was a risk factor for fluorosis, it was not the most important and the majority of those exposed to fluoridated water did not display any dental fluorosis (1). It has long been accepted that low levels of mild fluorosis accompany the use of water fluoridation and the issue of concern in Western Australia was the high prevalence of fluorosis rather than the presence of any fluorosis. It thus seemed logical to examine the contribution of discretionary additional sources of fluoride exposure before considering changes to the water fluoridation regimen.

A review of the literature on fluoride supplements suggested that the scientific basis for fluoride supplements as a community caries preventive strategy was weak; randomised controlled clinical trials of fluoride supplements were few and frequently methodologically deficient (10). Referring in part to generally poor compliance with fluoride supplement regimens, Ismail (11) stated: 'The scientific evidence supports the efficacy of fluoride supplements in caries prevention but there is weaker support for their effectiveness' and they are generally accepted not to be a useful public health strategy (12, 13). In many countries, recommendations to use fluoride supplements have recently been modified either to discontinue their use altogether, or to reduce the quantity of fluoride children ingest (14–19).

All fluoride dentifrices available in Western Australia in 1990 had a fluoride concentration of 1000 mg/L, except for one Colgate® product introduced about that time with fluoride concentration 400 mg/L. Ingestion of toothpaste by small children has been reported (20–23) and such fluoride is virtually totally bio-available (24). The caries preventive effect of low fluoride toothpastes may be slightly less than that of adult strength alternatives (25) but the generally good caries status of the child population (26) made the use of low-fluoride toothpaste by children under 6 years of age seem warranted. To encourage such products to be marketed, a conference was held in Perth in 1993 at which all the larger toothpaste manufacturers, as well as some 25 Australian and international experts were present. Subsequently, in the early 1990s, all the major manufacturers introduced low fluoride toothpastes to the Australian market with fluoride concentrations in the range 400–550 mg/L.

The School Dental Services in Western Australia chose therefore to discourage the use of fluoride supplements by any children regardless of the local water fluoride concentration, and a new dosage schedule for fluoride supplements, entailing ingestion of much lower quantities of fluoride by children younger than 8 years, was published for the benefit of any dentists who felt that the continued use of fluoride supplements was warranted. Advice on toothpaste use, designed to reduce the quantity of fluoride ingested by children under 6 years, was offered; low fluoride toothpaste was recommended, alternatively parents were advised to ensure minimal toothpaste ingestion by using a very small quantity of regular toothpaste, and by encouraging children under 6 years not to swallow toothpaste. Toothpaste manufacturers assisted by printing good advice on quantities of toothpaste to use on the packaging. Because the service reaches about 90% of the schoolchildren in the state (pop. 1.8 m) and because all clinicians employed in the service were made aware of the strategy, and various printed brochures were prepared in support of these policies, it is likely that most parents would have come across the messages at some time.

The purpose of the present study was therefore to evaluate the effect of the changed regimen on discretionary fluoride use by conducting a new survey to measure dental fluorosis and dental caries.

Participants and methods

The study followed, as far as possible, the same protocol as had been used in 1989/90 (1), but participants in the present study were aged 10 years rather than 12 years as in the 1989/90 study. All participants were enrolled patients in the School Dental Service whose parents had agreed to provide background information for the purpose of this survey. All examinations were conducted between May and July 2000.

Participants

In the Perth metropolitan area (water fluoride concentration 0.85 mg/L), a sample of 14 schools was

drawn at random from a list of schools that had on-site Dental Therapy Centres (DTCs), the clinics in which school dental care is provided. Because one DTC was undergoing building maintenance, a mobile clinic was being used temporarily.

In and around the city of Bunbury, 200 km south of Perth, there is no water fluoridation. Natural fluoride levels vary depending on which bore is used to supply water, but they average around 0.2–0.3 mg/L (communications from Bunbury and Busselton water boards). Because the population in this area is much smaller than in Perth, the same DTCs as were used in 1990 were chosen, except that one clinic had been closed so another was chosen. One mobile clinic was used; it was fortunately located in the same town (Donnybrook) as it had been in 1990. Mobile clinics are slightly cramped but are otherwise equipped to the same standard as fixed clinics.

Background information

DTC staff were asked to distribute study information and questionnaires to all children born in 1990 (i.e. about 10 years old) who attended the school to which the DTC was attached and who already were enrolled in the school dental service. The information and questionnaire were to be brought home to a parent. The information explained the purpose of the study and invited the parent to complete the questionnaire which requested information about residence from birth, drinking water sources, fluoride supplement use, use of infant formula, toothpaste use and toothpaste preferences. The questions were identical to those used for the 1989/90 study. Two weeks after the first questionnaire was distributed, DTC staff contacted children from whom no response had been received and if necessary provided a second questionnaire. In a few cases, the background information was collected by telephone directly from a parent.

Fluoride exposure

For each participant, residence in the period from birth to five years of age was categorised as 'F' if more than half the period had been spent in a fluoridated area and 'Non-F' if not. In the case of children who had migrated from other parts of Australia and New Zealand, the child's fluoride exposure could be determined without difficulty. Migrants from other parts of the world were excluded because their fluoride exposure from water, toothpaste and supplements could not be determined with accuracy.

Fluorosis and caries examinations

The standard of clinical equipment was similar in the fixed and mobile clinics. Routine clinical hygiene measures (disposable gloves, autoclaved instruments) to avoid cross-infection were employed. Children were brought from their classroom to the DTC in groups of 3–6. They were provided with a new toothbrush* and toothpaste, and supervised while they cleaned the upper anterior teeth. Each child rinsed with water and was helped to place a cotton roll under the upper lip in an attempt to keep moisture away while waiting to be examined. For examination, each child sat in a normal dental chair, and the maxillary anterior teeth were dried using an air blower. Using the normal clinical light, the upper right central incisor (tooth 11) was examined and fluorosis scored using the TF index (27). In one clinic, a short power cut led to the light failing and about 20 children were examined outdoors under winter sunlight. Caries examinations were conducted according to WHO criteria (28) and generally without probing except to remove debris. Fissure sealants were not scored as restorations. Children not at school were lost to the survey, except that there was an opportunity to revisit one school in Bunbury a few days after the initial visit, and three additional children were examined. All clinical examinations were conducted by one experienced examiner, with one or two clinic assistants.

Reliability of examinations

Since an important part of the present survey was to compare its fluorosis findings with data from the 1989/90 survey, an estimate of examiner variation was necessary. In 1989/90, colour slides were made in a standard way for almost all participants. In 2000, no photographs were taken but a randomly selected set of 80 colour slides (selected independently by an assistant to cover TF scores in the range 0–3) from the 1989/90 survey were re-examined and re-scored by the same examiner. The weighted Kappa value for this re-examination was 0.75 indicating 'substantial' agreement on the scale of Landis and Koch (29).

Analyses

For each child, the clinical findings were called by the examiner and noted by an assistant on a list of participants derived from the school class lists.

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* Toothbrushes, which the children kept, were very kindly provided by Colgate Oral Care Pty, Sydney New South Wales.

Later, the same assistant transferred the findings to computer and combined them with data from the questionnaires. They were analysed using the StatView program (SAS Institute Inc, Cary, NC). Fluorosis and caries prevalence were defined as the proportion of participants who had, respectively, TF score > 0 or DMFT > 0. ANOVA and nonparametric methods were used to estimate differences between subgroups and to identify variables possibly associated with the presence of fluorosis. Such variables were entered into a logistic regression analysis as independent predictors and the outcomes expressed in terms of odds ratios and confidence intervals. The methods were analogous to those used previously (1).

Results

Participation

Altogether 672 children, chosen on the basis of class lists, were invited to participate in the survey. Of these, 58 were excluded for reasons such as not born in 1990, consent refused ($n = 24$), absence from school on the day of the examinations or having left the school by the time the examinations were conducted. A further 31 children who had been examined were not included in the study because they had not lived their first four years of life in any part of Australia or New Zealand. This report is thus based on information from the remaining 582 children, 283 girls and 299 boys.

Fluoride exposure

Three hundred and seventy-five children (64.4%) had lived the major part (≥ 2.5 year) of their first 4 years of life in areas with fluoridated water supplies.

Infant formula had been used from birth by 160 (27.5%) children, and a further 90 commenced formula use between 3 and 6 months of age. By the age of 12 months, 336 children (57.7%) had used formula but the duration of use was not recorded.

Toothpaste use commenced below the age of 1.5 years for 287 children (49.5%) and after that age for 244 children (41.9%). Information was not provided for 51 children (8.8%). Low fluoride toothpaste was reported to have been used by 127 children (21.8%) and regular toothpaste by 66.8% of the children while aged under 6 years. Fluoride supplements had been used by 40 (6.9%) of participants, not used at all by 539 participants (92.6%) and no information was available for 3 persons (0.05%). Almost all supplement users were residents of the nonfluoridated areas (Table 1).

Table 1. Use of fluoride supplements by children ($n = 582$) resident in Perth (fluoridated) and Bunbury (nonfluoridated) areas

	All		Perth Metro		Bunbury region	
Supplement use	*n*	%	*n*	%	*n*	%
Yes	40	7.0	2	0.5	38	18.4
No	539	92.6	370	98.7	169	81.6
No information	3	0.5	3	0.8		
	582	100.1	375	100.0	207	100.0

Table 2. Distribution of TF scores among children ($n = 582$) currently resident in Perth (fluoridated) and Bunbury (nonfluoridated) areas

	All		Perth Metro		Bunbury region	
TF score	*n*	%	*n*	%	*n*	%
0	476	81.8	293	78.1	183	88.4
1	85	14.6	66	17.6	19	9.2
2	19	3.3	15	4.0	4	1.9
3	2	0.3	1	0.3	1	0.5
Totals	582	100.0	375	100.0	207	100.0

Table 3 Distribution of DMFT scores among children ($n = 582$) currently resident in Perth (fluoridated) and Bunbury (nonfluoridated) areas

	All		Perth Metro		Bunbury region	
DMFT	*n*	%	*n*	%	*n*	%
0	480	82.5	309	82.4	171	82.6
1	57	9.8	39	10.4	18	8.7
2	28	4.8	19	5.1	9	4.3
3	8	1.4	4	1.1	4	1.9
4	9	1.5	4	1.1	5	2.4
Totals	582	100.0	375	100.1	207	99.9

Fluorosis and caries

The distribution of TF fluorosis scores for boys and girls was almost identical and the two sexes were combined for analysis. Overall, 18.2% of participants had some degree of dental fluorosis. Prevalence among persons currently resident in the fluoridated area was 20.68% and among persons resident in the nonfluoridated areas it was 15.1%. People resident in the Perth region as a child (birth − 4 years) were more likely to have some fluorosis than persons then resident in nonfluoridated areas and this difference was statistically significant (21.9% versus 11.6%, $P < 0.05$ ANOVA) but almost all those recorded as having fluorosis had TF score 1 (Table 2).

The overall prevalence of permanent tooth caries was 17.5%. Mean caries experience was 0.3 DMFT. Caries experience in girls was marginally higher than in boys (0.318 versus 0.278 DMFT) but the difference was not statistically significant. Caries differences between the fluoridated and nonfluoridated areas were very slight (Table 3) and not statistically significant (e.g. DMFT $P > 0.4$, ANOVA). The range of DMFT scores was 0–4.

Analyses

In bivariate analysis, no relationships were found between the presence of fluorosis and the age of commencement of toothpaste use, reported swallowing of toothpaste, reported liking of toothpaste, the duration of breast feeding and the duration of formula use (in all cases, $0.2 < P < 0.8$). Fluoride supplement use was not associated with the presence of fluorosis in bivariate analysis ($\chi^2 = 0.111$, d.f. = 1, $P > 0.7$) but residence in a fluoridated area from birth to 4 years of age showed a strong bivariate association ($\chi^2 = 9.45$, d.f. = 1, $P < 0.0025$).

There were 169 participants who had not lived in a fluoridated area between birth and 4 years of age, and who had not reported using fluoride supplements. Nineteen (11.2%) of these were scored as having dental fluorosis (15 had TF 1, 3 had TF 2 and 1 had TF 3). No fluoride-related variable was statistically significantly associated with this finding.

The only statistically significant risk factor identified using multiple logistic regression analysis was residence in a fluoridated area from birth to 4 years of age (Table 4).

Discussion

When measurements are made over time, good examiner reliability is the key to a valid comparison of prior and current findings. In the original 1989/90 fluorosis study (1), clinical re-examination

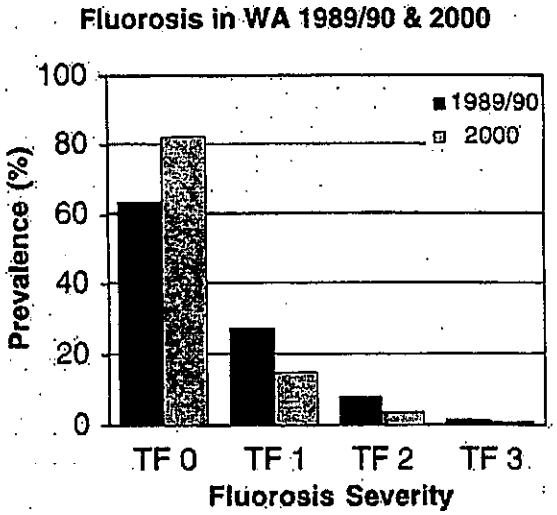

Fig. 1. Comparison of distribution of fluorosis severity (TF) scores between children examined in 1989/90 (n = 659) and children examined in 2000 (n = 582).

of 50 participants yielded a kappa score of 0.78 which is characterised as 'substantial' agreement (29). Good quality colour slides of almost all the 1989/90 cases were available and it has been documented that the use of photographs is a valid method for assessing dental fluorosis (30, 31). Re-examination of 80 of these photographs in 2000 and comparison of the recording with the results of the 1989/90 clinical examinations yielded a kappa score of 0.75, categorised as 'substantial' agreement. On this basis, it seems warranted to compare the current findings with the 1989/90 findings. Nevertheless, the possibility of examiner bias is present and it would be desirable to have different examiners corroborate the current findings.

