

Should Natick Fluoridate?

A Report to the Town and the Board of Selectmen
Prepared by the Natick Fluoridation Study Committee
13 E. Central Street, Town of Natick, MA October 23, 1997

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Natick Fluoridation Study Committee Report 9/27/97 Page 2

Cover Letter to the Selectmen

Fluoridation Study Committee
Town of Natick, Town Hall
13 E. Central St.
Natick MA 01760

27 September, 1997
Jay H. Ball, Clerk
Office of the Board of Selectmen
Town Hall
13 East Central Street
Natick, MA 01760

Benedict J. Gallo, Ph. D.
Jason Kupperschmidt, B.
Norman R. Mancuso, Ph.D.
Alfred J. Murray, M. S. T.
Harlee S. Strauss, Ph. D.
Chairman

Dear Selectman Ball:

This letter accompanies transmission of the report "*Should Natick Fluoridate?*" prepared jointly by the Fluoridation Study Committee of the Town of Natick. A summary version of this report is being prepared and copies will be provided as soon as it has been completed. Please advise if we can be of further service to the Board of Selectmen.

Sincerely,

☒

Norman R. Mancuso, Ph. D.
Chairman, Natick Fluoridation Study Committee

Findings, Conclusion, and Recommendations of the Natick Fluoridation Study Committee

Introduction

This statement of Findings, Conclusion and Recommendation specifically addresses the following question of the Board of Selectmen:

On the basis of the documentation provided to you by the proponents and opponents, do you

believe that the potential side effects associated with fluoridating Natick's public water supply outweigh the potential benefits?

Findings The Natick Fluoridation Study Committee conducted a thorough review of the scientific literature and made the following findings regarding the benefits and risks of water fluoridation.

- **Recent studies of the incidence of cavities in children show little to no difference between fluoridated and non-fluoridated communities.**
- **Ten to thirty percent (10-30%) of Natick's children will have very mild to mild dental fluorosis if Natick fluoridates its water (up from probably 6% now). Approximately 1% of Natick's children will have moderate or severe dental fluorosis. Dental fluorosis can cause great concern for the affected family and may result in additional dental bills. It should not be dismissed as a "cosmetic" effect.**
- **Fluoride adversely effects the central nervous system, causing behavioral changes and cognitive deficits. These effects are observed at fluoride doses that some people in the US actually receive.**
- **There is good evidence that fluoride is a developmental neurotoxicant, meaning that fluoride effects the nervous system of the developing fetus at doses that are not toxic to the mother. The developmental neurotoxicity would be manifest as lower IQ and behavioral changes.**
- **Water fluoridation shows a positive correlation with increased hip fracture rates in persons 65 years of age and older, based on two recent epidemiology studies.**
- **Some adults are hypersensitive to even small quantities of fluoride, including that contained in fluoridated water. At least one such person is a Natick resident.**
- **The impact of fluoride on human reproduction at the levels received from environmental exposures is a serious concern. A recent epidemiology study shows a correlation between decreasing annual fertility rate in humans and increasing levels of fluoride in drinking water.**
- **Animal bioassays suggest that fluoride is a carcinogen, especially for tissues such as bone (osteosarcoma) and liver. The potential for carcinogenicity is supported by fluoride's genotoxicity and pharmacokinetic properties. Human epidemiology studies to date are inconclusive, but no appropriate major study has been conducted.**
- **Fluoride inhibits or otherwise alters the actions of a long list of enzymes important to metabolism, growth, and cell regulation.**
- **Sodium fluorosilicate and fluorosilicic acid, the two chemicals Natick intends to use to fluoridate the water supply, have been associated with increased concentrations of lead in tap water and increased blood lead levels in children, based on case reports and a new, as-yet-unpublished study.**
- **If Natick fluoridates its water supply at the proposed level, most children under**

the age of three will daily receive more fluoride than is recommended for them.

The scientific literature supporting these findings is summarized in the full report which also discusses a variety of non-health related concerns that have been raised about water fluoridation

Conclusion

The Committee reached the firm conclusion that the risks of overexposure to fluoride far outweigh any current benefit of water fluoridation.

Recommendations

- 1. The Natick Fluoridation Study Committee unanimously and emphatically recommends that the town of Natick NOT fluoridate the town water supply.**
- 2. The Natick Fluoridation Study Committee unanimously and emphatically recommends that the Board of Selectmen take appropriate action to ensure that fluoridation of the town water supply does not take place.**

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Fluoride: Protected Pollutant or Panacea?
Are the claimed benefits of ingesting fluoride over-rated
and the risks to our health and eco-system under-reported?



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Should Natick Fluoridate?

A Report to the Town
and the
Board of Selectmen

Prepared by the
Natick Fluoridation
Study Committee

13 E. Central Street
Town of Natick, MA
October 14, 1997

Cover Letter to the Selectmen

Benedict J. Gallo, Ph. D.
Jason Kupperschmidt, B.
Norman R. Mancuso, Ph.D.
Alfred J. Murray, M. S. T.
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**Fluoridation Study Committee
Town of Natick**

Town Hall 13 E. Central St. Natick MA 01760

27 September, 1997

Jay H. Ball, Clerk
Office of the Board of Selectmen
Town Hall
13 East Central Street
Natick, MA 01760

Dear Selectman Ball:

This letter accompanies transmission of the report **"Should Natick Fluoridate?"** prepared jointly by the Fluoridation Study Committee of the Town of Natick.

A summary version of this report is being prepared and copies will be provided as soon as it has been completed.

Please advise if we can be of further service to the Board of Selectmen.

Sincerely,



Norman R. Mancuso, Ph. D.
Chairman, Natick Fluoridation Study Committee

Findings, Conclusion, and Recommendations of the Natick Fluoridation Study Committee

Findings

Introduction

This statement of Findings, Conclusion and Recommendation specifically addresses the following question of the Board of Selectmen:

On the basis of the documentation provided to you by the proponents and opponents, do you believe that the potential side effects associated with fluoridating Natick's public water supply outweigh the potential benefits?

Findings

The Natick Fluoridation Study Committee conducted a thorough review of the scientific literature and made the following findings regarding the benefits and risks of water fluoridation.

- Recent studies of the incidence of cavities in children show little to no difference between fluoridated and non-fluoridated communities.
- Ten to thirty percent (10-30%) of Natick's children will have very mild to mild dental fluorosis if Natick fluoridates its water (up from probably 6% now). Approximately 1% of Natick's children will have moderate or severe dental fluorosis. Dental fluorosis can cause great concern for the affected family and may result in additional dental bills. It should not be dismissed as a "cosmetic" effect.
- Fluoride adversely effects the central nervous system, causing behavioral changes and cognitive deficits. These effects are observed at fluoride doses that some people in the US actually receive.
- There is good evidence that fluoride is a developmental neurotoxicant, meaning that fluoride effects the nervous system of the developing fetus at doses that are not toxic to the mother. The developmental neurotoxicity would be manifest as lower IQ and behavioral changes.
- Water fluoridation shows a positive correlation with increased hip fracture rates in persons 65 years of age and older, based on two recent epidemiology studies.

- Some adults are hypersensitive to even small quantities of fluoride, including that contained in fluoridated water. At least one such person is a Natick resident.
- The impact of fluoride on human reproduction at the levels received from environmental exposures is a serious concern. A recent epidemiology study shows a correlation between decreasing annual fertility rate in humans and increasing levels of fluoride in drinking water.
- Animal bioassays suggest that fluoride is a carcinogen, especially for tissues such as bone (osteosarcoma) and liver. The potential for carcinogenicity is supported by fluoride's genotoxicity and pharmacokinetic properties. Human epidemiology studies to date are inconclusive, but no appropriate major study has been conducted.
- Fluoride inhibits or otherwise alters the actions of a long list of enzymes important to metabolism, growth, and cell regulation.
- Sodium fluorosilicate and fluorosilicic acid, the two chemicals Natick intends to use to fluoridate the water supply, have been associated with increased concentrations of lead in tap water and increased blood lead levels in children, based on case reports and a new, as-yet-unpublished study.
- If Natick fluoridates its water supply at the proposed level, most children under the age of three will daily receive more fluoride than is recommended for them.

The scientific literature supporting these findings is summarized in the full report which also discusses a variety of non-health related concerns that have been raised about water fluoridation

Conclusion

The Committee reached the firm conclusion that the risks of overexposure to fluoride far outweigh any current benefit of water fluoridation.

Recommendations

1. The Natick Fluoridation Study Committee unanimously and emphatically recommends that the town of Natick **NOT** fluoridate the town water supply.
2. The Natick Fluoridation Study Committee unanimously and emphatically recommends that the Board of Selectmen take appropriate action to ensure that fluoridation of the town water supply does not take place.

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Background and History

History of the Fluoridation Issue in Natick

In May of 1988, the Natick Board of Health ordered the upward adjustment of fluoride in the town water supply. A petition was filed by more than ten (10%) per cent of the town's registered voters to place the question on the ballot. A public referendum was held on Nov. 8, 1988 on the question:

"Shall the public water supply for domestic use in this Town be fluoridated?"

The voters approved the issue by a vote of 7453 (51.4%) yes to 7044 (48.6%) no. A subsequent referendum was held on Mar. 25, 1997 on the identical question, except that in this case, the status of the question was non-binding. In this latter case, the voters did not approve this question by a vote of 2635 (54.3 %) no to 2220 (45.7 %) yes, a reversal of opinion of almost six (6%) percent.

Subsequent Town Meeting Articles 35 and 36 sought to put aside the 1988 vote and to pass special legislation which would place the issue on the ballot at the next annual town election or biennial state election, whichever was held first. Town Meeting voted to indefinitely postpone both articles.

The Chairmen of the Board of Health and the Selectmen requested a legal opinion from the Town Counsel, John P. Flynn, Esq., which was provided on May 8, 1997. (1)

Appointment of the Natick Fluoridation Study Committee

Recognizing the existence of an incredibly large database of conflicting opinion and that both proponents and opponents on the fluoridation issue were entrenched and unlikely to reach a consensus, on April 28, 1997, the Board of Selectmen voted to appoint a special committee of unbiased and qualified people to study the fluoridation issue and to report back on their findings to the Board of Selectmen within approximately ninety (90) days. Hearings at two subsequent Selectmen's meetings produced a number of qualified, scientifically trained and experienced people and from that group, the Natick Fluoridation Study Committee (hereinafter NFSC) was formed by the Board of Selectmen. (2) The present document constitutes the report requested by the Board.

