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

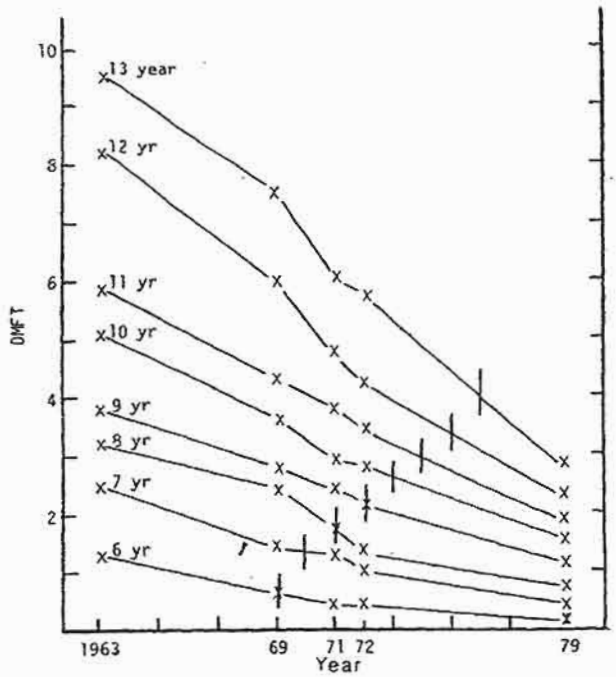

Fig. 1 Decline in caries, as measured by DMFT, in Tamworth, Australia, for children in age groups 6 years to 13 years. Data compiled from refs 14, 15. The vertical line cutting graph for each age group denotes year at which maximum possible benefit from fluoridation was reached. Tamworth was fluoridated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

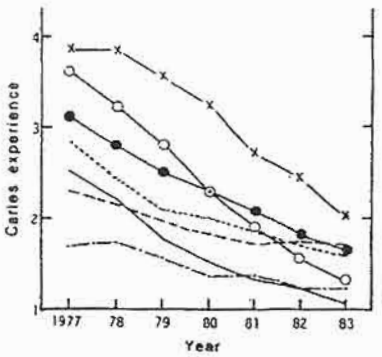

Fig. 2 Decline in the average number of (permanent) teeth per child with caries experience in each Australian state and the Australian Capital Territory as observed in school dental services[17]. 'Caries experience' can be one or more decayed, missing or filled teeth, and consists of an average for children 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.

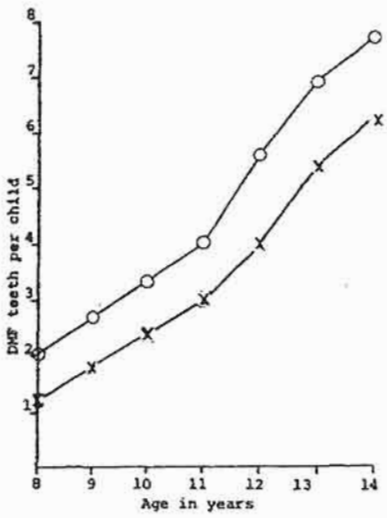

Fig. 3 The variation with age of decayed, missing and filled permanent teeth (DMFT) in fluoridated test towns (×) and unfluoridated control towns (○) in Britain, graphed from data published by the UK Department of Health[33]. Note that the rate of increase of DMFT is essentially the same in both groups.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

Sources for this story include:

http://www.vancouversun.com/health/...

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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.

.....

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.

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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.**

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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.

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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.

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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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Journal of the American Medical Association
jama.ama-assn.org

FATAL POI[S] FLUOROAC[ETATE]

REPORT OF A CASE

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

JAMA. 1955;159(16):1529-1532. doi: 10.1001/jama.1955.02960330029010

[+] Author Affiliations

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

Sodium Fluoroacetate Poisoning
Arch Pediatr Adolesc Med. 1975;129(10):1224-1226.
Abstract Full Text (PDF)

Journal of the American Medical Association
Vol 159, No. 16.

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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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.

912 Fairway Dr., High Point, N. C. (Dr. Brockmann).

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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Toxicological Reviews:
2006 - Volume 25 - Issue 4 - pp 213-219
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."