The principal finding of interest in this study is the substantial reduction in the prevalence of dental fluorosis compared with the situation in 12-year-old-children 10 years previously. The overall preva-

Table 4 Logistic regression coefficients, odds ratios and 95% confidence intervals for predicting the presence of dental fluorosis

			95% Confidence Interval	
Exposure term	Coefficient	Odds Ratio	Lower	Upper
Constant	−2.60			
Residence F Area[a]	0.72	2.06	1.21	3.50
Other F Exposure[b]	0.58	1.79	0.21	14.80
F Supplement[c]	0.03	1.03	0.37	2.88

Reference exposures were: [a] No residence in a fluoridated area birth-4 years of age;[b] No other early fluoride exposure; and,[c] No use of fluoride supplements before 4 years of age.

lence then was 0.37 against 0.18 in the current study. The magnitude of the reduction was unexpected but it is supported by information on the use of fluoride supplements: in 1989/90, 12% of children were reported to have used supplements between birth and the age of 4 years (1), whereas in the present study only 6.9% had done so ($P < 0.001$). In 1989/90, low fluoride toothpaste had just arrived on the Australian market but it could not have affected the teeth then examined. In 2000, the teeth of interest in the study would have mineralised approximately in the period 1992–95, a period during which one low fluoride toothpaste was well established on the market and others were being introduced. Recalled information on early toothpaste use by children is known not to be reliable (32) and this may explain why the use of low-fluoride toothpaste did not figure as a protective factor in the quantitative analysis. Direct observation in the local supermarkets confirms that children's low-fluoride toothpastes are prominently promoted today.

As a result of the 1989/90 study findings, consideration was given to advising that the water fluoride concentration in the southern part of Western Australia (where the present study was undertaken) be lowered. For the period in which the teeth of interest in these studies were mineralising, the target water fluoride concentration remained about 0.85 mg/L although there probably have been fluctuations (33). A similar problem of high fluorosis prevalence in Hong Kong was tackled by reducing water fluoride concentration from 1.0 to 0.7 mg/L, which brought about a reduction in fluorosis prevalence from 0.64 to 0.47 (8). The lower initial water fluoride concentration in Western Australia, coupled with the fact that despite the relatively high fluorosis prevalence recorded, most children had no dental fluorosis even though the recording tool, the TF index, identifies fluorosis at a very early stage on dried teeth, argued for not changing the water fluoride concentration initially. The identified risk factors for fluorosis in 1989/90 listed supplement use as a more important risk factor for fluorosis than area of residence, and at that time a number of authors were querying the usefulness of fluoride supplements as public health measures, querying the then current dosage schedules, and attempting to balance their caries preventive effect against the risk of dental fluorosis (10, 11, 13, 34–37). It was therefore decided in Western Australia to attempt to discourage the ingestion of discretionary fluorides such as supplements and toothpastes. In the case of toothpaste, ingestion could not be totally eliminated in children under six years of age (38) because the use of toothpaste was considered desirable; therefore the use of products with lower fluoride concentrations was encouraged. This decision has recently been corroborated by Pendrys (39), who estimates that up to two-thirds of fluorosis prevalence in US fluoridated areas, and one-third in nonfluoridated areas, can be attributed to inappropriate use of discretionary fluorides.

The teeth examined in the present study would have mineralised fairly soon after several of these decisions on fluoride exposure were made, approximately 1992–95. This may account for the fact that the present study reports some continued use of supplements by participants, and some continued use of full strength fluoride toothpaste. New advice given by the staff of the dental services and private dental practitioners probably did not reach all parents by these dates. It seems therefore likely that in five years time the modified recommendations will have reached a greater proportion of parents and if so, fluorosis prevalence should be even lower.

Insofar as fluoride supplements contribute to caries prevention, a contentious question, their reduced use should entail a greater risk of dental caries. The 12-year-old children examined in 1989/90 had caries experience levels of 0.89 DMFT in the Perth region and 1.57 DMFT in the Bunbury (nonfluoridated) region (26). In the present study, the corresponding caries experience scores were 0.31 (SD 0.75) DMFT in the fluoridated area and 0.28 (SD 0.77) DMFT in the nonfluoridated area, but since the current data are derived from 10-year-olds in 2000 against 12-year-olds in 1989/90, these figures are not directly comparable. Australian Child Dental Health Survey (40) data for 10-year-olds in all of Western Australia in 1990 reported a DMFT value of 0.84 for 10-year-olds, substantially greater than reported in this study for 2000. The major part of the Western Australia population lives in the Perth and Bunbury regions, so these data suggest that caries levels have continued to decline despite the reduced use of fluoride supplements. The very small difference between caries levels in the two regions (0.31 versus 0.28 DMFT) should not be ascribed great importance: the teeth at greatest caries risk in 10-year olds, the first permanent molars, would have been exposed in the mouth about 2 1/2 years, a period during which few carious cavities would have become established under today's conditions.

The prevalence of dental fluorosis has been reported to be unexpectedly high in many countries since the late 1980s. This has largely been ascribed to water fluoridation because generally the highest

prevalences have been reported from fluoridated regions, and community fluorosis prevalence even has been considered to be irreversible (41). However, fluoride intake comes from many sources which cumulate to produce fluorosis if ingestion occurs when teeth are mineralizing. The findings of this study suggest that dental fluorosis can be brought under control at a population level by eliminating discretionary fluoride intake from supplements and by reducing the possibility of intake from toothpaste, without apparent adverse changes in the impact of dental caries. The study should encourage other communities to consider limiting discretionary fluoride intake to levels that prevent disease but 'produce only sporadic instances of the mildest forms of dental fluorosis of no practical esthetic significance' (42).

Acknowledgements

Data analyses for this study were done while the author was visiting Associate Professor at the Faculty of Dentistry, Université d'Auvergne, Clermont Ferrand, France and the author acknowledges with gratitude the assistance of colleagues there.

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Fluoride Supplementation for Children: Interim Policy Recommendations

Committee on Nutrition

Pediatrics 1995;95;777

AMERICAN ACADEMY OF PEDIATRICS

Fluoride Supplementation for Children: Interim Policy Recommendations

Committee on Nutrition

The Committee on Nutrition of the American Academy of Pediatrics (AAP) last issued a statement in 1986 on the topic of fluoride supplementation for children. The recommendations made at that time recently have been reassessed because of what seems to be an increased incidence of dental fluorosis in children living in the United States. Dental fluorosis appears during tooth formation and is caused by excessive fluoride ingestion, which leads to enamel protein retention, hypomineralization of the dental enamel and dentin, and disruption of crystal formation. The effect is cosmetic only, ranging from barely perceptible white striations or specks to confluent areas of pitting or brownish gray staining. Teeth affected by fluorosis seem to continue to be resistant to dental caries.

The main sources of fluoride include fluoridated water, foods or drinks reconstituted or prepared with fluoridated water, dentifrices, and fluoride supplements. Water is not fluoridated to a uniform level throughout the United States, and young children ingest significant but variable amounts of fluoride while brushing their teeth with fluoride-containing toothpaste. Because both of these sources of fluoride are difficult to control, attention has been directed again at the dosage of fluoride supplements to attempt to prevent dental fluorosis.

In January 1994, a Dietary Fluoride Workshop sponsored by the American Dental Association was convened to address the issue of dental fluorosis. Although children can receive substantial amounts of fluoride from beverages and dentifrices, the experts at this workshop thought the only source of fluoride that could be easily altered was the supplement prescribed by physicians and dentists. The participants at the workshop recommended the schedule for fluoride supplementation given in the Table.

These recommendations for fluoride supplementation represent a modification of those adopted in 1979[1] and reaffirmed in 1986.[2] Fluoride supplementation is no longer recommended from birth, and doses have been decreased during the first 6 years of life. The level of water fluoride content when supplements are not needed has been lowered from 0.7 to 0.6 ppm. Recently the American Dental Association Council on Dental Therapeutics affirmed the recommendations adopted at the workshop. A council report of the workshop will appear in a future issue of the *Journal of the American Dental Association*, and the proceedings will be published in the *Journal of Public Health Dentistry*. The AAP concurs with these dosage recommendations, and its Committee on Nutrition is proceeding with a complete revision of the 1986 AAP policy statement entitled "Fluoride Supplementation." These recommendations supersede those contained in the 1986 statement and republished in the 1993 AAP *Pediatric Nutrition Handbook*.

TABLE. Fluoride Supplementation*

Age	Water Fluoride Content (in ppm)		
	<0.3	0.3–0.6	>0.6
Birth–6 mo	0	0	0
6 mo–3 y	0.25	0	0
3–6 y	0.50	0.25	0
6–16 y	1.00	0.50	0

* Fluoride daily doses are given in milligrams.

COMMITTEE ON NUTRITION, 1994 TO 1995
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Rudolph L. Leibel, MD
John N. Udall, MD

The recommendations in this statement do not indicate an exclusive course of treatment or procedure to be followed. Variations, taking into account individual circumstances, may be appropriate.

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Fluoride Supplementation for Children: Interim Policy Recommendations

Committee on Nutrition

Pediatrics 1995;95;777

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FOR IMMEDIATE RELEASE

COMMON COUNCIL CITY CLERK COMMUNICATIONS CENTER
CITY OF MILWAUKEE
NEWS RELEASE

FOR INFORMATION CALL

May 27, 2012

Ald. Jim Bohl
(414) 286-2221

News conference set for fluoridation ending proposals

Alderman Jim Bohl will hold a news conference **Tuesday** (**May 29**) at City Hall to discuss his resolution that seeks to end the fluoridation of Milwaukee's drinking water – and a companion resolution that would create a grant program to provide dental care services to those in Milwaukee who have either no access to dental care or simply cannot afford it.

The news conference will begin at 11 a.m. in the first floor rotunda at City Hall, 200 E. Wells St.

The practice of fluoridating Milwaukee's water began in 1953 prior to fluoridated toothpastes and mouthwashes being readily available to the public. Alderman Bohl's primary resolution orders "the immediate cessation and prohibition of the introduction of sodium silicofluoride or any fluoride compound into water distributed by the Milwaukee Water Works," and if approved, the measure would bring Milwaukee in line with an increasing number of cities that are ending the practice of fluoridation.

The resolution is scheduled to be heard by the Common Council's **Steering and Rules Committee** during its meeting on Thursday, May 31 at 1:30 p.m. in room 301-B at City Hall.

Alderman Bohl's statement about his primary resolution to end fluoridation is attached, as are copies of both legislative files.

-More-

Fluoridation measures/ADD ONE

Alderman Bohl said the companion dental care services proposal – file #120188 – would take the $540,000 the Milwaukee Water Works uses to add fluoride to the city's water, and instead use those funds to provide critical dental care to those who need it. He said Milwaukee needs a local solution to the rampant cavity problem that exists in lower income neighborhoods, where kids have more cavities despite having fluoridated water.

"My proposal would take the money that the city uses to put toxic fluoride in the water and instead put it to something useful," he said.

Please go to Alderman Bohl's web page – www.milwaukee.gov/district5 – to see a complete package of information on his effort to end fluoridation of Milwaukee's water.

-30-

COMMITTEE CHAIR
• Zoning, Neighborhoods and Development

COMMITTEE MEMBER
• Steering and Rules
• Public Works
• Public Safety

JAMES A. BOHL, JR.
Alderman, 5th District

Fluoride in Milwaukee's Water Unsafe, Unnecessary and Unhealthy: Alderman Jim Bohl proposes legislation to stop Milwaukee's Fluoridation Program

Statement from Alderman Jim Bohl
May 22, 2012

New research shows that ingesting fluoride delivers health risks without benefit of less tooth decay which makes water fluoridation obsolete, unhealthy and a waste of money, and that is why I introduced legislation today calling for the end of Milwaukee's water fluoridation program.

Fluoride chemicals have been added to Milwaukee's water supply since 1953 when it was believed that ingested fluoride incorporated into children's developing teeth would resist cavities. But science disproved that theory.

There is little convincing scientific evidence that swallowing fluoride actually reduces tooth decay. According to World Health Organization figures there is virtually no difference in tooth decay in 12-year-olds whether they have grown up in fluoridated or non-fluoridated countries. The Centers for Disease Control (CDC) says that fluoride hardens teeth from topical application and swallowing fluoride can lead to fluoride overdose symptoms such as dental fluorosis (yellowing, brown, white spotting or pitted teeth). Fluorosis rates increased from 22% of teens in 1986 to an alarming 41% in 2010, according to CDC statistics. If fluoride works topically why are we forcing people to swallow it, especially those who after doing their own research on the issue have emphatically stated that they do not wish to drink fluoridated water? If any one wants fluoride in Milwaukee they can buy fluoridated toothpaste and brush it onto their teeth and spit it out. Milwaukee shouldn't be playing either doctor or dentist to our citizens. We are not qualified to do this and those who urge us to continue accept no legal responsibility for any harm it may be causing.