Recommendation and Charter of the NFSC

Mechanism of Approach to be Taken

Since an exhaustive literature search of the issues of fluoride and fluoridation could consume the resources of the committee for several months, the Board of Selectmen recommended that the committee obtain from the proponents and opponents of the issue suitable documentation with which to begin the pursuit of the resolution of the issue. (3)

The fluoridation proponents in Natick consist of the Natick Board of Health (hereinafter BOH) and those citizens of the town who are similarly disposed on the issue of fluoridation. A letter was sent by NFSC to the BOH on July 3, 1997 requesting that the BOH provide:

Five sets of documents, each comprised of a maximum of six study reports which in the opinion of the BOH most clearly explain why the public water supply of Natick *should* be fluoridated.

In addition, a maximum of six endorsements, letters and other non-data-intensive documents that support their contention that fluoridation is *both beneficial and lacking in undesirable side effects*.

On August 11, the BOH complied with this request by providing five copies of each of the above sets of documents (see Appendix A - Source Materials).

The opponents of the fluoridation issue in the town are represented by Ms. Shirley Brown of Megonko Road in Natick and Dr. Myron Coplan of Intelleguity, Inc., also of Natick. Each of these individuals were also requested on July 3, 1997 to provide to NFSC similar packets of materials as follows.

Five sets of documents, each comprised of a maximum of three study reports which in the opinion of the opponents most clearly explain why the public water supply of Natick *should not* be fluoridated.

In addition, a maximum of three endorsements, letters and other non-data-intensive documents that support their contention that the *undesirable side effects* of fluoridation outweigh its beneficial effects.

The above materials were provided immediately by Ms. Brown and on July 9, 1997 by Dr. Myron Coplan (see Appendix A - Source Materials).

In this manner, it was anticipated that the most driving arguments for and against the issue would be immediately present, as would be the best reference list(s) of supporting original research papers. All of the above notwithstanding, the NFSC was not constrained to limit its search to the above materials and has instituted wide-ranging literature searches on the entire issue, often spending several hours per day in reading and/or pursuing further information.

Charter of the Fluoridation Study Committee

The Board of Selectmen, in appointing this committee, established the need to address several issues. (4) These are:

- 1. On the basis of the documentation provided to you by the proponents and opponents, do you believe that the potential side effects associated with fluoridating Natick's public water supply outweigh the potential benefits?*
- 2. If your answer to question 1 is "No" -- i.e. you believe Natick's water should be fluoridated -- do you believe that steps should be taken to establish the appropriate dosage before such fluoridation begins?*
- 3. If your answer to question 2 is "Yes", do you believe that an outside organization should be engaged to examine Natick school children and determine their DMFS (decayed, missing and filled surfaces) levels as an aid to selecting an optimum fluoridation level?*
- 4. If your answer to question 3 is "Yes", what organizations (identify at least two) are qualified to conduct such a survey, and what are preliminary estimates of the costs involved?*

Format of the Report

The format being used for this report is based upon the four *charges* of the Board of Selectmen with regard to the fluoridation issue. Each *charge* or question constitutes one section of the report. The individual issues addressed within each section result from the nature of the associated charge or question. In any publication of this nature, the issue of providing references must be addressed. On the one hand, material for public consumption is rarely well-received when numerous references are included. This is particularly so when the references take the form of foot-notes as opposed to end-notes. **On the other hand, nothing makes a report such as this more suspect than when no verifiable references are included and the reader is tacitly expected to accept the discussion and conclusions on faith alone.** We feel strongly enough about this issue to insist on the presence of references. The only relaxation of this requirement was an attempt to reduce the tedium that would be caused by having to constantly flip to the end of the report to find a reference or

explanatory note. This was accomplished by placing all of the references for each section immediately following that particular section. This has the necessary consequence of occasionally having more than one occurrence of the same reference but improves the readability and overall usefulness to the reader.

General Background of the Fluoridation Issue

Scientific information that has an impact on political and economic interests often generates controversy, even within the scientific community. This controversy is often magnified when the information is presented in the lay press. It is not that truly non-partisan reporting is unavailable, rather it is that the entrenched partisans of any such issue are only willing (able?) to accept those portions of the report which support their contentions. Be that as it may, there are some reviews of the fluoridation issue that are unbiased, chief among which is a review by Bette Hileman appearing in Chemical & Engineering News. (5) As background material, it is required reading for anyone interested in the issue of fluoridation. A brief portion of this review is quoted below in order to set the stage for further discussion. (Contrary to normal usage, direct quotes appearing within the text below are emboldened and not italicized in order to distinguish them from the remaining commentary.)

Throughout this report, the reader will note the recurrent use of the words "**optimal**", "**optimum**" and such phrases as "**optimally fluoridated**". It is important to understand that this usage is a direct contribution of the profluoridation argument and is therefore vigorously objected to by the antifuoridation contingent. In most cases, we have placed quotation marks around these words and phrases to indicate that the term is disputed and should be read as "**so-called optimum**".

".....The style of promotion that fluoridation's proponents have used from the very beginning probably made the issue more controversial than it need have been.

The idea of fluoridating water supplies first arose from studies of dental mottling in areas, such as communities in Texas, where the water supply is fluoridated naturally. In the 1930s, H. Trendley Dean, a dental surgeon at the U.S. Public Health Service, correlated the occurrence of mottling or dental fluorosis with the fluoride content of water supplies in 345 U.S. communities. Fluorosis was most common in cities that had the highest concentration of fluoride in their water. He and his colleagues also unexpectedly found a lower incidence of dental caries in areas of endemic dental fluorosis.

Dean concluded that the fluoride content of the drinking water causes a lower rate of dental caries. He also determined that the incidence of mottling was very minor when the fluoride content was 1 ppm or lower but rose linearly at higher concentrations. From this, PHS officials decided in 1943 that 1 ppm was an optimal level at which to artificially fluoridate water supplies in temperate climates. In areas where the fluoride

content exceeded 2 ppm, they recommended fluoride be reduced to a level near 1 ppm.

In 1945, PHS initially planned to conduct 10-year studies of artificial fluoridation in two experimental projects, one in New York and one in Michigan. One city in each state would be fluoridated artificially and another would serve as a control. PHS officials intended to complete these projects before deciding whether to recommend fluoridation of drinking water as a general practice for all communities.

However, two public health officers in Wisconsin, Francis A. Bull and John Frisch, quickly became convinced of the effectiveness of fluoridation and launched a nationwide campaign to persuade PHS to endorse it. Also, results from the two projects that leaked out in 1950, after the trials had been going on for five years, revealed a sharp reduction in dental caries in the fluoridated cities. As a result of this disclosure and Bull's and Frisch's campaign, PHS officials endorsed fluoridation on June 1, 1950.

Several deficiencies in research by PHS were subsequently aired at Congressional hearings in 1952 and 1957. There had been almost no careful studies to assess the possible adverse health effects of lifelong consumption of fluoridated water. Aside from their dental health, the medical condition of residents of naturally fluoridated areas had been examined superficially, at best. In one of the fluoridation trials, research plans included a study of adverse effects of artificial fluoridation on children, but none on adults. No studies focused on malnourished children and infants, despite a warning in 1952 by Maury Massler, professor of pedodontics at the University of Illinois College of Dentistry, that **"low levels of fluoride ingestion which are generally considered to be safe for the general population may not be safe for malnourished infants and children, because of disturbances in calcium metabolism."**

Neither PHS nor anyone else had investigated potential carcinogenic effects, effects on pregnant women, or effects on people with chronic kidney impairment or other chronic diseases. Even in the early 1950s, enough was known of fluoride's toxicity profile to identify these as important topics to investigate.

From the beginning, the movement to fluoridate water was conducted more like a political campaign than a scientific enterprise. At a meeting of state dental directors with PHS officials in June 1951, Bull recommended tactics for promoting fluoridation. **"If it is a fact that some individuals are against fluoridation, you just have to knock their objections down. The question of toxicity is on the same order. Lay off it altogether. Just pass it over. 'We know there is absolutely no effect other than reducing tooth decay,' you say, and go on. If it becomes an issue, then you will have to take it over, but don't bring it up yourself."**

"The minute doubt is created in the minds of the public, any public health program is doomed to failure," Bull later wrote in the *Journal of the American Dental Association*.

The political role of dentists has been emphasized throughout the history of fluoridation. In 1970, even after 25 years of fluoridation, John W. Knutson, then

professor at the University of California Medical Center, advised dentists that when they discussed fluoridation with the public, they must realize that "they are propagandizing, not simply educating." This attitude, widely shared by political proponents, led early advocates to treat fluoridation campaigns as debates to be won with dogmatic assertions and attacks on the credibility of the opposition. To promoters, the debate has never been seen as a scientific search for truth.

As a result, profluoridationists prepare booklets for the public that contain highly biased information. If scientific studies are cited, only those that support their side of the argument are mentioned. Those opposed to fluoridation counter with equally biased propaganda....." (5)

According to many opponents of fluoridation, other tactics which were also widely used to denigrate any potentially negative effects include character assassination, inflammatory portrayal of the opposition, the widespread suppression of opposing results (see Appendix B) and the widespread use of sensationalism, etc. In the latter case at least, the antifuoridationists are no less culpable.

References

1. Flynn, JP, *Letter from the Town Counsel to the Bds. of Health & Selectmen*, May 8, 1997.
2. Challis, D, *Letter from the Bd. of Selectmen to NR Mancuso*, June 23, 1997.
3. Ball, J, *Thoughts on the Fluoridation Study Committee*, Memo to the Bd. of Selectmen, May 15, 1997.
4. Challis, D, *Letter from the Bd. of Selectmen to NR Mancuso*, July 30, 1997.
5. Hileman, B, *Fluoridation of Water*, Chem. & Eng. News, 66, 26 (1988).

Question 1 An Analysis of the Side Effects of Fluoridation

- 1. On the basis of the documentation provided to you by the proponents and opponents, do you believe that the potential side effects associated with fluoridating Natick's public water supply outweigh the potential benefits?*

This is the main question to be addressed in this report. It also includes the reasons why the issue of fluoridation is so controversial. In spite of the other topics presented in this report, the main issue remains whether the benefits of fluoridation outweigh the risks. Moreover, it appears that the only significant benefit of fluoridation is the reduction of dental caries, this in spite of other past reports touting the applicability of fluoridation to osteoporosis as well (see the section on other positive effects of fluoridation). The "profluoridationists" have repeatedly asserted that there are no negatives associated with the process, or alternatively, that all of the negative reports are without scientific justification or merit. Because of this position, an examination of these negative reports tends to cast the examiners in the role of a "devil's advocate", the chief difficulty of which is that the examiners are then also perceived as being "antifluoridationists", when in fact they are merely seeking to extract the truth from the polemics and hysteria of the issues and to expose this information to a critical and unbiased analysis. With this in mind we report on the following material.