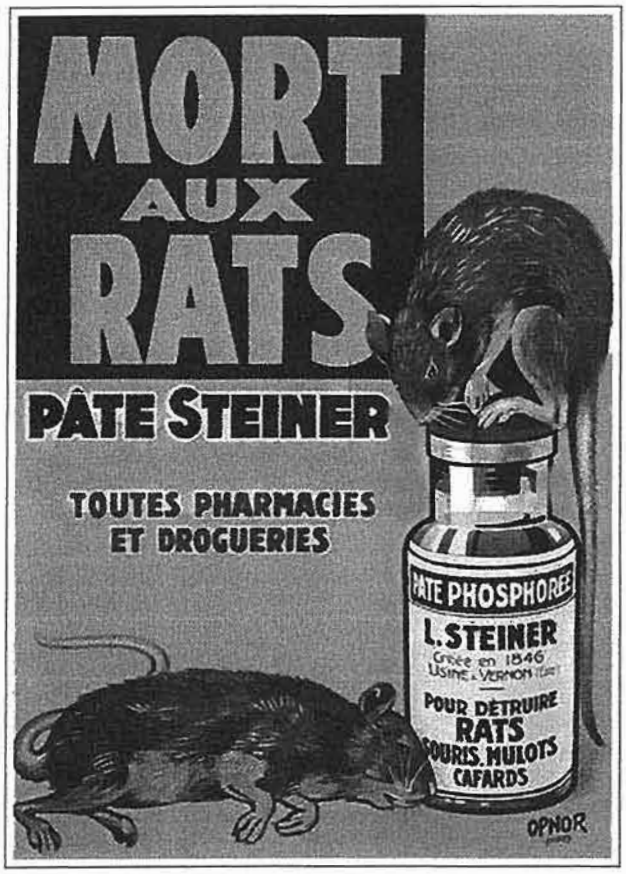
Terrierman's Daily Dose

* 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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We Kill All Zombies

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.

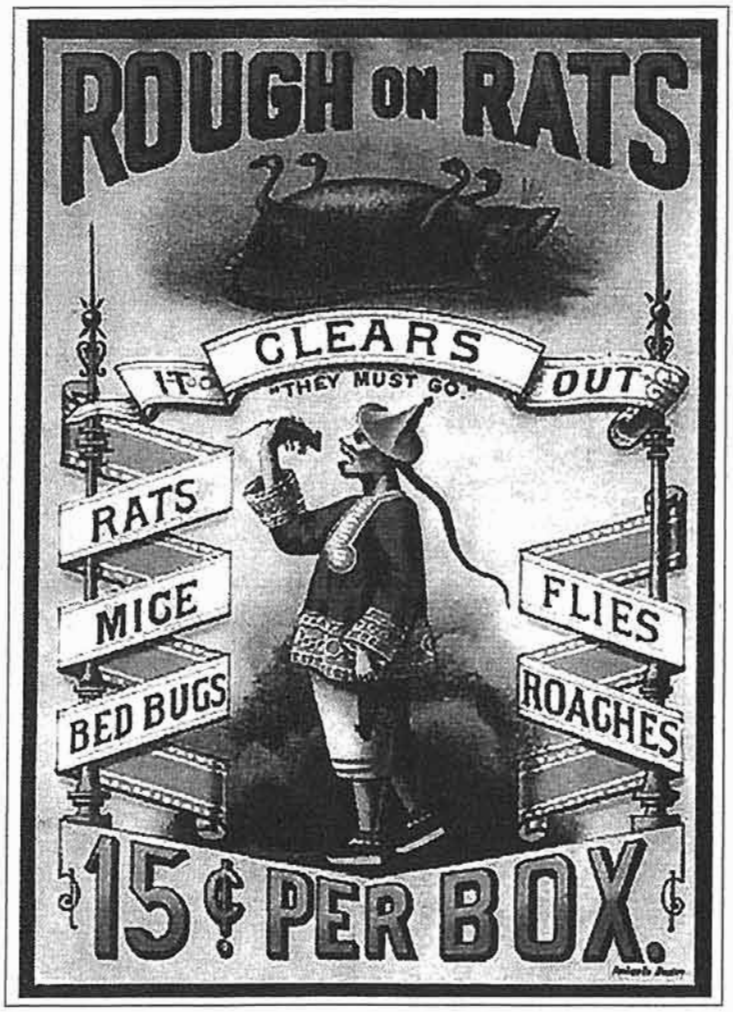
- **Vacor 1080 (Sodium fluoroacetate) is an extremely powerful single-dose rodenticide.** Death normally occurs 4 to 8 hours after ingestion, and little or no bait shyness develops since death generally follows ingestion. Vacor is available in a formulated ready-to-use bait mixture for licensed professional 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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Shelley R. Kramer

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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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CHEMICAL PROFILES | Product Profile

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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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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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

Host site National Institutes of Health

- 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.

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http://www.orgsites.com/ny/nyscof

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Study Proves Fluoridation is Money Down the Drain

NYSCOF View profile More options Sep 29 2009, 6:24 am

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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

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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

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