-More-

City Hall Room 205 • 200 East Wells Street • Milwaukee, WI 53202
PHONE (414) 286-3870 • **FAX** (414) 286-3456 • **E-MAIL** jbohl@milwaukee.gov • **WEBSITE** www.milwaukee.gov/district5

It's accepted that fluoride can also damage bones, but no studies have researched the bone damage caused to children with fluoride-induced tooth damage. The condition of teeth and bones may be the least of our worries, however. There have now been 26 published studies that indicate an association between fairly modest exposure to fluoride and lowered IQ in children. Promoters tell us that the level we add to water is very small (1.1 milligram per liter or 1.1 ppm) but that is actually greater than 250 times the level of fluoride that occurs in mothers' milk (0.004 ppm, National Research Council, 2006, p.40).

Further, the prestigious National Research Council's (NRC) 2006 fluoride report revealed that fluoride, even in low doses, can harm the thyroid gland, kidney patients, babies and high water drinkers. They also report studies linking fluoride to osteosarcoma (bone cancer). A study conducted at Harvard in 2001 and published in 2006 showed that young boys exposed to fluoridated water in their 6th to 8th years had a 5-7 fold increased risk of succumbing to osteosarcoma by the age of 20. Despite promises that this study would be refuted, no published study has done so.

As a result of the afore mentioned NRC report, government, health and dental organizations now advise against routinely mixing fluoridated water into infant formula to avoid dental fluorosis.

The National Kidney Foundation is among a growing list of organizations that has withdrawn its support of water fluoridation saying, "Individuals with CKD [Chronic Kidney Disease] should be notified of the potential risk of fluoride exposure." The American Dental Association itself now admits fluoride is a concern to all kidney patients, not just those on dialysis.

Fluoride is not a nutrient or essential for healthy teeth. Fluoride is regulated as a drug in toothpaste by the FDA. Fluoride supplements have never been FDA safety tested and it classifies fluoride as an "unapproved drug." The EPA regards the type of fluoride used to supplement water supplies as a contaminant. Even the CDC reports on its website saying, "It is not CDC's task to determine what levels of fluoride in water are safe." If it is not the CDC's task, and the FDA refuses to do it, then whose task is it? Is the Milwaukee Common Council expected to do this?

While the less toxic calcium fluoride does appear in some waters naturally (.17 ppm in Lake Michigan water), Milwaukee adds hydrofluosilicic acid, a waste product of phosphate fertilizer manufacturing captured in its smokestacks, trucked as hazardous waste then injected unpurified into the water supply. These industrial grade fluoridation chemicals contain trace amounts of lead, arsenic, mercury, and other toxins. Arsenic is a known human carcinogen for which there is no safe level. By using these fluoridating chemicals we are knowingly increasing the cancer risk for our citizens. Why should we take these risks to reduce a negligible amount of tooth decay? Such a policy is reckless to say the least.

-More-

The ADA continues to actively promote the expansion of water fluoridation programs at the same time the CDC admits growing numbers of kids are fluoride-overdosed. National statistics show that tooth decay has grown in numbers and severity in toddlers more recently, and untreated tooth decay has grown in many age groups. The Pew Foundation reports “preventable dental conditions were the primary reason for 830,590 ER visits by Americans in 2009—a 16 percent increase from 2006.”

Americans are fluoride-overdosed and dentist-deficient. The lack of accessible, affordable dental care to many Milwaukee and Wisconsin residents has been widely reported. With no evidence, some claim that fluoridation helps the poor the most. Actually, the poor and malnourished are the most harmed by fluoride. They also are least in a position to afford expensive filtration systems required to remove fluoride from water, making this an environmental justice situation. According to civil rights leader and former mayor of Atlanta, Andrew Young, “I am most deeply concerned for poor families who have babies: if they cannot afford unfluoridated water for their babies’ milk formula, do their babies not count?”

We have served as guinea pigs in this ongoing and failed experiment for far too long. In my position as Alderman, it is my duty to promote the health, safety and welfare of all our residents. Adding fluoride chemicals into our public water supply runs counter to this and therefore needs to end.

-30-

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Fluoride increases lead concentrations in whole blood and in calcified tissues from lead-exposed rats.

Sawan RM, Leite GA, Saraiva MC, Barbosa F Jr, Tanus-Santos JE, Gerlach RF.

School of Dentistry of Ribeirao Preto, University of Sao Paulo (FORP/USP), Av do Café s/n, 14040-904, Ribeirão Preto, SP, Brazil.

Higher blood lead (BPb) levels have been reported in children living in communities that receive fluoride-treated water. Here, we examined whether fluoride co-administered with lead increases BPb and lead concentrations in calcified tissues in Wistar rats exposed to this metal from the beginning of gestation. We exposed female rats and their offspring to control water (Control Group), 100mg/L of fluoride (F Group), 30mg/L of lead (Pb Group), or 100mg/L of fluoride and 30mg/L of lead (F+Pb Group) from 1 week prior to mating until offspring was 81 days old. Blood and calcified tissues (enamel, dentine, and bone) were harvested at day 81 for lead and fluoride analyses. Higher BPb concentrations were found in the F+Pb Group compared with the Pb Group (76.7+/-11.0microg/dL vs. 22.6+/-8.5microg/dL, respectively; $p<0.001$). Two- to threefold higher lead concentrations were found in the calcified tissues in the F+Pb Group compared with the Pb Group (all $p<0.001$). Fluoride concentrations were similar in the F and in the F+Pb Groups. These findings show that fluoride consistently increases BPb and calcified tissues Pb concentrations in animals exposed to low levels of lead and suggest that a biological effect not yet recognized may underlie the epidemiological association between increased BPb lead levels in children living in water-fluoridated communities.

PMID: 20188782 [PubMed - indexed for MEDLINE]

From: Witkowski, Terry
Sent: Thursday, May 24, 2012 5:08 PM
To: Black, Tobie
Subject: FW: water fluoride
Please send a hearing notice

From: toothmedic@aol.com [mailto:toothmedic@aol.com]
Sent: Wednesday, May 23, 2012 1:36 PM
To: Witkowski, Terry
Subject: water fluoride

Dear Mr. Witkowski,

Next week a measure introduced by Alderman Jim Bohl on the removal of fluoride from the community water system is going to be discussed. I cannot say enough this effort if very misguided and will cost the city of Milwaukee and it's residents increased health care costs cause more dental health problems

The Centers for Disease Control and Prevention calls community water fluoridation one of the greatest health advancements of the 20th century. Studies show that for every $1 invested in community water fluoridation saves $38 in dental treatment costs. There are studies that show decay in kids and young adults can be reduced by 30-50%.

I am a T19 provider and see many children from the city of Milwaukee and I can tell you decay is prevalent now and if you remove the fluoride from the community water system I may have to get off the system as I truly think the system will be overloaded with an increased amount of cavities. I would rather see you try to pass measures that will educate both parents and children on the hazards of junk foods and drinks that we also face today.

Thank you for your consideration in this matter

Dr. Paul S. Levine
toothmedic@aol.com
414-355-0213

From: Witkowski, Terry
Sent: Thursday, May 24, 2012 5:08 PM
To: Black, Tobie
Subject: FW: WDA on fluoridation
Please send a hearing notice

From: Evan Zeppos [mailto:ezeppos@zeppos.com]
Sent: Thursday, May 24, 2012 9:23 AM
To: Hines Jr., Willie; Bauman, Robert; Hamilton, Ashanti; Zielinski, Tony; jdavis@milwaukke.gov; Witkowski, Terry
Subject: WDA on fluoridation

The following is from my client, the Wisconsin Dental Association.

Of particular interest is the scientific info.

Please let me know if you have any questions, need more info or would
like to discuss this issues with credentialed and credible experts.

Some of you know local health care professional Dr. Ernestine Willis, a strong supporter of continued fluoridation. I encourage you to engage her in a discussion on this issue.

ez
For WDA

Community Water Fluoridation Facts

Overview

For 65 years, community water fluoridation has been a safe, cost-effective and healthy way to significantly prevent tooth decay in children and adults.

The Centers for Disease Control and Prevention has proclaimed community water fluoridation as one of the 10 great public health achievements of the 20th century, along with greater use of vaccinations and recognition of tobacco use as a health hazard.

According to 2012 figures from the CDC, 73.9 percent of the U.S. population on public water systems – 204 million people – had access to optimally fluoridated water. Approximately 90 percent of the population in Wisconsin that is connected to public water supplies receives the benefit of appropriate levels of fluoride.

Dental decay

- Tooth decay is the disease process that causes the destruction of tooth structure.

- Tooth decay is five times as common as asthma and seventimes as common as hay fever in 5 to 17 year-olds.

Fluoridation works

- Studies prove water fluoridation continues to be effective in reducing dental decay by 30-50 percent for children and adolescents, even in an era with widespread availability of fluoride from other sources, such as fluoride toothpaste. Decay is reduced by 27 percent for adults.

- Fluoridation benefits people of all ages and incomes, but it isespecially important for those without access to regular dental care. An estimated 51 million school hours and 164 million work hours are lost per year in the United States due to dental-related illness.

Fluoridation is safe

- Fluoride is naturally-occurring in most water sources and is minimally adjusted to maximize proven oral health benefits.

- In January 2011, the U.S. Department of Health and Human Services proposed changing the recommendedoptimal level of fluoride in public water systems to 0.7 milligrams per liter of water. The proposed recommendation is based on extensive research and shows federal health officials are using the best science to update their recommendations on fluoridated water.

Fluoridation is cost-effective

- Studies show that every $1 invested in community water fluoridation results in $38 saved in dental treatment costs.

- The average cost for a community to fluoridate its water is estimated to range from approximately $3.00 a year per person in small communities to just $0.50 a year per person in large communities.

The following organizations support appropriate fluoridation in Wisconsin's public water systems:

Children's Health Alliance of Wisconsin

Children's Hospital of Wisconsin

Marquette University School of Dentistry

Wisconsin Department of Health Services

Wisconsin Dental Association

Wisconsin Oral Health Coalition

American Dental Association

National Dental Association (forum for minority dentists)

May 24 2012

Milwaukee City Council Members May 31, 2012

Greetings to you all. My name is Kevin Boehm, a general dentist and certified nutrition consultant based in the West and Southwest suburbs of Chicago. Sadly, today I am unable to attend this meeting due to my youngest daughter's graduation. However, I wish to help stand in support of Councilman Bohl's petition with respect to removal of fluoride from the city's drinking water supply.

I must tell you that during my years in dental school, and for several years after my graduation, I would have thought that the removal of fluoride from drinking water was an act of lunacy. However, during my eighteen years in practice, I have learned quite a few contradictory things that have changed my entire viewpoint on the forced fluoridation of drinking water issue. Along with a growing number of health professionals, I have looked at the chemicals used in the fluoridation process, their associated risks, and their toxicity profiles with respect to effects on the human body's enzymes and hormones. I no longer believe that the process of fluoridation is in the best interest of the general public and have not for quite some time.

First, I would like to quickly approach the subject from its nutritional aspects. In my study of over fifty different text books and related material, I can find no reference anywhere that remotely pertains to the human body's biologic need for fluoride. The body needs essential vitamins, fatty acids, amino acids, and minerals to continue its life sustaining work, but nowhere does fluoride serve an essential function in the human body's biochemical processes. Your body cannot live without calcium, but it can live without fluoride.

Aside from the fact that fluoride/fluorine is not an essential nutrient, it performs many deleterious functions once inside the body, when ingested systemically. Fluoride reduces Kreb's cycle, or citric acid cycle efficiency, reduces effectiveness of body enzymes, reduces thyroid gland efficiency, and may well increase risk of certain cancers and bone fractures. Fluorine, as part of the periodic table of elements, is similar in its properties to other halides such as bromine, iodine, chlorine, and astatine, except it's much smaller in atomic weight. It is the smallest of the halides and the most aggressive in its binding strength in many ionic forms.

In the citric acid cycle, for example, if fluoride binds to acetyl-coA to form fluoroacetyl-coA this molecule blocks the enzyme Aconitase, which causes citrate to accumulate in the body. Essentially, this halts the normal ATP production in cellular mitochondria, which is how the body produces energy on a cellular level. A second example would be the binding of fluoride instead of iodine in the synthesis of thyroid hormone. Active functioning thyroid hormone consists of a centrally located tyrosine molecule surrounded by four iodine molecules. If fluorine is bound instead of iodine, due to its stronger binding strength properties, a molecule of thyroid hormone will be synthesized and will show up on a blood test. Sadly, it will not bind to the proper receptors in the body, rendering it useless due to its changed molecular size and bond angle discrepancies. These are only two simple examples out of potentially hundreds to thousands of ordinary chemical reactions within the body that can be affected by fluoride.

Consider for a moment the concentrations the human body uses for hormone effectiveness and the concentration of fluoride in a dosage of drinking water. All human hormones are measured

in blood or salivary tests using either picograms, 10^{-12}, or nanograms, 10^{-9}, per cc normally. At these concentrations the hormones are effective on their targets, run their course, and are allowed to dissipate. Fluoride at a concentration as small as one PPM, the normal drinking water concentration of 10^{-6}, is anywhere from one thousand to one million times more concentrated that our own hormones. That certainly seems to be a factor worth consideration.