The Beneficial Effects of Fluoridation

History of the Fluoridation Program in the United States

During the course of dental research conducted in the early part of this century on the condition then known as "Colorado Brown Stain" (a.k.a. "Texas Teeth" or dental fluorosis as it came to be medically known), it was discovered that individuals, living in areas where the water is known to contain elevated (relative to most water supplies) fluoride concentrations, exhibited a decreased rate of incidence of dental caries. (1) Several studies conducted during the decades prior to 1960 confirmed that when a small quantity (ca. 1 part per million, ppm) of fluoride was added to a community water supply, the incidence of tooth decay among the residents of those community decreased substantially. (2) The initial studies indicated a reduction in tooth decay of 50 to 60 per cent. (3) As a result of these achievements, the process of fluoridation of community water supplies has continued and resulted in more than half of the U. S. population being served by a fluoridated supply. (4) Numerous scientific papers have supported fluoridation throughout its history. (5-9) More recent studies, as interpreted by profluoridationists, indicate that reductions of between 20-40% are routinely achievable. (10-12)

Features of the Fluoridation Program

The desirability of the process of fluoridation of community water supplies, as maintained by the profluoridation community, is based upon the following reasoning: (13)

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

Supporters of Community Water Fluoridation

The following non-exhaustive list illustrates the wide-spread support for the fluoridation programs. (13, 14)

- Mass. Dept. of Public Health
- American Association of Public Health Dentistry
- American Dental Association
- Centers for Disease Control & Prevention
- American Medical Association
- World Health Organization

Other Positive Effects of Fluoridation

Proponents of fluoridation have also attempted to show that fluoride can be used to alleviate the symptoms of osteoporosis, and therefore that people living in fluoridated areas may be helped by the fluoride they are accumulating in their bones. Because fluoride increases bone mass, (see the section on Osteosclerosis) numerous patients have been given and are still being given large doses of fluoride as a treatment for osteoporosis. Recent data has not produced compelling evidence of beneficial results. The FDA has not approved the use of fluoride for osteoporosis. In spite of this, the National Osteoporosis Foundation reports that an FDA advisory committee has recommended that slow-release sodium fluoride be approved for the treatment of osteoporosis. (15)

References

1. Black, GV, and McKay, FS, *Mottled teeth: an endemic developmental imperfection of the enamel of teeth, heretofore unknown in the literature of dentistry.*, Dent. Cosmos, **58**:129-156, (1916)
2. Cox, GJ, *New knowledge of fluorine and its relation to dental caries.*, J. Am. Water Works Assoc., **31**:1926-1930, (1939)
3. Arnold, F, Jr., et al., *Fifteenth year of the Grand Rapids fluoridation study.*, J. A. D. A., **65**:781, (1962)
4. U. S. Dept. Health and Human Services, *1985 Fluoridation Census*, Atlanta, (1988)
5. Ast, D, and Fitzgerald, B, *Effectiveness of water fluoridation.*, J. A. D. A., **74**:2, (1967)
6. French, AD, et al., *Fluoridation and dental caries experience in 5-year-old children in Newcastle and Northumberland in 1981.*, Br. Dent. J., **156**:54-57, (1985)
7. Murray, J, *Efficacy of preventive agents for dental caries, systemic fluorides: water fluoridation.*, Caries Res. **27**:2-8 (1993)
8. Burt, BA, et al., *Dental benefits of limited exposure to fluoridated water in childhood.*, J. Dent. Res., **61**:1322-1325, (1986)
9. Jackson, D, *Has the decline of caries in English children made water fluoridation both unnecessary and uneconomic?*, Br. Dent. J., **165**:170-173, (1987)
10. Newbrun, E, *Effectiveness of water fluoridation.*, J. Pub. Hlth. Dent., **49**:279-289, (1989)
11. Lewis, DW and Banting, DW, *Water fluoridation: current effectiveness and dental fluorosis.*, Comm. Dent. & Oral Epidem., **22**:153-158, (1994)
12. Brunelle, JA and Carlos, JP, *Recent trends in dental caries in US children and the effect of water fluoridation.*, J. Dent. Res., **69**:723-727 (1990)
13. Mass. Dept. of Public Health, *Community Water Fluoridation*, (undated brochure)
14. American Dental Association, *Fluoridation Facts*, (1992[?]), 29 pp
15. National Osteoporosis Foundation, *FDA Advisory Committee Recommends Approval of Sodium Fluoride Treatment for Osteoporosis*, News Release, Nov. 17, 1995

Refutation - Water Fluoridation today is at best only minimally effective

Fluoride was first investigated as an anti-caries agent because of the inverse relationship noted in many areas of the country between the prevalence of dental caries and the level of fluoride in drinking water. At first, scientists believed that the anti-caries activity of fluoride was the direct result of its incorporation into the apatite crystal of enamel, thus increasing its stability and reducing its acid solubility. The theory of pre-eruptive fluoride incorporation as the principal mechanism of caries prevention has been largely discounted. (1) Recent studies have suggested that the anti-caries action of fluoride may be related to the fluoride levels in the saliva and plaque fluids rather than the enamel surface itself, i.e., the action is topical rather than systemic. (2,3) Indeed, if one diligently searches the literature of fluoridation, it becomes clear that there are widespread differences of opinion among experts as to the actual mechanism. Moreover, it is significant that in one survey, only 66% of physicians thought that community fluoridation is very effective and only 37% think that dietary supplements are very effective. (4) This same survey reported that only a small percentage of physicians and dentists believe that topical fluorides are very effective preventive measures, so it is clear that even among *"those who should know"*, there is a large measure of discordant opinion.

The sources of fluoride intake for the U. S. population are primarily water, food, dental products and air (see Tables I & II). Children may also receive fluoride in supplements. Although fluoride exposure is generally greater in areas with fluoridated water than in areas with non-fluoridated or low-fluoridated water, populations in both areas are exposed to fluoride from food sources, drinking water, processed beverages and dental products. In one recently published survey, Dabeka and McKenzie have reported that the average intake of fluoride from food, averaged over all ages and sexes, was 1.76 mg/day. (5) Fluoride exposure differs markedly, depending upon several factors, e.g., lifestyle, dietary practices, age, gender and health status. It is clear however that drinking water provides minimal topical fluoride. The Agency for Toxic Substances and Disease Registry (ATSDR) sets the Minimal Risk Level (MRL) for ingestion of fluoride at 0.4 mg/kg/day. (6) In a 20 pound child this amounts to 3.6 mg/day and for a 50 pound child, the minimal risk level is about 9 mg/day. The MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure. However, to avoid an undesirable degree of dental fluorosis, children should consume no more than 0.10 mg of fluoride per kg of body weight per day. (7)

Table I - Fluoride Concentrations (ppm) in Food

Foods (Note A)	Mean (ppm)	Standard Deviation	Range (ppm)
Dairy Products	0.25	0.38	0.02 - 0.82
Meat Fish & Poultry	0.22	0.15	0.04 - 0.51
Grain & Cereal Products	0.42	0.40	0.08 - 2.01
Potatoes	0.49	0.26	0.21 - 0.84
Leafy Vegetables	0.27	0.25	0.21 - 0.84
Legumes	0.53	0.05	0.49 - 0.57
Root Vegetables	0.38	0.11	0.27 - 0.48
Fruits	0.06	0.03	0.02 - 0.08
Oils & Fats	0.25	0.15	0.02 - 0.44
Sugar and Adjuncts	0.28	0.27	0.02 - 0.78
Nonclassifiable Foods	0.59	0.19	0.29 - 0.87

Note A The foods were ready to eat or prepared for eating. When preparation required the use of water (e.g. preparing juice from concentrate or boiling vegetables), the local water was used which contained 1 mg/L (1 ppm) of fluoride was used. Nonclassifiable foods included certain soups and puddings, among other items. (1)

Table II - Estimated Daily Fluoride Intake of Children (1)

F Concentration in Water (ppm)	Intake from Food	Intake from Beverages	Intake from Dentifrices	Intake from F Supplements	Estimated Total Intake
<0.3	0.15 - 0.30	0.10 - 0.30	0.20 - 1.20	0.50	0.95 - 2.30
0.7-1.2	0.40 - 0.60	0.30 - 1.30	0.20 - 1.20	NR	0.90 - 3.60
>2.0	1.00 - 2.00	0.60 - 3.00	0.20 - 1.20	NR	1.80 - 6.20
Units	mg/day	mg/day	mg/day	mg/day	mg/day
Notes			(a)	(b)	