There are also issues related to the form of fluoride used in water fluoridation methods. The American public has been misled as to what has been added to the water supply. Only about ten percent of the water being fluoridated contains sodium fluoride, and about ninety percent of the water is treated with hexafluorosilicic acid. The latter compound is a toxic by-product of the fertilizer industry that comes from a process that captures two toxic gases, hydrogen fluoride (HF) and silicon tetrafluoride (SiF_4), through water spray during phosphate manufacture. As far back as 1975, the U.S. EPA began mandating reclamation of the phosphate fertilizer industry's waste products noted above, and this became their eventual solution. What is more interesting to note is that these fluoride containing compounds may contain arsenic, and due to their acidic nature may leech lead out of brass fittings or solder used in water fixtures and water lines. The world seems to have a fairly good grip on lead and its effects on brain development in young people, and arsenic has its cancer related issues. This seems to be a very unnecessary health risk. When you consider that likely ninety-eight percent of fluoridated water is used for laundry, bathing, watering lawns, and dishwashing, the obvious conclusion is that very little is actually consumed. So why does this practice continue? There seem to be far better uses for our tax dollars than fluoridating our dishwater.

Some other interesting history of fluoridation came in 1999. The U.S. Centers for Disease Control (CDC) finally conceded that fluoride's main action occurred topically not systemically. A brief excerpt from that CDC report is as follows:

> Fluoride's caries preventing properties were initially attributed to changes in enamel during tooth development because of the association between fluoride and cosmetic changes in enamel and a belief that fluoride incorporated into enamel during tooth development would result in a more acid-resistant mineral. However, laboratory and epidemiologic research suggests that fluoride prevents dental caries predominantly after eruption of the tooth into the mouth, and its actions primarily are topical for both adults and children.[1]

There are a couple ideas that come to mind upon closer inspection of data on both sides of this issue. Upon the day of graduation, a physician recites the Hippocratic Oath. One line resonates quite loudly on this subject, which loosely translated says, "First, do thy patient no harm". Others have stated, "If in doubt, leave it out" in relation to fluoride usage. I am not God, and nor would I like to think that I am all seeing and all knowing. I am a concerned physician who tries to look at the big picture and thinks caution would be a better way to look at this subject instead of a rubber stamp. Most people, if they had their choice, would elect to have informed consent

[1] Centers for Disease Control and Prevention. "Achievements in Public Health, 1900-1999: Fluoridation of Drinking Water to Prevent Dental Caries". *Mortality and Morbidity Weekly Review* 48, 41 (Oct. 22, 1999): 933-40. http://www.cdc.gov/mmwr/preview/mmwrhtml/mm4841a1.htm

in matters related to their health. It appears that there is a complete lack of informed consent as it relates to the unknown dispensation of hexafluorosilicic acid into their water in concentrations one thousand to one million times more concentrated than a human hormone. One should not be able to conduct such activity against another's wishes. Why should informed citizens be forced to purchase expensive water filtration equipment to remove an unwanted and harmful chemical additive from water systems from which they are forced to purchase water? Should this not be a personal decision freely elected by a consenting individual? If an individual has been made aware of the potential risks and dangers involved with fluoride usage and chooses to use it, that would be perfectly acceptable for that specific individual. However, this should be done by individuals for their own personal consumption topically or systemically with full knowledge of their undertaking prior to consumption. This should not be done as a mass science experiment using by products of questionable safety and expose non-consenting individuals in the process. Freedom of choice should reign in matters such as these.

We should leave the dental aspect of this as a separate issue. The CDC has deemed fluoride's "best" effects to be topical. Drinking water fluoridation is systemic and a very different issue with a huge host of potential biochemical complications. Is it truly worth the risk? In our current situation America spends far more than any nation on Earth on healthcare. More and more drugs are dispensed, all having side effects which, when combined with other drugs, complicate matters even further. Often when drugs are taken in combination, unforeseen complications occur. Could fluoride and bromide usage from industrial applications be creating unforeseen new compounds when combined with prescription drugs, due to its high binding affinity? Could we be creating patients with low thyroid function among a host of other possible imbalances? Is this why we're spending so much more than any other nation on Earth, due to unforeseen drug interactions or changes in drugs due to addition of other compounds, that were never tested together before? A host of concerned physicians ponder that very question now, perhaps more than ever.

I would like to take a moment to thank Dr. Paul Connett, Dr. James Beck, and Dr. Spedding Micklem who have done more research on this subject than I could hope to do in two lifetimes. They made me look harder at the systemic possibilities of fluoridation than I had early in my career. I am grateful for your work.

For those of you who must decide how best to serve Milwaukee's citizens, there are a number of websites where you can educate yourselves about fluoride. One, http://poisonfluoride.com/pfpc/index.html, shows how frighteningly similar fluoride poisoning and hypothyroidism are in presentation. Another, www.fluoridealert.org, covers numerous fluoride related topics and keeps visitors abreast of other communities who have eliminated water fluoridation or are contemplating doing so. The Swedish government and people rejected fluoridation in the 1970's, and their children have not suffered greater incidence of tooth decay. I think it is worth a few hours of your time to do some research for the greater good of your health. I have also enclosed a report prepared for the Department of Health and Human Services by the IAOMT (International Academy of Oral and Maxillofacial Toxicology, www.iaomt.org) from February 2011, by Dr. Kathleen Theissen, PhD, that is well worth the read. It may be a bit technical in jargon, but it is very informative and well written. I wish you all well in your deliberations.

Best Regards,
Kevin M. Boehm DDS, CNC
President Holistic Dental Association

League of United Latin American Citizens

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June 1, 2012

Milwaukee Common Council
City Hall
Milwaukee, WI

Dear Alderman:

On behalf of the League of United Latin American Citizens (LULAC), our nations oldest and largest Hispanic membership based organization, I would like to thank you for reviewing the issue of drinking water fluoridation.

Since the late 1950s, it was generally accepted the municipal water fluoridation provided tremendous benefits to long term dental health. For this reason, without thorough testing, many states and countries implemented this practice with the of intentions. There was also a superficial correlation between water fluoridation and the improvement in dental health. However, in recent years many experts now believe the reason for this improvement is attributed to the wide spread availability of toothpaste with fluoride in the same period, as everyone, including dental experts agree fluoride is most effective when topically applied and not ingested.

Today, countless studies are now calling into question the benefits of fluoridation, and more importantly, a growing amount of data now suggests a link between increased general health risks and overexposure to fluoride.

As many in the Hispanic community rely on public drinking water to be safe, and as many have no economically feasible option to access non-fluoridated water, the issue of water fluoridation becomes one of forced medication, as well as one that may introduce potential health risks.

For this reason, the LULAC National Assembly adopted the following resolution regarding water fluoridation.

Thank you once again for your serious review of this important issue.

In shared commitment to the youth of Milwaukee,

Darryl D. Morin
Immed. Past State Director
& State Board Member

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WISCONSIN ORAL HEALTH COALITION
Children's Health Alliance of Wisconsin

Oral Health eBYTES

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MAY 2012
(Special Edition)

IMPORTANT DATES

May 31
Milwaukee City Council Steering & Rules Committee
Milwaukee, WI

June 29-30
Mission of Mercy
Madison, WI

September 11
WOHC Statewide Meeting
Stevens Point, WI

If you have an event you would like added to this list, please e-mail Alex Eichenbaum.

WOHC SPECIAL EDITION

URGENT CALL TO ACTION: Anti-fluoride legislation threatens oral health in metro Milwaukee

Milwaukee Alderman James Bohl is putting forward legislation to remove fluoride from the city of Milwaukee's public water system. This threatens the oral health of more than 861,000 people who live in Milwaukee and 16 other communities served by Milwaukee Water Works.

Your voice as an ongoing oral health partner of Children's Health Alliance of Wisconsin, Wisconsin Oral Health Coalition and Children's Hospital of Wisconsin is needed.

The city Steering and Rules Committee will hold a public hearing on Ald. Bohl's misguided proposal on Thursday, May 31, in Room 301-B at Milwaukee City Hall. Please consider coming to speak in support of continuing to fluoridate Milwaukee's water. If you are unable to attend, please email or call the committee members listed here before Thursday. Use these talking points to ensure we are sending a consistent

message on the safety and effectiveness of fluoride. Let them know the critical role community water fluoridation plays in oral health care. Educate local officials on the many proven positive effects of community water fluoridation and its safety. You may request that your comments be included in the city's official file.

For more information, contact Alex Eichenbaum at 414-292-4003.

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REVIEW ARTICLE

Effects of the fluoride on the central nervous system

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KEYWORDS
Fluoride;
Health;
Nervous system

Abstract
Introduction: Fluorine (F) is a toxic reactive element and exposure to it passes almost unnoticed with the consumption of tea, fish, meat, fruits, etc. and articles in common use such as: toothpaste additives; dental gels, non-stick pans and razor blades as Teflon. It has also been used with the intention of reducing dental caries.
Development: Fluoride can accumulate in the body, and it has been shown that continuous exposure to it causes damaging effects on body tissues, particularly the nervous system directly without any previous physical malformations.
Background: Several clinical and experimental studies have reported that F induces changes in cerebral morphology and biochemistry that affect the neurological development of individuals as well as cognitive processes, such as learning and memory.

F can be toxic by ingesting one part per million (ppm), and the effects are not immediate, as they can take 20 years or more to become evident.
Conclusion: The prolonged ingestion of F may cause significant damage to health and particularly to the nervous system. Therefore, it is important to be aware of this serious problem and avoid the use of toothpaste and items that contain F, particularly in children as they are more susceptible to the toxic effects of F.

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PALABRAS CLAVE
Flúor;
Salud;
Sistema nervioso

Efectos del flúor sobre el sistema nervioso central

Resumen
Introducción: El flúor (F) es un elemento tóxico y reactivo; la exposición al mismo pasa casi inadvertida con el consumo de té, pescado de mar, carnes, frutas, etc., y el uso de artículos como aditivo en pastas de dientes, enjuagues bucales, antiadherentes sobre sartenes y hojas de afeitar como el teflón. Asimismo, ha sido utilizado con la intención de reducir la caries dental.
Desarrollo: El F puede acumularse en el organismo y se ha demostrado que la exposición crónica al mismo produce efectos nocivos sobre distintos tejidos del organismo y de manera particular sobre el sistema nervioso, sin producir malformaciones físicas previas.
Fuentes: Diversos trabajos, tanto clínicos como experimentales, han reportado que el F provoca alteraciones sobre la morfología y bioquímica cerebral, que afectan el desarrollo neurológico de los individuos y, por ende, de funciones relacionadas con procesos cognoscitivos, tales como el aprendizaje y la memoria.

La toxicidad del F se puede presentar a partir de la ingesta de 1 parte por millón (ppm) y los efectos no son inmediatos ya que pueden tardar 20 años o más en manifestarse.
Conclusión: La ingesta prolongada de F provoca daños a la salud y de manera importante sobre el sistema nervioso central, por lo que es importante considerar y evitar el uso de artículos que contengan flúor y de manera particular en individuos en desarrollo, debido a la susceptibilidad que presentan a los efectos tóxicos del F.

Introduction

Fluorine (F) is a toxic, reactive element; exposure to F in human beings goes practically unnoticed thanks to the use of fluoride-containing compounds as additives in toothpaste (1,000 to 1,500 ppm), mouthwash (230-900 ppm of fluoride), in dietary supplements, and surface polymers found as non-stick surfaces in frying pans and razor blades[1] and the use of compounds with fluoride in the form of: industrial compounds, fertilizers, glass, oil refineries, fluorinated fuels, and others.[1] Thus, a significant proportion of F in the body comes from being exposed to it and from eating certain food with a naturally occurring high content in F, such as tea, sea fish, meats, eggs, fruits, and cereals. However, regular drinking water is the leading source of intake of this element.[1]

The water containing the highest concentration of fluorides corresponds to water resources located in mountainous regions or areas with geological deposits of marine origin, such as in Southeast Asia and Northeast Africa.[1] Studies conducted in the last 15 years show that a significant number of people in towns exposed to fluoridated drinking water suffer health issues and varying degrees of fluorosis.[2]

In Mexico, 5 million people (approximately 6% of the population) are affected by fluoride as a result of drinking groundwater,[2] often hydrothermal groundwater characterized by containing potentially toxic chemical elements, including F, and concentrations of up to 6.8 ppm have been detected. It is worth mentioning that the World Health Organization (WHO) recommends a concentration of 0.7 ppm for drinking water.[2]

The aim of this review is to set out information regarding the toxic potential of F and its effects on the nervous system, with special attention to populations exposed to the intake of this mineral at concentrations outside official guidelines.

Development

The main route for the incorporation of F into the human body is the digestive tract; 90% of the F ingested is absorbed in the stomach. In adults, some 10% of it is deposited in the bones, whereas in children, up to 50% is fixed to bone tissue. The maximum concentration of F in plasma is reached between 30 and 60 min after intake.[1]

In newborn children, close to 90% of the F absorbed is retained in the skeletal system. This affinity decreases with age and stabilizes. In children, around 50% of the F absorbed is fixed to the skeleton by the time the first stage of development has been completed and the remaining 50% is excreted through the kidney.[1]

Fluorine is capable of crossing the blood-brain barrier,[3] which can cause biochemical and functional changes in the nervous system during pregnancy since F accumulates in the brain tissue prior to birth.[4] Exposure to F during embryonic development has been reported to be related to learning disorders.[5] In this sense, other research mentions the consumption of large amounts of F as associated with decreased intelligence in children.[6] Studies carried out to evaluate the toxicity of F on neurodevelopment during pregnancy have demonstrated significant differences in neurobehavioural performance in newborns in areas that

are endemically rich in F compared to controls when assessing visual and auditory orientation reactions.[7] On the other hand, the levels of neurotransmitters such as norepinephrine, 5-hydroxytriptamine, and their receptors have been found to be decreased in the brain of aborted foetuses in areas that present cases of endemic fluorosis, while the level of epinephrine is higher than the concentrations found in subjects from areas where this problem does not occur. Hence, these results suggest that the accumulation of F in brain tissue can disrupt the synthesis of certain neurotransmitters and receptors in cells of the nervous system and may even go so far as to provoke neural dysplasia or other damage.[8] Likewise, F has been reported to have a specific effect on protein synthesis in the brain, entailing degenerative changes in the neurons, varying degrees of loss of grey matter, and changes in Purkinje cells in the cerebellar cortex;[9] moreover, it causes swelling of the mitochondria, granular endoplasmic reticulum, chromatin clumping, damage to the nuclear membrane, and a decrease in the number of synapses, mytochondria, microtubules, and synaptic vesicles, as well as damage at the synaptic membrane level. These changes indicate that F can delay cell growth and division in the cortex, and that the reduced number of mytochondria, microtubules, and vesicles in the synaptic terminal may reduce efficiency in neuronal connections and give rise to abnormal synaptic functioning and impact cognitive development during postnatal life.[4] Likewise, these changes might account for some of the neurological alterations present in patients with skeletal fluorosis, such as numbness in arms and legs, muscle spasms and pain, tetanus-like convulsions, and spastic paraplegia.[2] On the other hand, exposure to F increases the production of free radicals in the brain by activating different metabolic pathways related to Alzheimer's disease. On the experimental level, F has been seen to have an inhibitory effect on free fatty acids, in the brains of both male and female rats,[9] in addition to significant changes in the morphology of the hippocampus, the amygdala, the cortex, and the cerebellum.[10,11]

In this regard, animal studies have yielded information about the direct toxic effects of fluoride on brain tissue, including: decreased number of acetylcholine (ACh) receptors, lower lipid content, damage to the hippocampus and Purkinje cells, increased formation of β-amyloid plaques (classic cerebral anomaly in patients presenting Alzheimer's disease), exacerbation of lesions caused by iodine deficit, and accumulation of fluoride in the pineal gland.[12,13]

On the other hand, in studies using experimental models, the offspring of rats given a dose of 5, 15, or 50 ppm of F in their drinking water during gestations and lactation were seen to exhibit significantly high levels of the enzyme acetylcholinesterase 80 days after birth. The high activity of acetylcholinesterase might lower ACh levels and, given that said enzyme degrades the neurotransmitter ACh, it has an important impact on brain development.[14,15] ACh plays a part in regulating several different functions, such as the transition from sleep to wakefulness and processes that have to do with learning and memory, among others. At the level of the brain, there are precise mechanisms regulating its synthesis and release, which is important insofar as changes in the concentration of any neurotransmitter during development may have permanent neurological consequences that manifest in adulthood.[11]

Both learning and memory have been reported to be altered in mice treated with fluorinated water. The ability to learn has been found to be decreased in subjects who drink water with high concentrations of F in comparison with those who drink water containing a lower concentration of this element.[15]

Some studies performed in individuals who have been chronically exposed to F due to industrial contamination report that they have difficulties concentrating; certain aspects of their memory are altered, and they suffer fatigue and general malaise.[16]

For their part, studies carried out with humans in China shoed that a concentration of 3-11 ppm of fluoride in drinking water affects the functioning of the nervous system without causing physical malformations. The intelligence quotient (IQ) was assessed in children in communities in which there is a high degree of exposure to F (4-12 ppm) and it was found to be significantly lower than that of children living in communities with concentrations of close to 0.91 ppm.[6]

Another study conducted in children aged 6 to 8 years found poor visual spatial organization, which can affect reading and writing abilities; moreover, a concentration of 4.3 mg/creatinine was recorded in urine. In this regard, there have been reports that the levels of this chemical element are also high in the urine of people who drink water containing a high F content, which suggests that there is a relationship between the intake of F in drinking water, the concentration of F excreted in urine, and IQ.[17]

Some researchers suggest that the adequate intake of iodine might treat or counteract the toxic effect of F on the brain and IQ. On the other hand, in research performed in animals, a partial recovery has been seen in all the parameters studied when exposure to F is withdrawn; nevertheless, this recovery with respect to the toxic effects is more complete when ascorbic acid, calcium, or vitamin E are administered either individually or in combination, although recovery has been shown to be more effective with combination therapy.[18] Nonetheless, more studies are needed in this regard.

Conclusion

Fluorine is a chemical element found in high concentrations in the earth's crust. In many countries where the main source of drinking water is hydrothermal, F concentrations exceed those contemplated by the corresponding official regulations. Until now, the reports pose interesting controversies as to the role F plays in health. However, there are data showing that F has toxic effects on the central nervous system, depending on the dose administered, age, and exposure time; hence, it is recommended that the geographical location of a given population and the quality of the water they drink should be taken into consideration so as to take preventive measures for its use and, in areas where the fluoride concentration exceeds 0.7 mg/L, to avoid the intake of the drinking water, fluorinated salt, and the use of toothpastes and articles containing F.

Conflicts of interest

The authors declare no conflict of interest.

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May 31, 2012

VIA EMAIL and FAX (414) 286-3456; Page 1/6

Milwaukee Common Council Steering and Rules Committee
Council President Willie Hines, Chair
Ald. Robert Bauman
Ald. James Bohl
Ald. Joe Davis, Sr.
Ald. Ashanti Hamilton
Ald. Michael Murphy
Ald. Terry Witkowski
Ald. T. Anthony Zielinski

Proposed resolution ordering the immediate cessation and prohibition of the introduction of sodium silicofluoride or any fluoride compound into water distributed by the Milwaukee Water Works.

We are writing in staunch opposition of the above-referenced resolution proposing to cease fluoridation of the community water supply in Milwaukee and surrounding communities served by the Milwaukee Water Works.

All children and adults have benefitted from the practice of water fluoridation for over 65 years. The Centers for Disease Control and Prevention cite community water supply fluoridation one of the 10 greatest public health achievements of the 20th Century.

The practice is proven to be a safe and inexpensive way to reduce tooth decay for all members of the community, **especially in populations who struggle with access to health and dental care**, like many of your constituents. Simply by drinking fluoridated water or beverages and foods prepared with it, beginning at the age of 6 months, everyone has access to the benefits of fluoride protection.

We know that good preventive oral health, from birth, can determine the overall well-being and cost of providing health care for individuals over the course of their lifetimes. For every $1 invested in water fluoridation, approximately $38 is saved in future dental treatments.

In Milwaukee, its surrounding communities and elsewhere, fluoridating the community water supply is an important element of ensuring good oral health for everyone, not just those with regular access to preventive services. To end this practice would be pound-foolish from a cost standpoint. More critically, it would jeopardize the health of the approximately 850,000 people in 16 communities.

Other preeminent experts also weigh in strongly in favor of fluoridation, including:

American Academy of Pediatrics (AAP)
American Association of Public Health Dentistry (AAPHD)
American Dental Association (ADA)
American Medical Association (AMA)
American Public Health Association (APHA)
Association of State and Territorial Dental Directors (ASTDD)
Centers for Disease Control and Prevention (CDC)
International Association of Dental Research (IADR)
National Institute of Dental & Craniofacial Research (NIDCR)
World Health Organization (WHO)

We urge you to consider the ramifications of this proposal. Support the best possible public health for **all** your constituents, now and in the future.

Thank you for your consideration on this important issue.

Sincerely yours,

AMERICAN ACADEMY OF PEDIATRICS
Wisconsin Chapter

James A. Meyer MD, FAAP, FSAHM

James Meyer, MD, FAAP
President

Attachments:

(1) WIAAP Position Statement: Fluoridated Water
(2) American Academy of Pediatric Dentistry Fluoridated Water Policy

Policy on Use of Fluoride

Originating Committee
Liaison with Other Groups Committee

Review Council
Council on Clinical Affairs

Adopted
1967

Revised
1978, 1995, 2000, 2001, 2003, 2007, 2008

Reaffirmed
1977

Purpose
The American Academy of Pediatric Dentistry (**AAPD**), affirming that fluoride is a safe and effective adjunct in reducing the risk of caries and reversing enamel demineralization, encourages public health officials, health care providers, and parents/caregivers to optimize fluoride exposure.

Methods
A MEDLINE search was conducted using the terms "fluoride", "fluoridation", "acidulated phosphate fluoride", "fluoride varnish", "fluoride therapy", and "topical fluoride". Expert opinions and best current practices also were relied upon for this guideline.

Background
The adjustment of the fluoride level in community water supplies to optimal concentration is the most beneficial and inexpensive method of reducing the occurrence of caries.[1] Epidemiologic data within the last half-century indicate reductions in caries of 55 to 60% and recent data still show caries reduction of approximately 25%, without significant enamel fluorosis, when domestic water supplies are fluoridated at an optimal level.[2] Evidence accumulated from long-term use of fluorides has demonstrated that the cost of oral health care for children can be reduced by as much as 50%.[3] These savings in health dollars accrue to private individuals, group purchasers, and government care programs. An even higher caries reduction can be obtained if the proper use of fluorides is combined with other dietary, oral hygiene, and preventive measures[4,5] as prescribed by a dentist familiar with the child's oral health and family history.

A large body of literature supports the incorporation of optimal fluoride levels in drinking water supplies. When fluoridation of drinking water is impossible, effective systemic fluoridation can be achieved through the intake of daily fluoride supplements. Before supplements are prescribed, it is essential to review dietary sources of fluoride (eg, all drinking water sources, consumed beverages, prepared food, toothpaste) to determine the patient's true exposure to fluoride.[1,6-9] Fluoride content of ready to use infant formulas in the US and Canada ranges from 0.1 to 0.3 mg/L[10], which provides only a modest source of fluoride. Non-milk based formulas have higher fluoride content because the calcium that is added to formula contains fluoride. The more important issue, however, is the fluoride content of concentrated or powdered formula when reconstituted with fluoridated water. Considering the potential for mild fluorosis, caution is advised for infants consuming formula that is reconstituted with optimally-fluoridated water.

Significant cariostatic benefits can be achieved by the use of fluoride-containing preparations such as toothpastes, gels, and rinses, especially in areas without water fluoridation.[11] Monitoring children's use of topical fluoride-containing products, including toothpaste, may prevent ingestion of excessive amounts of fluoride.[12]

A number of clinical trials have confirmed the anticaries effect of professional topical fluoride treatments, including 5% neutral sodium fluoride varnish.[13,14] Fluoride varnishes can prevent or reverse enamel demineralization.[15] In children with moderate to high caries risk, fluoride varnishes[14,16] and fluoride-releasing restorative and bonding materials have been shown to be beneficial and are best utilized as part of a comprehensive preventive program in the dental home.[17-19]

Policy statement
1. The AAPD endorses and encourages the adjustment of fluoride content of domestic community water supplies to optimal levels where feasible.
2. When fluoride levels in community water supplies are suboptimal, and after consideration of sources of dietary fluoride, the AAPD endorses the supplementation of a child's diet with fluoride according to the guidelines jointly recommended by the AAPD[8], the American Academy of Pediatrics[20], and the American Dental Association (**ADA**)[21] and endorsed by the Centers for Disease Control and Prevention.[1]

3. The AAPD encourages dental professionals to inform medical peers of the potential of enamel fluorosis when excess fluoride is ingested prior to enamel maturation.
4. The AAPD encourages continued research on the causes of enamel fluorosis.
5. The AAPD does not support the use of prenatal fluoride supplements.[19]
6. The AAPD recommends an individualized patient caries-risk assessment before prescribing the use of supplemental fluoride-containing products.[8,22]
7. The AAPD encourages the continued research on safe and effective fluoride products, including fluoride-releasing restorative materials.
8. The AAPD supports the delegation of fluoride application to auxiliary dental personnel, or other trained allied health professionals, by prescription or order of a qualified dentist, after a comprehensive oral examination has been performed.
9. The AAPD endorses ADA 2002 House of Delegates Resolution 67H to encourage labeling of bottled water with the fluoride concentration and company contact information.[23] The resolution also supports including information with each home water treatment system on the system's effects on fluoride levels.
10. The AAPD encourages all beverage and infant formula manufacturers to include fluoride concentration with the nutritional content on food labels.
11. The AAPD encourages dentists and other health care providers to educate parents that both infant formula and the water used to reconstitute the formula may contain fluoride. Dentists and other health care providers, therefore, should assist parents in determining the infant's fluoride exposure.

References

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Wisconsin Chapter | **POSITION STATEMENT | FLUORIDATED WATER**

WIAAP
563 Carter Court, Suite B
Kimberly, WI 54136
Phone: 262.490.9075
E-mail: KLaBracke@wiaap.org

***Background*:**

Water fluoridation is a community-based intervention that optimizes the level of fluoride in drinking water, resulting in protection of the teeth before and after eruption. There is emerging opposition to the practice of community water fluoridation, unsupported by scientific evidence. The possibility of banning fluoridation of community water supplies poses a significant threat to the public health of the citizens of Wisconsin.