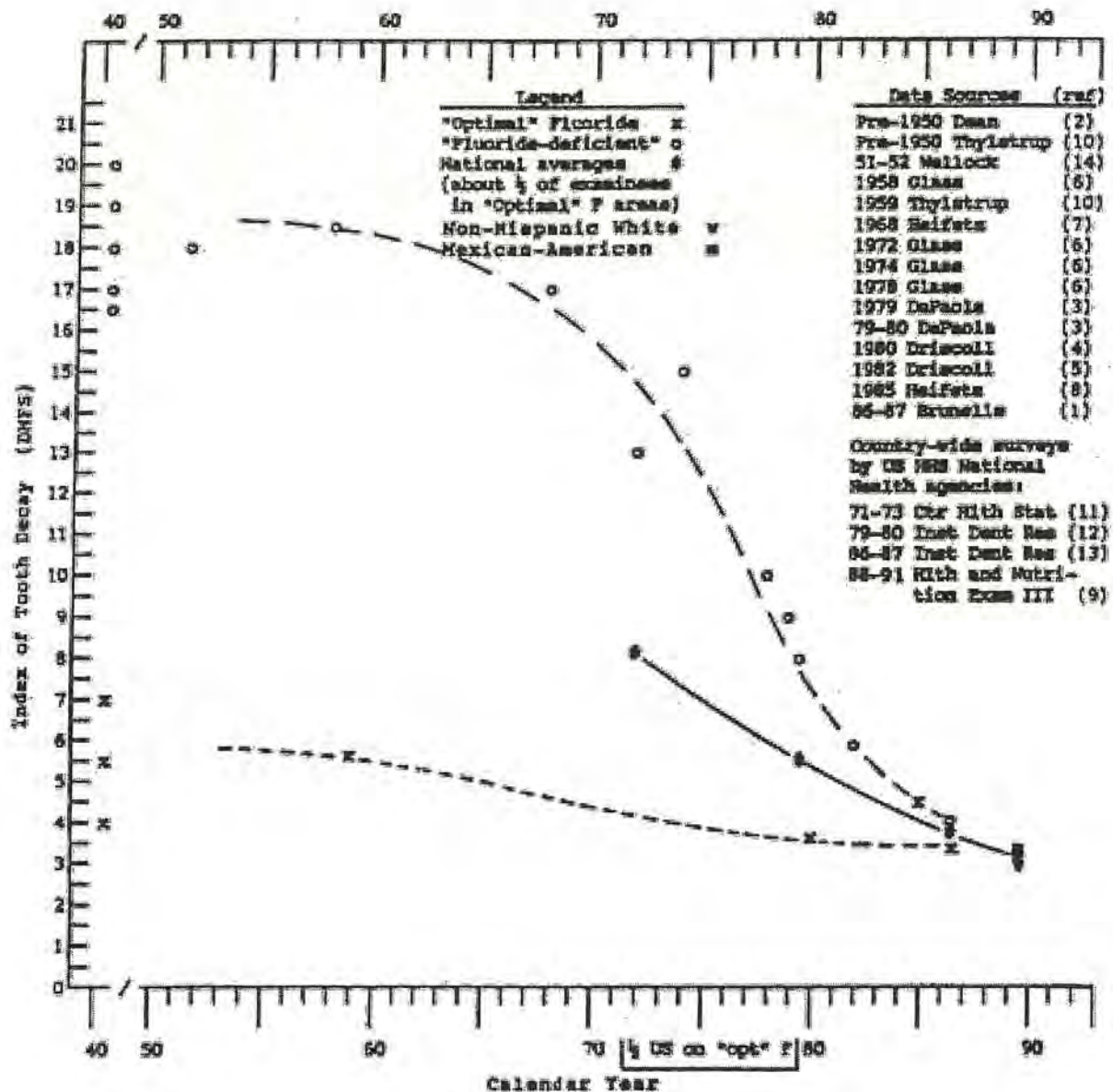
(a) Assumed that dentrifice used twice daily

(b) Assumed that dentrifice fluoride supplement taken daily

Studies show that tooth decay has declined in every country in the developed world. (2,3,9) The largest study in this country (the NIDR 1986-87 survey of 39,000 U.S. school children, (1)) showed no significant difference in dental caries in those living in fluoridated as opposed to those living in non-fluoridated communities. (11) See Fig. I.

Figure I - Decline in DMFS Index in 12-14 Year Olds (12)

50-YEAR HISTORY OF TOOTH DECAY PREVALENCE AMONG 12-14 YEAR-OLDS LIVING IN "OPTIMAL FLUORIDE" AND "FLUORIDE-DEFICIENT" AREAS



References Cited in Figure I

1. Brunelle, JA and Carlos, JP; *Recent Trends in Dental Caries in U. S. Children and the Effect of Water Fluoridation.*, Journal of Dental Research; **69**(Special Issues);723-727, (1990)
2. Dean, HT, *Epidemiologic Studies in the United States.*, via Moulton, FR, *Fluorine and Dental Health.*, Washington, DC. 1942.
3. DePaola, PF, et al., *Dental Survey of Massachusetts Schoolchildren.*, Journal of Dental Research; **61**(Special Issue):1356-1360; (1982)
4. Driscoll, WS, et al., *Prevalence of Dental Caries and Dental Fluorosis in Areas with Optimal and Above-Optimal Water-fluoride Concentrations.*, Journal of the American Dental Association; **107**:43-47, (1983)
5. Driscoll, WS, et al., *Prevalence of Dental Caries and Dental Fluorosis in Areas with Negligible, Optimal, and Above-Optimal Fluoride Concentrations in Drinking Water.*, Journal of the American Dental Association; **113**:29-33, (1986)
6. Glass, RL.; *Secular Changes in Caries Prevalence in Two Massachusetts Towns.*; Caries Research; **15**:445-450, (1981)
7. Heifetz, SB, et al., *Effect of School Water Fluoridation on Dental Caries: Results in Seagrove, NC after 12 Years*; Journal of the American Dental Association, **106**:334-336, (1983)
8. Heifetz, SB, et al., *Prevalence of Dental Caries and Dental Fluorosis in Areas with Optimal and Above-Optimal Fluoride Concentrations: A 5-Year Follow-Up Survey.*; Journal of the American Dental Association, **116**:490-495, (1988)
9. Kaste, LM, et al., *Coronal Caries in the Primary and Permanent Dentition of Children and Adolescents 1-17 Years of Age: United States, 1988-91.*, Journal of Dental Research; **75**(Special Issue):631-641, (1996), (Source for 1988-91 NHANES data).
10. Thylstrup, A, *Clinical Evidence of The Role of Pre-eruptive Fluoride in Caries Prevention.*, Journal of Dental Research; **69**(Special Issue):742-750, (1990)
11. US PHS, Special dental tape (unpublished) from 1971-73 National Health Survey HANES I conducted by National Center for Health Statistics via NIH Pub. No. 82-2245: Dec 1981.
12. US PHS, *The Prevalence of Dental Caries in United States Children.*, NIDR National Survey 1979-80, NIH Pub. 82-2245 (1981)
13. US PHS, *Oral Health of United States Children*, NIDR National Survey 1986-87; NIH Pub. No. 89-2247.
14. Wellock, W, *Report of the Special Commission on Dental Health and of Measures to Eliminate Dental Decay Including Fluoridation of Community Water Supplies.*, Commonwealth of Massachusetts, House No. 3902; December 1967 (via Ref. 3).
- A. (A) US PHS, *Review of Fluoride Benefits and Risks.*, Report of the Ad Hoc Subcommittee on Fluoride; Un-numbered publication of the Department of Health and Human Services, September 1991
- B. (B) US PHS, *Water Fluoridation A Manual for Water Plant Operators.*, Center for Disease Control and Prevention, Division of Oral Health, Atlanta, April 1994.
- C. Hilderbolt CF, et al., *Caries Prevalences Among Geochemical Regions of Missouri.*, American Journal of Physical Anthropology **78**:79-92, (1989)
- D. Leroux BG, et al., *The Estimation of Caries in Small Areas.*, Journal of Public Health Dentistry, **75**:1947-56, (1996)
- E. Moreno EG, et al., *Fluoridated hydroxyapatite Solubility and Caries formation.*, Nature **247**:64-65, (1974)
- F. Pendrys DG, and Stamm JW, *Relationship of Total Fluoride Intake to Beneficial Effects and Enamel Fluorosis.*, Journal of Dental Research **69**:529-538 (1990)
- G. Evans RW and Stamm JW, *Dental Fluorosis Following Downward Adjustment of Fluoride in Drinking Water.*, Journal of Public Health Dentistry, **52**(2):91-98, (1991)

In British Columbia, only 11% of the population lives in areas containing fluoridated water, as opposed to 40-70% in other Canadian regions. However British Columbia has the lowest rate of tooth decay in Canada. (10) According to a 1987 report by Dr. Allan Gray, then director of the Division of Dental Health services for British Columbia, DMFT (decayed, missing or filled teeth) rates (see Appendix D) were falling drastically in both fluoridated and non-fluoridated areas. (11)

Mark Diesendorf, an applied mathematician and health researcher in the Human Sciences Program at Australian National University has found, by comparing results from about 24 studies of unfluoridated districts in eight countries, that reductions in dental caries are just as great in non-fluoridated areas as in fluoridated areas. (2)

One of the most significant factors in any comparison of the incidence of dental caries is the manner in which this is expressed (see Appendix D for a discussion of the methods of describing these incidence rates). In the early days of DMFS (decayed, missing or filled surfaces) scores (in the range of 18-20), a 20% difference (fluoridated vs. non-fluoridated) would indicate several cavities per child. However, given the current average DMFS scores (in the range of 2-4) it is clear that a 20% difference represents less than one cavity per child.

Summary

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

References

1. The Ad Hoc Sub-Committee on Fluoride of the Committee to Coordinate Environmental Health and Related Programs, U. S. Public Health Service, Department of Health and Human Services, *Review of Fluoride Benefits and Risks.*, (1991)
2. Diesendorf, M, *The mystery of declining tooth decay.*, *Nature*, **322**:125-129, (1986)
3. Colquhoun, J, *Some investigations into the 'DMF' measurement of fluoride dental benefit.*, *Fluoride* **23**(2):111-118 (1990)
4. Gift HC, and Hoerman KC, *Attitudes of dentists and physicians toward the use of dietary fluoride supplements.*, *A. S. D. C. Dent. Child.*, **52**(4):265-268, (1985)
5. Dabeka RW, McKenzie AD, *Survey of lead, cadmium, fluoride, nickel, and cobalt in food composites and estimation of dietary intakes of these elements by Canadians in 1986-1988.*, *J. A. O. A. C. Int.*, **78**(4):897-909, (1995)
6. Agency for Toxic Substances and Disease Registry (ATSDR), *Toxicological Profile for Fluorides, Hydrogen Fluoride and Fluorine.*, April, 1993
7. Levy, S, et al., *Sources of fluoride intake in children.*, *J. Pub. Health Dent.*, **55**:39, (1995)
8. Hileman, B, *Fluoridation of water*, *Chem. & Eng. News*, **66**:26-42, (1988)
9. Kalsbeek, H and Verrips, GHW, *Dental caries prevalence and the use of fluorides in different European countries.*, *J.Dent. Res.* **69**:728-732, (1990)
10. Gray, A, *Fluoridation: time for a new baseline.*, *J. Can. Dent. Assoc.*, **10**:763-764 (1987)
11. Yiamouyiannis, JA, *Fluoridation*, *Fluoride*, **23**:55-67, (1990)
12. Coplan, MJ, *Fifty Years of Tooth Decay and Fluoridation*, (unpubl.) (1997)

Toxic Effects of Fluoridation and Inorganic Fluoride

The next several sections deal with those issues which have been raised as being either detrimental or non-beneficial side-effects caused by the fluoridation process and/or the ubiquity of various forms of (chiefly) inorganic fluoride compounds in the environment or as used in the treatment of dental caries. Each section analyzes the scientific literature, paying particular attention to those primary research results which have appeared recently. Each section is comprised of both a discussion as well as a summary with respect to the particular issue treated.