***Rationale*:**

Water fluoridation is an economical means of reducing tooth decay by 20-40%. For 65 years, community water fluoridation has been a safe and healthy way to effectively prevent tooth decay. The estimated cost savings is clear. The lifetime cost per person equals less than the cost of one dental restoration. The Centers for Disease Control and Prevention has proclaimed the practice one of the 10 greatest public health achievements of the 20th century, along with greater use of vaccines and recognition of tobacco as a health hazard.

Currently more than 204 million people in the United States are served by community water supplies containing enough fluoride to protect teeth.

The American Academy of Pediatrics' Bright Futures standards recommend children drinking **community-fluoridated** water by the age of six (6) months.

There is **no study nor evidence** to demonstrate health problems associated with the intake of optimally fluoridated water. Drinking fluoridated water is safe.

An untreated community water supply does not provide the protective oral health upon which people in Wisconsin have benefitted for decades.

Every $1 invested in water fluoridation saves a community approximately $38 in future dental treatment costs.

Therefore, the Wisconsin Chapter of the American Academy of Pediatrics opposes any efforts to eliminate fluoridation of community-based water supply.

REFERENCES:

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http://brightfutures.aap.org/pdfs/Guidelines_PDF/1-BF-Introduction.pdf

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http://www.astho.org/Display/AssetDisplay.aspx?id=147

May 29, 2012

Dear Sir/Madam:

Mr. Bohl's proposal to cease Milwaukee's fluoridation program does nothing more than seek to undo a highly effective, repeatedly proven, exceedingly economical, unquestionably safe, and demonstrably fundamental public health measure that the city's political leadership had the wisdom, foresight, and regard for the general welfare to implement nearly 60 years ago. As a result, millions of Milwaukee city residents since then, the current 589,697 residents of the city, and future generations have or will have derived the health and economic benefits of community water fluoridation across the age spectrum.

These long-established benefits need not be recited here– they have been amply provided by others rightfully opposed to this proposal– other than to say they are affirmed in a concise statement from the Centers for Disease Control: Community water fluoridation is one of the 10 great public health achievements of the 20th century.

In contrast to this is Mr. Bohl's statement of May 22, 2012, which is unfortunately filled with scripted falsehood, fabrication, distortion of fact, indefensible assertions, selective half-truth, cunning use of innuendo and demonstrate a lack of awareness or attention to science and health care issues. A prime example is his opening allegation that "New research shows that ingesting fluoride delivers health risks without benefit of less tooth decay which makes water fluoridation obsolete, unhealthy and a waste of money…" No such "new research" in any legitimate, refereed journals exists. This is hardly the basis upon which a representative body responsible to its constituency is to decide a public health issue, or any public policy matter for that matter.

Milwaukee has every reason to take pride that its aldermen of the day made their city an early leader among all major cities in the nation by adopting community water fluoridation in 1953 to benefit its residents, a decision made through ingenuous and intelligent assessment of the scientific and economic facts and sense of responsibility for the public welfare. The subsequent avoidance of unnecessary disease and the cost of its treatment have accrued monumentally not only to individual families and to the city's coffers, but to its character as well.

Prevention of disease and avoidance of unnecessary health care costs remain key responsibilities of elected officials, and are only underscored nowadays. Present day aldermen already possess an extremely potent tool to do just that, and are now called upon to reaffirm Milwaukee's proud legacy by soundly rejecting Mr. Bohl's proposal. The entire American Public Health Community stands behind this scientifically proven disease prevention measure.

Sincerely,

|Judy Gelinas, President

American Association for Community Dental Programs represent more than 500 local, county and non-profit dental public health programs with a vision is to promote and protect the oral health of the residents of cities, counties, and communities in the United States by stimulating, improving, and maintaining city, county, and community-based oral health programs.

From: Leuthner, Steven [sleuthne@mcw.edu]
Sent: Thursday, May 31, 2012 11:28 AM
To: Black, Tobie
Subject: Fluoride in Water

Dear Steering and Rules Committee,

I am writing as a concerned citizen and pediatrician about the proposal to eliminate fluoridation of the Milwaukee water supply. As a physician I am aware of a vast array of literature supporting this practice as an incredible public health achievement. Not only does it in the long run support children's dental health, but also adult and children's global health. There is more and more evidence of dental disease increasing risks of stroke and cardiovascular disease. So when the AAP argues it that "For every $1 invested in water fluoridation, approximately $38 is saved in future dental treatments.", I would suggest that is the minimum health care savings and it is probably hundreds if not thousands of dollars more in savings and quality of life.

I have just traveled to Guatemala on a medical mission trip with Timmy Global Health. I would claim that perhaps the biggest health risk to the children in that country is malnutrition and poor dentition. The Timmy Foundation and the WHO are now recommending fluoride washes for these children on these visits, hoping that even this small amount could help some. It is a clear demonstration how fluoridation of water sets our country apart as far as health. I would hate to see us move backwards toward a third world country health standards.

Finally, I speak as a concerned Foster parent of 2 Milwaukee city children at the age of 16 months and 4 ½ years of age. While my wife and I still do not know what will happen to these children, it is disheartening to think that should they return to the city of Milwaukee as children of a single, poor mother, that our city and council do not think of them and their dental, and subsequently global, health. I can provide them with what they need, but the poor cannot. I consider this to be an un-Christian abandonment move toward the poor children in Milwaukee County. Is this all for a Buck?

Please re-consider and agree to continue to spend the money on one of the best public health programs that our country has achieved. Stand up for the children, especially the poor children that have no voice.

Sincerely,

Steve Leuthner, Foster Father and Citizen

Steven R Leuthner, MD, MA
Professor of Pediatric and Bioethics
Medical College of Wisconsin
phone: 414-266-6820
pager: 414-907-3335

Steven R Leuthner, MD, MA
Professor of Pediatric and Bioethics
Medical College of Wisconsin
phone: 414-266-6820

pager: 414-907-3335

FILE NUMBER: 120187

NAME	ADDRESS	DATE SENT		
Amy Hefter	LRB	5/29/12		
All Council Members	CC	x		
William Lobb, Marquette Dental	william.lobb@marquette.edu	x		
Mark Paget, WDA	mpaget@wda.org	x		
Mara Brooks, WDA	mbrooks@wda.org	x		
Dr. Paul Levine	Toothmedic@aol.com	x		
Evan Zeppos	ezeppos@zeppos.com	x		

Legislation Details (With Text)

File #:	120188 **Version:** 0		
Type:	Resolution	**Status:**	In Committee
File created:	5/22/2012	**In control:**	STEERING & RULES COMMITTEE
On agenda:		**Final action:**	
Effective date:			
Title:	Resolution authorizing the transfer and expenditure of funds within the Milwaukee Water Works to create an oral health program for medically-underserved children.		
Sponsors:	ALD. BOHL		
Indexes:			
Attachments:	E-Mail from Carrie Lewis_Subject-Fluoridation levels on behalf of Alderman.pdf, 07-25-2011 Dispute Over State Grant for Dental Program - WSAU News-Talk 550AM 99.9FM.pdf, 12-28-2010 Wis. Groups Clash Over Proposed Dental School - DrBicuspid.pdf, 10-14-2010 Milwaukee Named a Cavity Capital - JSOnline.pdf, 05-10-2010 Investigation Dentists Won't Treat Children on state health plan - WKOW 27 Madison WI.pdf, 05-28-2011 State Lags Neighbors in Needy Children's Dental Care - JSOnline.pdf, 12-14-2009 The Root Problem - JSOnline.pdf, Hearing Notice List		

Date	Ver.	Action By	Action	Result	Tally
5/22/2012	0	COMMON COUNCIL	ASSIGNED TO		
5/29/2012	0	STEERING & RULES COMMITTEE	HEARING NOTICES SENT		
5/29/2012	0	STEERING & RULES COMMITTEE	HEARING NOTICES SENT		
5/29/2012	0	STEERING & RULES COMMITTEE	HEARING NOTICES SENT		

Number
120188
Version
ORIGINAL
Reference

Sponsor
ALD. BOHL
Title
Resolution authorizing the transfer and expenditure of funds within the Milwaukee Water Works to create an oral health program for medically-underserved children.
Analysis
This resolution authorizes the transfer of funds, in the amount of $540,000, used for the fluoridation of water distributed by the Milwaukee Water Works to a new account to create the “To Every Child a Healthy Smile” program, an oral health program for medically-underserved children in the City.
Body
Whereas, In 1953, the Common Council voted to add fluoride to the water supply at a time when Milwaukee had a serious problem of tooth decay and, unlike today, fluoridated tooth pastes, gels and mouthwash were not readily available; and

Whereas, Fluoridation is unethical as individuals are not asked for their informed consent prior to medication, as is standard practice for the administration of all other medications; and

Whereas, Medically-underserved children are often without dental care due to the economic constraints dentists experience as a result of low reimbursement rates through Medicare programs such as BadgerCare; and

Whereas, As a result, low-income children have less access to dental care, less preventive measures and less access to education, all of which contribute to oral health problems; and

Whereas, Statistics show that out of about 111,000 Medicaid-eligible children in Milwauke County, only about 21,000 children have received dental exams; and

Whereas, The Milwaukee Water Works estimates the cost for 2012 of introducing sodium silicofluoride or other fluoride compounds into water distributed by the Milwaukee Water Works to be $540,000; and

Whereas, The Milwaukee Water Works Account No. 632000-Fund No. 0410-Dept. No. 6412-Program No. 5221-Class No. R999-Year 2012 and Account No. 632000-Fund No. 0410-Dept. No. 6416-Program No. 5222-Class No. R999-Year 2012, have an approximate combined existing balance of $2.8 million; now, therefore, be it

Resolved, By the Common Council of the City of Milwaukee, that the City Comptroller is directed to establish that appropriate account within the Milwaukee Water Works entitled “To Every Child a Healthy Smile” program; and, be it

Further Resolved, That the City Comptroller is directed to transfer the amount of $270,000 from the Milwaukee Water Works Account No. 632000-Fund No. 0410-Dept. No. 6412-Program No. 5221-Class No. R999-Year 2012 to the account established for the “To Every Child a Healthy Smile” program; and, be it

Further Resolved, That the City Comptroller is directed to transfer the amount of $270,000 from the Milwaukee Water Works Account No. 632000-Fund No. 0410-Dept. No. 6416-Program No. 5222-Class No. R999-Year 2012 to the account established for the “To Every Child a Healthy Smile” program; and, be it

Further Resolved, That the Superintendent of the Water Works is directed, in collaboration with the Commissioner of the Milwaukee Health Department, to establish guidelines related to the “To Every Child a Healthy Smile” program for the expenditure of funds to provide oral healthcare for medically-underserved children in the City; and, be it

Further Resolved, That the Superintendent of the Water Works and the Commissioner of the Milwaukee Health Department are authorized to enter into such contracts and agreements as necessary to accomplish the intent and purpose of this resolution; and, be it

Further Resolved, That the appropriate City officials are authorized to seek Public Service Commission approval for the expenditure of funds to provide oral healthcare for medically-underserved children in the City through the “To Every Child a Healthy Smile” program.

Requestor

Drafter
LRB139299-1
Amy E. Hefter
5/21/2012

Peterson, Todd

From: Lewis, Carrie
Sent: Thursday, November 17, 2011 2:19 PM
To: Peterson, Todd
Cc: Korban, Ghassan; Daniels, Laura; Silletti, Leslie
Subject: FW: Fluoridation levels on behalf of Alderman

Hi Todd,

Here is the remaining info for which Ald. Bohl has asked:

The current annual cost of adding fluoride is approximately $540,000. This is $530,000 of for the fluoride itself and about $10,000 for maintenance. (If the dosage was 0.7 mg/L, the cost of the fluoride would be approximately $337,000.)

Maintenance costs should be reduced in future because the Linnwood fluoride system was refurbished in 2010 at cost of $777,602. There is no immediate need to replace the Howard Avenue system.

Carrie

From: Lewis, Carrie
Sent: Wednesday, November 16, 2011 8:23 AM
To: Peterson, Todd
Cc: Korban, Ghassan; Daniels, Laura
Subject: FW: Fluoridation levels on behalf of Alderman

In response to this inquiry, I wanted to let you know that the CDC and DHS have not officially released their recommendation to lower the fluoride dosage in drinking water to 0.7 mg/L and the DNR has not changed the code for Wisconsin water utilities. Wisconsin Administrative Code NR 809.74 still states:

"(1) PUBLIC WATER SYSTEMS WHICH ADD FLUORIDE. (a) The water supplier for a community water system artificially fluoridating the water shall establish a monitoring program in order to maintain the fluoride concentration within the range of 1.0 to 1.5 milligrams per liter as recommended by the dental health section of the department of health services for optimum dental benefits."

Therefore, Milwaukee Water Works continues to add fluoride to drinking water to reach a dosage of 1.1 mg/L. MWW has this target of 1.1 mg/L because there is a slight amount of variability in the fluoride addition, and by aiming for 1.1 we do not violate the lower limit of 1.0 mg/L.

I am collecting the information on the annual cost to fluoridate the water and will send that to you as soon as I have it.

Carrie
X2801

From: Tmara Rahim [mailto:tmara.rahim@gmail.com]
Sent: Friday, November 11, 2011 3:54 PM

To: Lewis, Carrie
Cc: Peterson, Todd
Subject: Fluoridation levels on behalf of Alderman

Good Afternoon,

I am an intern in the City Clerk's Office and am contacting you on behalf of Alderman Bohl. The alderman would like to know whether Milwaukee Water Works has reduced the water fluoridation level to the .7mg of fluoride per liter of water as recommended by the Department of Health and Human Services. If it hasn't, he would like to know if there is a requirement to wait to reduce the current level. Additionally, he would like to know the current annual cost of fluoridating the water supply at 1.1mg/L for Milwaukee.

If you could please send your response to Todd Peterson, it would be much appreciated.

Regards,

--
Tmara Abidalrahim
International Business and Marketing '13
College of Business Administration
Marquette University
(414) 243-3944

Dispute over state grant for dental program

Monday, July 25, 2011 4:22 a.m. CDT

Marshfield Clinic logo.

MARSHFIELD, Wis (WSAU) A proposed dental school at Marshfield Clinic is in danger of losing a $10-million state grant.

The state says the grant was intended to provide a rural outreach dental facility, not to build a dental school. Some of the money could be used to train dentists who already have their degrees to help in rural areas. But the state sent a letter to Marshfield Clinic saying that building a dental school would not be covered.

Some reports say Marquette University -- which runs the only dental school in the state -- objected. The Wisconsin Dental Association also wanted all of the funding used for rural outreach, which they consider an urgent need.

Marshfield Clinic says the still intend to build a school of dentistry, and will continue talks with the state on how the grant can be used.

Tags:clinic (/news/articles/tagged/clinic/), dental (/news/articles/tagged/dental/), grant (/news/articles/tagged/grant/), marshfield (/news/articles/tagged/marshfield/),

Similar Articles

Marshfield Clinic saves Medicare money (/news/articles/2011/aug/11/marshfield-clinic-saves-medicare-money/)

"Touchy" doctor reaches plea in criminal case (/news/articles/2011/jul/25/touchy-doctor-reaches-plea-in-criminal-case/)

Leukemia drug shortage averted (/news/articles/2012/feb/16/leukemia-drug-shortage-averted/)

Wis. groups clash over proposed dental school

By Rabia Mughal, Contributing Editor

December 28, 2010 – Dental groups in Wisconsin are clashing over the recently approved $10 million in state money intended to support the construction of a new dental school – a proposition that the Wisconsin Dental Association (WDA) calls "ill-advised" and a "lame-duck decision."

Marquette University, home to the state's only dental school, is also vigorously opposed to the proposition, and its position recently led to a tense exchange between university representatives and state Sen. Bob Jauch (D-Poplar).

The disagreement stems from a bill passed in April by Jauch that offered a $10 million grant for the construction of a rural dental education outreach facility at the Marshfield Clinic if the clinic secured matching funding of at least $10 million in nonstate funds.

The Marshfield Clinic, established in 1916, provides dental access for the rural and economically impoverished communities of Wisconsin through a combination of state and federal grants. Marshfield currently operates five federally qualified dental health clinics in northwest Wisconsin.

Marshfield was awarded the grant in December after Security Health Plan of Wisconsin, a nonprofit health insurance company affiliate of the clinic, matched the amount.

The dispute arose when Marshfield's intention to start a new dental school was discovered by Marquette, which had been in favor of the bill earlier this year.

> "This $10 million was spent without much debate."
>
> — Gene Shoemaker, DDS, president, Wisconsin Dental Association

"We were told that the proposal was for postbaccalaureate and residency programs that would be complementary to Marquette's program," William Lobb, DDS, dean of Marquette University School of Dentistry, told *DrBicuspid.com*.

Marquette officials initially supported the idea, but had they known that the final proposal would include a new dental school, they would have opposed it, Dr. Lobb said.

"If the state is investing money, it should be in the existing dental school," he added.

Different student-patient populations

In a list of questions sent to the State Building Commission after it approved the project earlier this month, the WDA asked why Marshfield is now building a dental school when it was "vaguely sold as a 'dental educational outreach facility' when the bill originally passed the Legislature in the waning days of session."

Greg Nycz, director of health policy at the Marshfield Clinic and executive director of the Family Health Center of Marshfield, said he was open about his desire to set up a dental program from the start and that there was an article about it in the local paper as far back as April.

The new school is intended to serve a different purpose than the existing school at Marquette, he emphasized. It will have a rural focus and will try to attract students who can go to underserved areas, with clinical training provided in the rural dental clinics.

"Most of the students in the existing school tend to come from higher income families, and we will try to select students from the underserved populations and areas so they can serve in those areas," explained Nycz.

The program will be ideal for students seeking to benefit from expanded National Health Services Corps under the healthcare reform, which will make students servicing underserved areas or in a community health center eligible for federal loan repayment, he added.

Wisconsin has one of the poorest percentages of getting dental care to BadgerCare (the state's Medicaid program) and Medicare patients, and hundreds of thousands are not getting care, according to Nycz.

"The dentists that we do have are not evenly distributed," he said. "We want to help the state find solutions. If we continue to train dentists like we always have in the past, we will get the same results. The aim is to address the problem of undersupply in the rural communities."

Underserved needs being met?

Marquette feels that rural needs are already being met by its current graduates, and any additional state money would be best spent bolstering the existing dental school, according to Dr. Lobb.

"There are 80 graduates in our program, and most of them stay within Wisconsin and are well distributed," he said. "We service 64 of the 72 counties in Wisconsin, and we feel we are meeting the needs of people."

Marquette's dental school also draws students from underserved communities, and there is no guarantee that the students in this new program will stay in the underserved communities, he contended.

Dr. Lobb had asked the building commission to postpone the allocation of these funds because he felt they were handed out without much public debate, an objection echoed by Gene Shoemaker, DDS, president of the WDA.

Also, the school and the WDA have pointed out that the state's own study on the need for rural dental education that was completed in March 2010 does not support a new dental school.

According to the WDA, the study found that "while lack of dentists is an issue in selected counties, the core problem is Medicaid fees are low and do not provide Medicaid members adequate financial access to care, and that the establishment of a new dental school at the Marshfield Clinic, as currently proposed, does not appear feasible."

There are other ways to increase access to care besides spending $10 million on this project, said Dr. Shoemaker.

"Wisconsin, like many other states, has very low Medicaid reimbursement, and one solution is raising these rates, not creating more dentists," he said.

Dentists want to serve this population, he added, "but if from a small business standpoint it does not make sense and you cannot even meet your overhead, they will eventually move to more affluent areas."

Dr. Shoemaker also feels that the state money would be better used on the existing dental school. Marquette currently has a proposal to add 20 more dental students to its school and could use the funds to do more research and add more faculty, he said.

"The state already says there is no money, but this $10 million was spent without much debate," concluded Dr. Shoemaker.

"State funding is tight," wrote Dr. Lobb in a December 17, 2010, letter submitted to the *Milwaukee Journal Sentinel.* "Funding should be directed to entities with a proven record of meeting the needs of the state and its citizens."

Related Reading

Marquette questions need for 2nd dental school, December 16, 2010

Wis. Legislature allocates $10M for dental education, April 8, 2010

Wis. weighs options for improving access to care, January 20, 2010

Wis. mulls new dental school, October 23, 2009

Forum Comments

7 comments so far ...

12/29/2010 11:43:05 AM taddds	I think having another dental school in WI would be great. I am from WI and did not get into Marquette two years in a row and ended up going to dental school at a private school out of state. I now have no intention of moving back there because of the opportunity created for me by the other school. Marquette has a win-win situation with there being no competing schools in

	state. I went to Marquette for my undergraduate degree and know that there are plenty of out of state students that graduate from their program, and they don't always stay in WI to work. It would be great to have a state school that would focus on residents and not just where the money comes from (affluent families, as the article states). Illinois has UIC and Southern, Minnesota has UM, etc., and these schools accept residents of their states for the vast majority as dental students (I know, I applied there). I don't know why Marquette would need $10 million... the school there is almost brand new. Why didn't they think of adding 20 additional spots when they built that brand new facility back in 2002? I think another school will bring more opportunity to more pre-dental residents, which would lead to dentists practicing in the areas they grew up in. I fully intend on working to serve those without access to care. Unfortunately, that's in another state, not WI.
12/29/2010 1:32:51 PM glenp	Need I say any more about the need for more dental schools. We all can see the lower level students that are now going to the private institutions. Do we need more of that graduating to our profession?
12/29/2010 2:25:03 PM mldds	I can't believe this debate continues. Didn't anybody learn anything form the 80's? They tried opening lots of dental schools, expanding the class sizes, etc., and guess what? Very few graduates want to move to a remote area and set up shop. There is probably tremendous potential for start-up practices, but you still have to live there. Most people live in larger metro areas and medium sized cities for a reason. The only way opening more schools is going to solve access to care is if they REQUIRE new graduates to practice a minimum of 10 years in one of the pre-designated underserved areas. [Is that even legal?] Get the licensing boards involved: No service as promised? Then your license is revoked. They can continue to HOPE that new graduates of these new schools will go to underserved areas, but in reality, all that will happen is the overserved areas will become even more saturated with dentists and make survivng more difficult for the rest of us. If there weren't too many dentists back in the early 90's, all the HMOs and PPOs would not exist, because no one would have signed up with them out of desperation. "Those who do not learn from history are doomed to repeat their mistakes."
12/29/2010 3:07:45 PM WhiteLake69	Creating more dental schools, with associated costs and graduates who are unlikely to benefit the poor/near poor over the long run, is not a better idea than trying two year therapists, who practice under the supervision of dentists. They can be taught in community colleges and their supply can readily be expanded or contracted, depending upon market demand. In addition, they can target poor and near poor, rather than creating expensive to educate dentists, who have never evidenced a high level of interest in or commitment to practice among the poor and near poor. The five or six basic services therapists have successfully performed, since the 1920s, meet much of their needs, the rest provided by dentists if needed. There is no evidence that therapists have adversely impacted private practice dentistry nor have they provided substandard or inappropriate care. Once a dental school is built, what is the likelihood that it will turn out graduates who will seek primarily to serve poor populations, rather than migrate to where they can make the most? As the saying goes, "How you gonna keep them down on the farm now that they've seen Paris? It seems that dentistry will eventually do what makes sense (therapists), after it attempts to do everything else that has never worked not first.
12/29/2010 4:27:18 PM Nucrona	Another school won't change the lives of students like yourself. Your problem was that you were noncompetitive academically.

More Forum Comments »»

Milwaukee named a "Cavity Capital"

By [redacted] of the Journal Sentinel
Oct. 14, 2010

Well, we're in the Top 10 of a national list but it bites.

Milwaukee is No. 8 among the Top 10 "most dentally challenged cities" according to Men's Health magazine. St. Louis ranks No. 1 on that list, which is based on community water fluoridation; the percentage of people who saw a dentist in the past year or have had their teeth extracted; the percentage of households using dental floss; money spent on oral hygiene products; and the number of dentist offices per capita.

You're not going to believe the cities with the brightest smiles. We have to think there might be David Letterman-sized gaps in the research.

Read about it here.

Investigation: Dentists won't treat children on state health plan

Posted: May 10, 2010 9:05 PM CDT

By Dan Cassuto - bio | email | Twitter | Facebook | Blog | (608) 661 2727

Most of us don't love going to the dentist. But most everyone would admit it's important, especially for kids.

Right now, tens of thousands of Wisconsin children are being turned away from dentists for one simple reason: they use the state health plan, BadgerCare.

BadgerCare is a form of medical assistance that gives one million low-income Wisconsin residents access to doctors and dentists through the federal Medicaid program.

A 4-month WKOW investigation revealed major problems with BadgerCare's dental coverage. Virtually no dentists in Dane, Sauk, Rock or Columbia counties are accepting new patients on the BadgerCare health plan, leaving many families with no options for pediatric dental care in southern Wisconsin.

"We can't find a dentist for nothing in the world," said Jill Schwantz of Portage, who told WKOW she called hundreds of dentists in southern Wisconsin. "Every single dentist said no."

Schwantz is looking for a dentist to treat her three children, ages nine, six and two-and-a-half.

Another mother, Susan Hessel of Madison, also told us of her struggle to find a dentist to treat her 14-year-old son.

"If you're one of the first ten callers, three months from now, on this day, on this morning, we might be able to add you as a new client," said Hessel, recounting her experience calling dental offices in the Madison area.

Hessel has been trying to find a dentist for two years.

The state's Dept. of Health Services say 600 families a week call its hotline, most of them looking for dentists for their children.

WKOW confirmed the claims of these and other families. Our reporter personally called hundreds of dentists in southern Wisconsin using a published list of participating dentists posted on BadgerCare's official website. Nearly every single dentist refused to accept new BadgerCare patients. The only exceptions were public clinics, usually with long waiting lists, or other special age, referral or emergency care restrictions.
Families say it's esspecially frustrating to call dentists listed on the BadgerCare website, only to be turned down. The state says the website is merely a list of dentists who are treating some BadgerCare patients, not necessarily willing to treat new ones.

Dentists tell us the BadgerCare dental program is broken.

They blame low reimbursement rates, complicated government paperwork, and a history of BadgerCare patients who don't show up for appointments.