The Acute Toxicity of Fluorine-Containing Materials

"Fluoride is a halogen with unique properties that enable it, in toxic quantities, to alter calcium availability to tissues, to effect changes to blood clotting parameters, to cause severe cardiac dysfunction, to alter bone and tooth structure, to cause severe anxiety in those who drink fluoridated water and to lead to death where it has been misused either accidentally, in suicide or in homicide."(1,2)

The principal uses of fluoride include prophylaxis of dental caries, toothpastes, animal husbandry, timber preservation and pesticides. Both sodium fluoride and sodium fluorosilicate have been widely used as insecticides and rodenticides. (9) Ellenhorn and Barceleaux list the therapeutic dose at 0.25 to 0.50 milligrams per day (mg/d) and the oral lethal dose as 5-10 grams of sodium fluoride, (3) although less than 1 gram by mouth has caused severe poisoning. (4) Overfluoridation has resulted in mass intoxication (5) and death has resulted from ingesting 2 g of fluoride. (10)

Waldbott and others (6,7) have also remarked upon the extremely small safety margin between therapeutic doses and those causing toxic effects in humans, when compared to safety margins normally employed in medicinal products.

H. C. Hodge, a toxicologist of wide repute, notes that the minimum safety factors in the dietary regimen of any toxic material should be at least one hundred times the therapeutic dose. (8) Other workers in the field consider even this safety margin far too narrow.

Summary and Conclusions on Acute Toxicity

On the basis of acute toxicity, it is unreasonable to take a strong position against water fluoridation. On the other hand, there are questions about the nature and significance of chronic toxicity effects which must be addressed, as in the case of proper therapeutic quantities of fluoride. Moreover, the question which must be

resolved prior to taking such a position appears to be the determination of what constitutes the proper or safe and effective therapeutic dose. This question has great significance to many of the subjects treated in the following sections. Therefore, Appendix C is included to provide the responses to this question by a number of independent organizations as well as to provide guidelines relative to the total daily individual ingestion of fluoride for different age groups.

References

1. Throughout this document, the use of the term fluoride refers to the materials to be used in process of fluoridation. When a specific fluorine-containing compound is being referenced, the full name will be used, as, e. g., sodium fluoride, or the chemical formula, in this case NaF.
2. Ellenhorn, MJ and Barceloux, DG, *Medical Toxicology*, p. 531, Elsevier (1988)
3. Haynes, RC, Jr., in Gilman, AG, et al., (Eds.), *The Pharmacological Basis of Therapeutics*, 6th Ed., MacMillan, p. 1524, (1980)
4. Greenwood, DA, *Fluoride intoxication*, *Physiol. Rev* **20**, 582 (1940)
5. Waldbott, GL, *Mass intoxication from accidental overfluoridation of drinking water*, *Clin. Toxicol.*, **18**, 531 (1981)
6. Waldbott, GL, *Fluoridation: The Great Dilemma*, p. 107, Coronado Press (1978)
7. Schour, I, and Smith, MC, in *Fluorine and Dental Health*, Moulton, F. R., Ed., (1942)
8. Hodge, HC, *Research needs in the toxicology of food additives*, *Food Cosmet. Toxicol.*, **1**:25, (1963)
9. Agency for Toxic Substances and Disease Registry, Clement International Corp. *Toxicological profile for fluorides, hydrogen fluoride and fluorine (F)*. U.S. Dept. Health Human Services, Public Health Service, TP-91/17, (1993).
10. *The Merck Index*, (Eighth Ed.) p. 959, (1968)

Dental Fluorosis - An Undesirable Effect of Fluoridation

Varying amounts of fluoride are found naturally in the water supplies of many communities, including Natick. Natick's water has only a small amount of fluoride, on the order of about 0.1 ppm (0.1 mg fluoride per liter water). (1) If too much fluoride is ingested by children it results in a toxic dental condition known as dental fluorosis. This condition is marked by visible mottling and/or discoloring of tooth enamel, pitting of the enamel and disturbed tooth shape. (2, 3) Teeth with moderate dental fluorosis typically ***"....may have yellow and brown strains..... they are pitted, brittle, and susceptible to fracture."*** Severe dental fluorosis ***"...not only produces unattractive teeth but also may increase the risk of tooth loss because it destroys parts of the protective enamel."*** (4) Historically, dental fluorosis was first noted in children who grew up in areas where the drinking water supplies had a relatively high content of dissolved fluoride as shown in Table I which also lists communities with little or no dissolved fluoride in their water. Children in these latter communities had very little dental fluorosis. (5) It was also noted that children with dental fluorosis had fewer cavities. (2) Thus began the start of the "fluoride tradeoffs" which resulted in 80% to 90% of "treated" children with fewer cavities and 10% to 20% of those with dental fluorosis.

Table I - Dean's Survey(s) of Dental Fluorosis 1939-1940

Percentages of Children Experiencing Various Degrees of Dental Fluorosis

City	Year	N	F** (ppm)	Very Mild	Mild	Moderate	Severe	Total
Waukegan IL	1939	423	0.0	0.2	0.0	0.0	0.0	0.2
Oak Park IL	1939	329	0.0	0.6	0.0	0.0	0.0	0.6
Evanston IL	1939	256	0.0	1.6	0.0	0.0	0.0	1.6
Mi City IN	1940	236	0.1	0.0	0.0	0.0	0.0	0.0
Quincey IL	1940	330	0.1	0.3	0.0	0.0	0.0	0.3
Elkhart IN	1940	278	0.1	0.4	0.0	0.0	0.0	0.4
Portsmouth OH	1940	469	0.1	1.3	0.0	0.0	0.0	1.3
Middletown OH	1940	370	0.2	1.1	0.0	0.0	0.0	1.1
Zanesville OH	1940	459	0.2	1.5	0.0	0.0	0.0	1.5
Lima OH	1940	454	0.3	2.2	0.0	0.0	0.0	2.2
Marion OH	1940	263	0.4	5.3	0.8	0.0	0.0	6.1
Elgin IL	1939	403	0.5	3.5	0.7	0.0	0.0	4.2
Pueblo CO	1940	614	0.6	6.2	0.3	0.0	0.0	6.5
Kewanee IL	1939	123	0.9	10.6	1.6	0.0	0.0	12.2
Aurora IL	1939	633	1.2	13.9	1.1	0.0	0.0	15.0
Joilet IL	1940	447	1.3	22.2	3.2	0.0	0.0	25.3
E Moline IL	1940	152	1.6	29.6	2.0	0.0	0.0	32.0
Maywood IL	1939	171	1.6	29.2	4.1	0.0	0.0	33.3
Elmhurst IL	1939	170	1.8	30.0	8.8	1.2	0.0	40.0
Galesburg IL	1940	273	1.9	40.3	6.2	1.1	0.0	48.0
C Springs CO	1940	404	2.6	42.1	21.3	8.9	1.5	73.8

When, why and how does Dental Fluorosis occur?

Dental fluorosis occurs during early childhood while deciduous and permanent teeth and tooth enamel are still being mineralized and before they erupt within the mouth. (6,7) It is believed that dental fluorosis occurs because of the toxicity of fluoride to the enamel-forming cells of the teeth. (6) The degree to which a child experiences dental fluorosis depends on the amount of fluoride (s)he ingests. (2, 3, 6, 8) Dental authorities estimate that a child should ingest daily 0.03 mg to 0.07 mgs of fluoride per kg of body weight. When this amount is exceeded, dental fluorosis results. Moreover, the greater the fluoride overdose, the more severe is the dental fluorosis. Even with supervision, it is possible for a small child to overdose on fluoride each day with only one brushing with a fluoride tooth paste by swallowing much of it during the brushing process. (7)

The current model of fluorosis development proposes that *"....fluoride affects the forming enamel by making it porous. The degree and extent of the porosity depend on the concentration of fluoride in tissue fluids when the teeth are developing..."* and *"....the porosity and discoloration can vary in degree among different areas of the same tooth....."* (2) The ultimate result is the increasing porosity of the teeth and, in extreme cases, loss of the affected teeth. (9) Dental fluorosis is an excellent biomarker of excess fluoride ingestion and fluoride intoxication. (10) It is a visible, sometimes easily seen and noticed marker of fluoride intoxication. Unfortunately it tells us of excessive fluoride intake after-the-fact, i. e. after the newly emergent teeth have already been altered.

Why is the Prevalence of Dental Fluorosis Increasing?

There is now widespread recognition of the fact that the prevalence of dental fluorosis has increased substantially throughout those countries where fluoridation is practiced. (11-13) However, in spite of some reports to the contrary, (2) there does not appear to be general agreement within the dental community as to whether the severity of dental fluorosis has increased.

The nationwide increase of dental fluorosis was first recognized, documented and published by the National Institute of Dental Research (NIDR) after conducting (1986-1987) a survey that involved 32,241 U.S. school children. The total prevalence of dental fluorosis in this group of children was estimated to be 22.3 percent and included (mostly) very mild to mild dental fluorosis. (2) However some moderate to severe dental fluorosis was also found in approximately 1% to 2% of the children in "optimally" fluoridated water districts. (4) Another NIDR report published in 1988, studied four areas in Illinois with water concentration of one, two, three and four times the recommended "optimal" fluoride level. As of 1985, in the "optimally" fluoridated areas, twenty nine per cent of all tooth surfaces examined were reported to be affected by dental fluorosis. In those areas that had 2 to 4 times the optimal dose of

fluoride in the water supply, dental fluorosis affected close to seventy per cent of the teeth involved. (2) An even more recent study, published in 1990 (Table II) listed dental fluorosis in additional comparable cities in the United States and New Zealand with water systems "optimally" fluoridated and those with low fluoride. (14)

Table II - Percentages of Children with Dental Fluorosis

"Optimal" Fluoride Communities	Age Range of Children	F Conc. (ppm)	Percent of Fluorosis	Ref. (Note b)
Auckland (NZ)	7-12	1.0	25	(2)
Auckland Region	9	1.0	25	(3)
Hastings (NZ)	10	1.0	23	(5)
Kewanee, IL	13-15	1.0	28	(12)
Kerrville, TX	7-18	1.4	16	(14)
Angleton, TX	7-18	1.3	33	(14)
Alvin, TX	7-18	1.3	29	(13)
Kingsville, TX	7-18	1.0	39	(13)
Richmond, MI	6-12	1.2	51	(13)
Redford, IL	6-12	1.0	48	(13)
Hudson, MI	6-12	0.8	32	(13)
New York State	12-17	1.0	27	(15)
Low Fluoride Communities:	Age Range of Children	F Conc. (ppm)	Percent of Fluorosis	Ref. (Note b)
Richmond (NZ)	12-14	0.2	6	(1)
Auckland (NZ)	7-12	0.2	4	(2)
Auckland Region	9	0-0.2	15 (Note a)	(3)
Napier (NZ)	10	0-0.2	3	(15)
Iowa towns	8-16	0.0	3	(10)
San Antonio, TX	7-18	0.4	2	(14)
San Marcos, TX	7-18	0.3	8	(14)
N. Braunsfels, TX	7-18	0.3	9	(14)
Cadillac, MI	6-12	0.0	12	(13)
New York State	12-17	0-0.3	4	(15)

Note a) 55% of the children received fluoride supplements.