"We've been trying for years to try to make the program better for the population and dentists, with no success at all," said Dr. Timothy Kinzel, a pediatric dentist in Madison. "Let me put it this way. What if Physicians Plus or Group Health or Dean Care was selling insurance to these patients and telling them they had these providers. But then they couldn't find a provider. My guess is the politicians would be a little worried about that."

BadgerCare pays dentists significantly lower rates than private insurance companies. At Dr. Kinzel's practice, a standard cleaning costs $74, a rate that most private insurers pay. BadgerCare pays $21 for the same service.

A two-surface white filling is billed at $215, also a rate paid by private insurance companies. Dr. Kinzel would only get $56 from the state's BadgerCare plan.

Dr. Kinzel's practice is made up of about 30 to 40 percent of medical assistance patients. If the practice opened its doors and accepted any BadgerCare child who wanted treatment, Dr. Kinzel says his practice would not survive.

"The reality is the program doesn't work," said Dr. Kinzel.

Dentists also say the state rejects reimbursement claims and refuses to pay ten times more often than private insurance providers.

"I wish I could convince these dentists to enter," said Jason Helgerson, Medicaid Director for the Dept. of Health Services. "Yes, we need more dentists to participate in the program. Yes, we're committed to trying to work with them. I wish I had all the money in the world to pay them more. The simplest, easiest solution to all of us would be about $100 million."

Helgerson stressed the state is acting aggressively to respond to what he calls "access challenges."

The department recently created a 12-person dental unit to field calls and match families with dentists. That special hotline gets about 600 calls a week, 30,000 or so a year from Wisconsin families. Helgerson says it's a start, but acknowledges many families have to travel farther than he'd like to find a dentist willing to treat new BadgerCare patients.

Susan Hessel, the Madison mother trying for two years to find a dentist to treat her son, says she's planning to drive to Chicago and pay in cash.

And that's yet another flaw with the BadgerCare program, according to the families.

Once you're enrolled in BadgerCare, dentists are prohibited by law from treating you outside of the official program. That means dentists can't accept cash payments, even if patients want to pay out of pocket.

"The system is not working right now," said Hessel.

Families don't simply drop out of BadgerCare entirely because it provides standard medical coverage, which doesn't appear to have any of the access problems the dental side has.

"I think the state is running a very good program," said Helgerson. "We're providing health services to a million people."

Helgerson encourages families to contact the state's dental unit for assistance finding a dentist.

The toll-free number to the Dept. of Health Service's BadgerCare dental unit is (800) 362-3002.

JOURNAL INTERACTIVE

State lags neighbors in needy children's dental care

Wisconsin receives 'C' grade, on par with Mississippi, West Virginia

By Gary Poulson of the Journal Sentinel
May 28, 2011

Wisconsin made only marginal progress last year in addressing one of the most entrenched problems in its health care system: access to dental care for needy children.

The state's performance lagged Minnesota, Iowa and Illinois in a report released last week by the Pew Center on the States. And it did no better than states such as Mississippi and West Virginia.

The state's poor performance in providing dental care to children in low-income families is a long-standing and widely acknowledged problem. And it will be even harder to address given the current budget crisis.

"We've been working on this, trying to find workable solutions, for years," said Kitty Rhoades, deputy secretary of the Department of Health Services.

The state has taken steps to lessen the problem. But Rhoades said, "There's not an easy solution."

The report by the Pew Children's Dental Campaign, a national effort to increase access to dental care for low-income children, found:

Fewer than one in three children insured by the state's BadgerCare Plus or Medicaid programs saw a dentist in 2009.

One in five third-graders had untreated tooth decay in the 2007-'08 school year.

The fees paid to dentists by the state's health programs are the fifth-lowest in the country.

The annual report, "The State of Children's Health: Making Coverage Matter," ranks state's progress in improving access to dental care based on eight benchmarks, such as the percentage of children covered by Medicaid programs who received dental care and the percentage of residents who live in communities with fluoridated water.

It gave Wisconsin a "C" grade. By comparison, Minnesota received an "A." Illinois and Iowa were given "B" grades.

Nine states, plus the District of Columbia, were given a "D" or "F."

The report found that 22 states had made progress last year in improving access to dental care for children.

Sealants could help

Wisconsin might have been among those 22 states had its expanded program to provide sealants to children in schools been taken into account.

Sealants are clear plastic coatings applied to the chewing surfaces of molars, the teeth most prone to cavities. They can prevent 60% of tooth decay at one-third the cost of filling a cavity.

Wisconsin is among the states that allow hygienists to place sealants in school-based programs without requiring a dentist's exam.

The statewide program will provide sealants to more than 20,000 children this school year, up from 10,000 in the previous school year, said Matt Crespin, the oral health project manager for Children's Health Alliance of Wisconsin, part of Children's Hospital and Health System. The program focuses on schools with a large number of children from families with limited incomes.

Now in its second year, the program is funded by a three-year federal grant, state funds and an annual contribution of $241,000 from Delta Dental of Wisconsin, which oversees and underwrites dental plans for employers.

The Joint Finance Committee has approved $250,000 in additional funding for the program starting July 1, 2012, when the federal grant ends, Crespin said.

The committee also approved $1.7 million in grants for nonprofit dental clinics that are not affiliated with community health centers.

In recent years, community health centers, which receive federal money, have been expanding to provide more dental care. Milwaukee Health Services and Westside Healthcare Association have added dentists and hygienists. And Family Health Center of Marshfield, an affiliate of Marshfield Clinic, now operates one of the largest rural networks of dental clinics in the country.

Children's Hospital and Health System also recently opened another dental clinic. And St. Elizabeth Ann Seton Dental Clinic, formerly Madre Angela Dental Clinic, a collaborative program sponsored by Columbia St. Mary's Health System, expanded.

As recently as 2008, though, only 18% of the children covered by the BadgerCare Plus program in Milwaukee County saw a dentist. The percentage was the lowest of any county in the state.

Dentists get lower rates

By federal law, state Medicaid programs must provide dental care to children. But states determine what to pay dentists, and the rates paid in Wisconsin typically don't cover a dentist's costs.

"We don't break even - we lose money," said Gene Shoemaker, a dentist and president of the Wisconsin Dental Association.

One result is that many dentists don't accept patients covered by state health programs. And those that do have to limit the number of patients they will see.

Only 11% of the state's dentists filed Medicaid claims of $10,000 or more a year, according to a state report.

Policy experts agree that raising reimbursement rates will increase children's access to dental care.

"We've got experience from a number of other states that show that is true," said Andy Snyder, a researcher with the Pew Children's Dental Campaign.

The campaign recommends states pay dentists an average of 60.5% of their retail fees.

States need to respond to dentists' concerns about rates, Snyder said.

"But that is not a reason to not look at the rest of the policy options," he said. "It's a problem that's bigger than the dental community can handle on its own."

The Department of Health Services plans to take that approach in the short term.

"We don't have the money for an overall rate increase," Rhoades said. "We understand that. But let's talk about how else we could do this."

Health Services Secretary Dennis Smith and Rhoades hope to work with dentists and others to find ways to improve access.

"This is something we take seriously," Rhoades said. "We are going to try to find a workable solution."

The dental association knows that raising rates is not an option given the budget shortfall. But it has proposed expanding the role of dental assistants, such as allowing them to complete a filling after a dentist has drilled the tooth. That would enable practices to be more efficient and lower costs.

The association opposes licensing dental therapists - hygienists trained to do fillings and simple extractions.

Their role would be similar to nurse practitioners and physician assistants - health care providers initially opposed by organized medicine but now seen as integral components of the health care system, particularly in primary care.

Minnesota passed a law in 2009 to allow dental therapists to practice in the state. And Crespin, of the Children's Health Alliance of Wisconsin, said the state has to look at different workforce models despite the dental association's opposition.

The state has taken steps to improve dental care for children, he said. And awareness has increased.

"But ultimately, there still is a lot to be done," Crespin said.

***

DENTAL NEGLECT

Access to dental care for children is a long-standing problem. A Pew Center report noted:

14% of children ages 6 to 12 had a toothache in the previous six months that was severe enough for a parent to know about.

Only 44% of the children covered by Medicaid programs received any dental care, compared with 58% of privately insured children, in 2009.

Among military recruits, 52% had oral health problems needing urgent attention; more than 15% had four or more teeth in urgent need of repair.

Find this article at:
http://www.jsonline.com/business/122781759.html

Check the box to include the list of links referenced in the article.

Editorial

The root problem

Until the state's reimbursement rate for dentists is addressed, we need to fully support programs that provide oral health services to the poor.

Dec. 14, 2009

The need for the St. Elizabeth Ann Seton Dental Clinic is obvious. Tooth pain is no respecter of income level.

Thousands of poor Wisconsin children are without dental care essentially because dentists can't afford to see them. And that's because of notoriously low reimbursements under BadgerCare Plus. The clinic is often the only option for the city's poor. The St. Elizabeth Ann Seton Dental Clinic addresses these issues for the neediest on Milwaukee's south side.

The Journal Sentinel's Georgia Pabst reported Monday that the clinic served more than 4,500 patients last year. Of that number, 1,500 were served at the clinic and 3,000 children through its Smart Smile, a portable dentist program that travels to schools.

Programs like these should be expanded. Especially during these tough economic times, dental needs often take a back seat to paying bills and putting food on the table. By the time patients walk into the clinic, they are often in pain because of this neglect. These clinics help to bridge the gap between need and access for the city's poor.

An estimated 60% of the children screened by the Smart Smiles program have cavities. But it's hard for them to get dental services in the community because few dentists accept patients with BadgerCare.

Earlier this year, the Journal Sentinel reported that fewer than one in four kids ages 3 to 19 who are insured through the Medicaid program BadgerCare Plus saw a dentist in 2007. That means more than 300,000 children in the state didn't get that necessary checkup.

This is happening because the state's reimbursement rate for dentists who accept BadgerCare Plus is too low. And with little money in the state budget to address the problem, the need for clinics like St. Elizabeth's grows.

Until the reimbursement issue is addressed, free clinics that serve the poor are the only access to dentists they are likely to get. We applaud the work done at the St. Elizabeth Ann Seton Dental Clinic, supported by Columbia St. Mary's Health System.

But ultimately, the solution lies with increasing the reimbursement rate for dentists so they can afford to

see BadgerCare Plus clients.

Should more be done to expand dental care to the poor? To be considered for publication as a letter to the editor, e-mail your opinion to the Journal Sentinel editorial department.

Find this article at:
http://www.jsonline.com/news/opinion/79257422.html

☐ Check the box to include the list of links referenced in the article.

City of Milwaukee Fiscal Impact Statement

A

Date 5/30/2012 File Number 120188 ☐ Original ☒ Substitute

Subject Oral Health Programs funded by Milwaukee Water Works

B

Submitted By (Name/Title/Dept./Ext.) Aaron Szopinski / Fiscal Planning Specialist / Budget & Mgmt. / x3384

C

This File

- ☐ Increases or decreases previously authorized expenditures.
- ☐ Suspends expenditure authority.
- ☒ Increases or decreases city services.
- ☒ Authorizes a department to administer a program affecting the city's fiscal liability.
- ☐ Increases or decreases revenue.
- ☐ Requests an amendment to the salary or positions ordinance.
- ☐ Authorizes borrowing and related debt service.
- ☐ Authorizes contingent borrowing (authority only).
- ☒ Authorizes the expenditure of funds not authorized in adopted City Budget.

D

Charge To

- ☒ Department Account
- ☐ Capital Projects Fund
- ☐ Debt Service
- ☐ Other (Specify)
- ☐ Contingent Fund
- ☐ Special Purpose Accounts
- ☐ Grant & Aid Accounts

E

Purpose	Specify Type/Use	Expenditure	Revenue
Salaries/Wages		$0.00	$0.00
		$0.00	$0.00
Supplies/Materials		$0.00	$0.00
		$0.00	$0.00
Equipment		$0.00	$0.00
		$0.00	$0.00
Services		$0.00	$0.00
		$0.00	$0.00
Other		$0.00	$0.00
		$0.00	$0.00
TOTALS		$ 0.00	$ 0.00

F Assumptions used in arriving at fiscal estimate.

The account balances specified in the Council File are appropriation balances, reflecting authority to spend water utility revenues as they are earned by the Water Works. The balances do not reflect cash on hand or availability of funds in the future, which is an important consideration and constraint when working with City enterprise funds.

G For expenditures and revenues which will occur on an annual basis over several years check the appropriate box below and then list each item and dollar amount separately.

☐ 1-3 Years ☐ 3-5 Years

☐ 1-3 Years ☐ 3-5 Years

☐ 1-3 Years ☐ 3-5 Years

H List any costs not included in Sections D and E above.

I Additional information.

There is currently no income in excess of that used for operations, maintenance, capital, debt, interest, depreciation, and other utility-specific obligations available in the Water Fund. That excess income availability is the statutory test for transferring water utility revenue to the General Fund or to use utility funds for non-Water municipal purposes. Currently, the Water Fund has no surplus funds meeting the test.

J This Note ☐ Was requested by committee chair.

FILE NUMBER: 120188

NAME	ADDRESS	DATE SENT		
Amy Hefter	LRB	5/29/12		
All Council Members	CC	x		
William Lobb, Marquette Dental	william.lobb@marquette.edu	x		
Mark Paget, WDA	mpaget@wda.org	x		
Mara Brooks, WDA	mbrooks@wda.org	x		