Note b) References in the last column of the above table are taken from Ref. 14

In carefully comparing the data in Table 1 and Table 2, a number of observations can be made:

- (1) the incidence of dental fluorosis in the children of Kewanee IL (selected because it is included in both studies and uses "optimally" fluoridated water) has increased from 1939 to 1990;
- (2) the prevalence of dental fluorosis is greater in "optimally" fluoridated communities than in communities with fluoride-deficient water and;
- (3) the percentage of dental fluorosis found today in "optimally fluoridated" communities approaches those found in communities with water containing 2, 3 and 4 times the "optimal level" of fluoridation 50 years ago.

The Anticipated Occurrence of Dental Fluorosis and Needed Corrective Measures

Now, as well as in the past few years, parents are being cautioned by the dental profession against excessive fluoride intake by infants and children by carefully regulating their total intake of fluoride in order to prevent dental fluorosis in developing teeth. This becomes increasingly more difficult as the infant/child grows older because of the ubiquity of fluoride in our country. Excessive amounts of fluoride can be ingested from a number of available sources: daily dietary fluoride supplemental pills, using fluoridated toothpaste, eating fluoride containing vegetables and fruits, other foods and drinks prepared with fluoridated waters and the application of topical fluoride products to teeth. (2, 7, 15) This is especially applicable if more fluoride is added to the communal water supply. Based on previously published data from other areas with drinking water fluoridated to about 1 ppm or 1 mg/liter ("optimal") our most optimistic scenario will show a minimum of one child out of every ten showing evidence of some degree of dental fluorosis. (2,5) However, if care is not exercised in preventing excessive fluoride intake, two to three children out of ten may develop dental fluorosis. The problem is exacerbated by the permissible fluoride levels in drinking water established by the U.S. Environmental Protection Agency's Safe Drinking water Act of 1974, in which the EPA set, on April 2, 1986, drinking water regulations for fluoride as follows:

1. ***"Primary Maximum Contaminant Level (MCL) of 4 mg F/L to protect against crippling skeletal fluorosis,"*** and
2. ***"Secondary Maximum Contaminant Level of 2 mg F/L to protect against moderate to severe dental fluorosis".***

This suggests that water-based consumer products should be made with water containing 2 ppm of fluoride or less.

The most effective corrective measure is to have children with developing teeth, especially permanent teeth, avoid the intake of toxic quantities of fluoride. This may be difficult to do for several reasons. Firstly, a child can unknowingly and unintentionally get unwanted fluoride from dental products, foods and drinks as mentioned above. Secondly, the expressed symptoms of dental fluorosis are not identical for all children exposed to the same dose of fluoride. Therefore there is difficulty in predicting fluorotic effects. Thirdly, there is also difficulty in diagnosing very mild to mild dental fluorosis by dental clinicians thereby missing opportunities to aesthetically correct objectionable fluorosis. (2)

The severity of the dental fluorosis and the psycho-socio-economic status of a child afflicted with dental fluorosis will determine if corrective action will be taken. Corrective procedures, when required or desired, include vital bleaching, abrasion and bonded veneers. These corrective procedures are performed by dental clinicians. However, at the present time dental fluorosis is considered to be a cosmetic effect (2) and therefore these corrective procedures are not covered by most dental insurance companies. Moderate-to-severe fluorosis results in unattractive misshapen teeth and probably will result in psychological damage to the affected child. However little research on the psychological effects of dental fluorosis on children has been conducted. (9)

Summary and Conclusions

Excessive fluoride intake by children causes a toxic dental condition known as dental fluorosis which is marked by visible mottling/discoloring of tooth enamel, pitting of the enamel and disturbed tooth shape. Dental fluorosis occurs during early childhood while the baby and permanent teeth and tooth enamel are still being mineralized and before they erupt in the mouth. The severity of the dental fluorosis is directly proportional to fluoride ingested in excess of 0.03 mg to 0.07 mg fluoride/kg of body weight/day. The ultimate result is the increased porosity of the teeth and, in extreme cases, loss of afflicted teeth. The prevalence of dental fluorosis is increasing in communities that have water supplies that are "optimally fluoridated" and in those with fluoride deficient drinking water because of the ubiquity of products containing fluoride. However the prevalence and severity of dental fluorosis is greater in "optimally fluoridated" communities than those with fluoride-deficient water. Parents are being advised to protect against excessive fluoride intake by infants and children by carefully regulating their total intake of fluoride. It is anticipated that fluoridation of the Natick water supply to 1 ppm or 1 mg/L will result in dental fluorosis to some degree in at least one child out of every ten. However if care is not exercised in preventing excessive fluoride intake, two to three children out of every ten may develop dental

fluorosis. Corrective procedures, when required, can be performed by dental clinicians. However, the cost of teeth rehabilitation will be borne, most likely, by the individual/parent since dental fluorosis is considered to be a cosmetic defect and therefore is not covered by most dental insurance plans.

References:

1. Wade, R, Personal Communication, Natick Board of Health, August. 1997
2. Ad hoc Committee on Fluoride of the Committee to Coordinate Environmental Health and Related Programs. *Review of Fluoride Benefits and Risks*. U.S. Dept. of Health and Human Services, Public Health Service, Feb. 1990, p. 20.
3. Tabers *Cyclopedic Medical Dictionary*. Thomas, CL, ed., 14th Edition, F. A. Davis Company, Philadelphia, 1981, p.547.
4. Hileman, B, *Fluoridation of Water*, Chem. & Eng. News, **66**, 26 (1988).
5. Coplan, M, *US Fluoridation Policy/Politics after Half a Century*, (Unpublished) 1997.
6. Diesendorf, M, *How science can illuminate ethical debates. A case on water fluoridation*. Fluoride, **28** (2):87-104, (1995).
7. Levy, SM, Kiritsy, MC, and Warren JJ, *Sources of fluoride intake in children*. J. Public Health Dent., **55**(1):39-52 (1995)
8. Xu, RQ, Wu, DQ, and Xu, RY, *Relations between environment and endemic fluorosis in Hohhot region, inner Mongolia*. Fluoride, **30**(1):26-28, (1997).
9. Meinig, GE, *The Root Canal Cover up*, Boon Publishing, Ojai, CA, 1994, 220 pp
10. Agency for Toxic Substances and Disease Registry (ATSDR), Clement International Corp. *Toxicological Profile for Fluorides, Hydrogen Fluoride and Fluorine (F)*. U.S. Dept.. Health Human Services, Public Health Service, TP-91/17, 1993, pp. 5-6, 94.
11. Szpunar, SM and Burt, BA, *Dental caries, fluorosis and fluoride exposure in Michigan schoolchildren*, J. Dent. Res., **67**:802-806, (1988)
12. Burt, BA, *The increase in dental fluorosis in the U. S.: should we be concerned?*, Ped. Dent., **16**(2):146-151 (1993)
13. Lewis, DW and Banting, DW, *Water fluoridation: current effectiveness and dental fluorosis*, Comm. Dent. & Oral Epidem., **22**:153-158, (1994)
14. Colquhoun, J, *Disfiguring, or White and Strong?*, Fluoride, **23**(3):104-111, (1990)
15. Leverett, DH, *Appropriate uses of systemic fluoride: Considerations for the '90s.*, J. Publ. Health Dent., **51**:42-47, (1991)

Skeletal Fluorosis, Osteosclerosis & Related Disorders

Two diseases of the skeletal system are osteosclerosis and osteoporosis. Osteosclerosis is a disease involving an increase in bone density (and thickening) accompanied by an increase in bone brittleness. Osteoporosis is a disease involving a decrease in bone density (due to loss of calcium) leading to decreased bone strength. (1) Osteofluorosis is an osteosclerosis caused by prolonged overdoses of fluoride.

Osteoporosis

Prolonged or increased ingestion of fluoride is known to result in increased bone mass. However, the bone formed in response to these high and/or prolonged doses of fluoride is reported to exhibit both reduced strength and increased fragility. (2, 3, 4) The abnormal bone resulting from fluoride is of poor quality and while the increased mass helps compressive strength, it generally leads to weakness of tensile strength. Thus, tests of fluoride "treatment" for osteoporosis finds a decrease in vertebral compression fractures but an increased incidence of hip and long bone fractures, compared to control patients. Other researchers have advised abandoning fluoride as a legitimate treatment for osteoporosis for that reason as well as for the well known toxicity of fluorides. In fact, in a 1987 review of fluoride therapy for osteoporosis, Louis V. Avioli, professor at the Washington University School of Medicine, concludes: ***"Sodium fluoride therapy is accompanied by so many medical complications and side effects that it is hardly worth exploring in depth as a therapeutic mode for postmenopausal osteoporosis, since it fails to decrease the propensity toward hip fractures and increases the incidence of stress fractures in the extremities."*** (5)

Skeletal (Osteo)Fluorosis

Osteofluorosis is a complicated disease with a number of stages. The first two stages are preclinical, that is, the patient feels no symptoms but changes have taken place in the body. In the first preclinical stage, biochemical changes occur in the blood and bone composition; in the second stage histological changes can be observed in bone biopsies. Some experts call these changes harmful because they are precursors of more serious conditions. Other experts say they are harmless. (6) Most admit that the effects of long term ingestion of fluoridated water on bone are poorly understood. (6)

The clinical stages of osteofluorosis includes pain in the bones and joints, muscle weakness, fatigue, calcification of ligaments and bone spurs. Most experts in skeletal fluorosis agree that ingestion of 20 mg of fluoride per day for 20 years or more can cause crippling skeletal fluorosis and doses as low as 2 to 5 mg per day over the same time period can cause the preclinical stages. (7) Moreover, the total quantity of

fluoride ingested is the single most important factor in determining the clinical course

Post Publication Correction: The correct figures for the development of crippling skeletal fluorosis should be 10-20 mg/day for 10-20 years. (See:

of osteofluorosis. (8) The severity of the symptoms correlates directly with the level and duration of exposure. For almost 40 years, investigators in the United States have searched for evidence of osteofluorosis. The U. S. Public Health Service (8) reports that:

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

Nevertheless, large numbers of people in Japan, China, India, the Middle East and Africa have been diagnosed with skeletal fluorosis. (9) In India, Tanzania and South Africa, crippling forms of skeletal fluorosis have been reported in pediatric age groups as well. (8)

Hip and other Fractures

In clinical practice, the occurrence of atraumatic minor compression fractures of vertebra is common in postmenopausal osteoporotic women and is frequently asymptomatic, being found only by radiographs, though the patient may have noted a slight decrease in height over time. The more morbid consequence of osteoporosis is hip fracture which has the potential for seriously disabling patients. It has been suggested that sodium fluoride could be used as a treatment for osteoporosis since it is associated with 'bone thickening'. Dr. C. Y. C. Pak and others are conducting a USPH funded and FDA approved study using slow-release sodium fluoride in the management of postmenopausal osteoporosis. (10) In this study, Dr. Pak is administering about 25 mg of fluoride per day in a slow release form to post menopausal women in order to raise their serum fluoride levels from 50 ng/ml to slightly over 100 ng/ml while avoiding fluoride's known gastric inflammatory effects such as mucosal erosions, ulcers, and bleeding which regularly accompany usual oral fluoride supplementation at this dosage. However, according to a critical review of this study appearing in the journal, "Fluoride", Dr. John Lee states that: "... (Dr. Pak's study) seems limited to demonstrating the obvious, i.e., that excessive fluoride causes osteofluorosis." (10)

The one interesting finding in Pak's interim report is the fact that fluoride supplementation did not cause any reduction in vertebral fractures in women on estrogen supplementation compared to controls. Among estrogen-treated women, the fracture-free rate of the placebo (no fluoride) group compared to that of the fluoride group was 75.0% and 76.9% respectively, an inconsequential difference

In a national study of ecological design (11), Jacobsen et al., examined the association between water fluoridation and the incidence of hip fractures. For the period 1984-1987, a total of 218,951 eligible hip fracture cases were studied. (12) Raheb characterized the results of Jacobsen's study as "...A small, statistically significant, positive association was found between fluoridation and fracture incidence rates." (13) However, a careful review of the data of Jacobsen and his co-workers show an eight (8%) percent increase in women [± 2 percent] and a seventeen (17%) percent increase for men [± 4 percent]. A more recent study on a smaller population (which was restricted to Mormon communities in Utah to correct for confounding factors such as smoking and/or use of alcohol) showed an increased incidence of hip fractures of 27% in women and 41% in men, albeit with a larger 95% confidence interval. (14) While four other studies indicate either no effect or a negative effect of fluoridation, these studies involved a total of only 6,874 subjects as opposed to positive correlation in the case of 781,575 subjects.

Summary

Well controlled studies have not demonstrated a beneficial effect of the use of high doses of fluoride in reducing osteoporosis and related bone fractures. However, there has been shown to be a positive relationship between water fluoridation and increased hip fractures in persons 65 years of age and older. Human crippling osteofluorosis is endemic in several countries of the world, but is extremely rare in the United States. A number of factors govern the amount of fluoride deposited in the skeleton. The important factors include:

- 1) age at exposure
- 2) duration of exposure
- 3) dose of fluoride
- 4) nutritional status
- 5) renal status
- 6) individual biological variation.

References

1. *The Family Doctor*, 3rd Edition, Creative Multimedia Corp., (1994)
2. Riggs, BL, et al., *Effect of fluoride treatment on the fracture rate in post-menopausal women with osteoporosis.*, N. Engl. J. Med., 322:802-809 (1990)
3. Kleerekoper, M and Balena R, *Fluorides and osteoporosis.*, Ann. Rev. Nutr. 11:309-324, (1991)
4. Riggs, BL, et al., *Drug therapy: the prevention and treatment of osteoporosis.*, N. Engl. J. Med., 327:620-627, (1992)
5. Alvioli, L, as quoted in Hileman (Ref. 6, below) p. 32.
6. Hileman, B, *Fluoridation of Water.*, Chem. & Eng. News, 66:26, (1988)
7. Ibid., p 35.
8. Ad hoc Committee on Fluoride of the Committee to Coordinate Environmental Health and Related Programs. *Review of Fluoride Benefits and Risks.*, U.S. Dept. of Health and Human Services, Public Health Service, Feb. 1991, p. 20.
9. Null, G, *The Fluoridation Fiasco.*, Internet publication, (www.GaryNull.com), p. 7
10. Lee, JR, *Critical Review of "Slow-release sodium fluoride in the management of postmenopausal osteoporosis" by CYC Pak et al.*, Fluoride, 27(4):27-228, (1994)
11. Ecological studies are those in which the units of analysis are populations or groups of people, rather than individuals.
12. Jacobsen, SJ, et al., *The association between water fluoridation and hip fracture among white women and men aged 65 years and older: a national ecologic study.*, Ann. Epidemiol. 2:617-626, (1992)
13. Raheb, J., *Water fluoridation, bone density and oral epidemiology.*, Comm. Dent. & Oral Epidem., 23:209-216, (1995)
14. Danielson, C, et al., *Hip fractures in Utah's elderly population.*, J. A. M. A., 268:746-8, (1992)

Carcinogenicity

Whether or not fluoride causes cancer in humans has been a subject of heated debate. Based on the journal articles and other documents reviewed by this committee, the debate has not been resolved and appropriate epidemiology studies have yet to be conducted. This section of the committee report summarizes the two major animal bioassays that have tested fluoride for its carcinogenic potential. It also reviews some of the human epidemiology studies that have been published.

Animal Studies

The US National Toxicology Program (NTP) tested sodium fluoride for carcinogenicity in a 2 year bioassay using rats (F344/N) and mice (B6C3F1) (1) The most often cited result of this study is that it provided "equivocal" evidence of carcinogenicity based on a statistically significant elevation of osteosarcoma (a type of bone cancer) in male rats. No bone cancer was observed in female rats and male or female mice. However, a careful review of the data presented in the published report of this study (1) shows that one additional osteosarcoma was observed, but not counted in male rats. In addition, one male and one female mouse treated with fluoride also developed osteosarcoma, although these were not included in the summary table, only in the footnotes.

In addition to bone cancer, the NTP study showed marginal differences between control animals and dosed animals with respect to cancer of the oral mucosa, thyroid gland and uterus of rats and the hematopoietic system and liver in mice. While several liver tumor types were observed in male and female mice, two tumor types were considered highly unusual and worthy of note: hepatoblastoma and hepatocholan-giocarcinoma (1).

In general, animal bioassays test chemicals at doses much higher than received by humans, and the results are then extrapolated to lower doses. In the NTP study, sodium fluoride was administered in drinking water at 25, 100 and 175 ppm. A special low fluoride diet was formulated. A measure of cumulative dose (dose taken in over a long period) for fluoride is the ash content in the bone. Comparison of the fluoride in the bones of the animals in the high (125 ppm) dose group with humans drinking water with fluoride concentrations above 2 ppm show that older humans have more fluoride in their bones than the high dose animals (1,2). In other words, this study was conducted using fluoride doses that humans actually receive.

The NTP study has been criticized by all sides: some charge the study understates the cancer potency, others say it overstates the potency and is irrelevant to humans (2,3,4).

William Marcus, an EPA toxicologist, compared the rate of osteosarcoma in historical controls (control animals used in previous toxicology testing studies) with those

obtained in the dosed animals in the fluoride study. He assumed the fluoride dose in the historical controls was due to ingestion of normal rat chow, and represented a dose between the 25 ppm and 100 ppm dose group in the fluoride drinking water study (the NTP agrees with this calculation). Marcus found that the observed "historical" control rate of osteosarcoma fit exactly where expected based on the fluoride dose (2). Marcus also reports that the original pathologist's classifications of the liver tumors in the rat, oral tumors in the rat, and adrenal pheochromocytomas were consistently downgraded by a review panel (2). This would serve to underestimate the evidence for cancer based on the animal study. Marcus recommended that EPA assemble an independent panel of pathologists to review the slides from the NTP study.

James Bawden, representing the American Associations for Dental Research (AADR) at a peer review panel of the NTP study, claimed that the types of bone tumors observed in the rat differ from osteosarcoma observed in humans, and thus the NTP study has no relevance to humans (4). However, Bawden's statement represents a fundamental misunderstanding of the purpose of the 2 year bioassay: it is not a model for a specific cancer at any particular site in the body. The purpose of the bioassay is only to show the potential for a chemical to induce cancer; correlation of cancer sites in the animal and humans is not required and indeed, is rarely observed. John Stamm, representing the American Dental Association at the peer review panel (5), expressed concern about how the NTP did its statistical analysis and suggested the data were not strong enough to call fluoride an "equivocal" carcinogen.

Proctor and Gamble (P&G) sponsored a 2 year bioassay in which rats (Sprague - Dawley) were administered sodium fluoride in food. Rats were fed 1.8, 4.5, or 11.3 mg fluoride per kg body weight each day in a low fluoride semi-synthetic diet. There were two control groups, one fed the low fluoride semi-synthetic diet and one fed normal rat chow (fluoride content not determined). The fluoride content of the drinking water was not reported. The study ended early because too many animals died in both the fluoride fed and control groups. There were multiple problems with this study, including that the semi-synthetic diet may not have provided the nutrients for normal growth and development and a virus is likely to have infected the animals (6).

The original laboratory conducting the P&G rat study reported one osteosarcoma in a low dose female and a few other tumors. The carcinogenicity assessment committee of the FDA reassessed the data and found another osteosarcoma in a low dose female and one in a high dose male. Moreover, not all the animals were carefully examined for bone cancer and thus other tumors may have been missed. The FDA review concluded that **"...there are flaws and uncertainties in the studies that keep them from providing strongly reassuring data"** (6).

The P&G study was also conducted with mice. Osteomas (non-malignant bone tumors) were observed in all groups with a significantly higher incidence in the high fluoride dose group. However, the mouse study hasn't been deemed useful for risk assessment because the mice in both the treatment and control groups were infected

with a virus (C-type retrovirus), and it is suggested that the tumors were formed via an interaction between the virus and fluoride (7).

Human epidemiology studies

Many epidemiology studies examining possible associations of fluoride and cancer have been conducted. Some studies examine bone cancer or cancers at particular sites, others examine overall cancer incidence rates or cancer mortality rates. Few of the studies are of individuals; rather they look at effects of populations who are assumed to be exposed or not exposed to fluoride or fluoridated water. Only a few will be summarized here.

Early epidemiology studies compared cancer mortality (death) rates in cities with and without water fluoridation. An analysis by Yiamouyiannis and Burk in 1977 found 4-5% lower death rates in non-fluoridated cities (comparison of 10 largest US cities with fluoridated water and 10 largest US cities without fluoridated water). At about the same time, three British scientists completed an analysis of the same 20 cities and found no effect of fluoridation on mortality rates. A review of these two studies by the US National Research Council concluded that the differences could be explained by use of different data sets and analytical methodologies; the differences showed the relative insensitivity of the data and measurements (3). Yiamouyiannis disputes this and claims that the British scientists omitted data and made mathematical errors (12). Freni and Gaylor examined international trends in bone cancer based on incidence (not mortality) data in a study published in 1992 (8). In general, they found no relationship between water fluoridation and bone cancer with the possible exception of an increased risk for females in fluoridated areas of the United States. The study was weakened by lack of good exposure data; non-differential misclassification of exposure will lead to an underestimate of an effect. Freni and Gaylor (8) also demonstrated that mortality data is a far less reliable measure of bone cancer than incidence data.

Several small case control studies examining the relationship between fluoride and bone cancer have been conducted, with mixed results. One small study in New Jersey found that males under age 20 years who resided in communities with fluoridated water at the time of diagnosis had a higher osteosarcoma rate than those who resided in nonfluoridated communities (9). A small case control study of osteosarcoma and water fluoridation (among other factors) conducted in Wisconsin showed no association between osteosarcoma and residence in an area with fluoridated water at time of diagnosis (10). Both of these studies suffer from lack of explicit exposure data. Exposure classification is based on residence at time of diagnosis, which may or may not reflect exposure to fluoridated water for any period of time.

A larger case control study examining the association between fluoride intake (and water fluoridation) and childhood (less than 25 years old) osteosarcoma was conducted in New York State (11). This study included contacting both cases (or their parents) and controls, and asking questions related to fluoride exposure. The study found no association between total fluoride exposure and osteosarcoma for either males or females. A statistically significant risk (odds ratio) for osteosarcoma was found at the lowest level of water fluoridation for females, and for males and females combined (but not for males alone). However, the risk did not increase with increasing exposure to fluoride in the drinking water, and the risk at the higher water fluoridation exposure was not significantly elevated (11).

Yiamouyiannis examined the relationship between incidence of and mortality from bone cancer in males and water fluoridation using several US data sets (12). He reported an association between water fluoridation and bone cancer incidence and death from bone cancer among males under the age of 20. He also suggested there is a 30-60% increase in oral cancers because of fluoridation. Several problems are apparent with this paper. For example, for bone cancers, he assumed that only males would have bone cancer linked to fluoride, and then used females as an unaffected reference population. The validity of this assumption has not been proven; indeed, other data reviewed here suggest this is an incorrect assumption.

A recently published study from Okinawa, Japan reports a relationship between fluoride concentration in drinking water and mortality from uterine cancer (13). However, it does not seem that important variables, such as water chlorination, were appropriately taken into account. It is also noted that, in Okinawa, the fluoridated water ranged between 0.19 and 0.37 mg/l fluoride. These waters would generally be considered non-fluoridated in the U.S.

Summary of cancer data

The animal study conducted by the National Toxicology Program (NTP) provides evidence that fluoride causes osteosarcoma, a malignant bone tumor. Although the NTP concluded that its study gave "equivocal" results with respect to cancer, the background memos and documents suggest that the results are actually stronger than suggested by the report. Similarly, the Procter and Gamble study likely gave stronger evidence of carcinogenicity, notably bone cancer, than suggested in the summary statements.

That fluoride is associated with bone cancer is reasonable from the point of view of what is known about the effects of fluoride: fluoride causes the division of immature bone cells (proliferation of osteoblasts) and fluoride accumulates in the bone and thus can cause damage there. Fluoride has been shown to be genotoxic in numerous test systems which is another property that is associated with carcinogens (1,5). In other

words, the biochemistry, pharmacokinetics, and other toxicology studies support the view that fluoride maybe a bone carcinogen.

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

References

1. 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).
2. Wm. Marcus. US EPA. *Memo to Alan Hais, Acting Director, Criteria and Standards Division, Office of Drinking Water, US EPA. on Fluoride Conference to Review the NTP Draft Fluoride Report.* May 1, 1990. [Reference provided by Shirley Brown].
3. Hileman, B. *Fluoridation of Water*, Special Report. Chemical and Engineering News, August 1, 1988.
4. Bawden, JW, *NTP Public Hearing on the NTP Technical Report on the toxicology and carcinogenesis studies of sodium fluoride.* April 26, 1990. [Reference provided by the Board of Health].
5. Stamm, JW, on behalf of the ADA. *NTP Public Hearing on the NTP Technical Report on the toxicology and carcinogenesis studies of sodium fluoride.*, April 26, 1990. [Reference provided by the Board of Health].
6. Agency for Toxic Substances and Disease Registry (ATSDR). *Toxicological Profile for Fluorides, Hydrogen Fluoride and Fluorine.*, April, 1993.
7. D'Amato, RA, Procter & Gamble prepared remarks for NTP peer review panel on sodium fluoride. April 26, 1990. [Reference provided by the Board of Health].
8. Freni, S, and D. Gaylor, *International Trends in the Incidence of Bone Cancer Are not Related to Drinking Water Fluoridation.*, Cancer **70**(3):611-618, (1992)
9. Cohn, PD, *A brief report on the association of drinking water fluoridation and the Incidence of Osteosarcoma among young males.*, Trenton, NJ, NJ Department of Health, Environmental Health Service. 1992 (cited in ref. 11).
10. Moss, M. M. Kanarek et al., *Osteosarcoma, Seasonality, and Environmental Factors in Wisconsin, 1979-1989.*, Archives of Environ. Health **50**(3):235-241, (1995)
11. Gelberg, KH, EF Fitzgerald, S Hwang, and R Dubrow, *Fluoride Exposure and Childhood Osteosarcoma: A Case-Control Study.*, American J. Public Health **85**:1678-1683, (1995)
12. Yiamouyiannis, JA, *Fluoridation and Cancer.*, Fluoride, **26**(2):83-96, (1993)
13. Tohyama, E, *Relationship between fluoride concentration in drinking water and mortality rate from uterine cancer in Okinawa Prefecture.*, Japan. J. Epidemiol., **6**(4):84-191, (1996).

Metabolic & Enzymatic Effects

This section discusses the impact of fluoride on metabolic and enzymatic processes. Included in these areas are the direct action of the fluoride anion as well as that of other inorganic fluorine-containing materials related to the process of fluoridation. In addition, the impact of fluoride on biochemical pathways and/or enzymatically controlled processes based upon either the formation of fluoride-metal complexes or upon the interference caused by fluoride in the interruption of enzyme-substrate spatial arrangements is discussed.

Background Material

Although it is beyond the scope of this report to present a pedagogical background in biochemistry or the chemistry of fluorine-containing compounds, a few principles should be discussed in order to understand the issues involved and the degree to which these issues would have had an impact on the widespread introduction of fluoride into the human food chain.

Fluorine is contained in significantly fewer than 10 % of more than 700 minerals. Of these, only 5 or 6 minerals are truly common and almost all of these are either insoluble or have very limited solubility in water of neutral pH, although some exhibit enhanced solubility in water in the lower pH (acidic) range. (1)

In those areas of the world where there is an abundance of the common fluorine-containing minerals in contact with either ground or surface water below pH 7, dissolved fluorine-containing minerals will be present in the indigenous water supplies. As a result, those areas will have an increased presence of fluorine in the vegetable and animal food-stuffs produced there. The fluorine that does enter the human food-chain, whether naturally occurring or as a result of artificial fluoridation, corresponds primarily to the sodium salt of the fluoride anion (F^-) and either sodium fluorosilicate or fluorosilicic acid. Clearly it is the nature of these materials which most concern us in this section and, in addition, the nature of the biological materials with which these interact. (2)

Characteristics of Fluorine and Fluoride Ion

The primary action of fluoride in metabolic and enzymatic reactions is related to the formation of "*complexes*" in one form or another. The fluoride anion has the highest charge density of any negative ion. (3) As a result of this, it is now known that fluoride forms an exceptionally strong hydrogen bond (> 148 kJ/mol.) with substrates in amide-fluoride systems. (4) Strong hydrogen bonding is now recognized as being clearly distinguishable from normal hydrogen bonding.

Another related characteristic of fluoride ion is that it exhibits an affinity for many metal ions, especially magnesium, manganese, aluminum, and calcium and therefore it can effect the bioavailability of these ions either separately or may cause either inhibition or otherwise interact with any enzyme system which requires one of these metals as a co-factor. (5,6)

Structural Susceptibility of Biomolecules

The impact of strong hydrogen bonding is that proteins, which consist of a repetitive sequence of amide linkages, are particularly susceptible to this type of hydrogen bonding. The end results of this type of interaction are two-fold. The lesser effect is that the carbonyl-nitrogen (amide) bond in proteins may become more susceptible to cleavage even though fluoride itself is a less nucleophilic anion. The second, and probably enormously greater, effect is that the spatial arrangement or macromolecular structure of these materials depends heavily upon normal hydrogen bonding to produce the secondary stereochemical structure required for appropriate enzymatic activity to take effect. (7) This has been demonstrated by Edwards and co-workers, who studied the perturbations caused by fluoride on the structure of Cytochrome C peroxidase. (8) Further, *ab initio* calculations by Emsley et al. lead to the conclusion that the fluoride ion may completely disrupt the Thymine-Adenine linkage in DNA. (9) A survey of the literature reveals no shortage of supporting research results. (10) The conclusions reached in several of these studies are listed below.

- Fluoride inhibits metalloproteins (12)
- Fluoride inhibits DNA polymerase (13)
- Fluoride induces chromosome aberrations (14)
- Fluoride effects the adenyl cyclase system (15)
- Fluoride inhibits yeast enolase (16)
- Fluoride inhibits protein synthesis enzymes (17)
- Fluoride inhibits glycolytic enzymes (18)
- Fluoride inhibits cell growth enzymes (19)
- Fluoride inhibits testosterone synthesis (11)

It is of interest to note that the latter interaction may be responsible for those deleterious effects of fluoride which appear to be restricted to males (e. g. testosterone is involved in bone growth in males but not in females). (11) The above list is by no means exhaustive. Rather, it should be taken to indicate that there is sufficient evidence to warrant more extensive research into this area. However, over all, the results described in the above references "*suggest that sodium fluoride is potentially dangerous to humans.*" (14)

Fluoride and Calcium Metabolism

The interaction of fluoride in those metabolic processes involving calcium are also of great significance. This type of interaction may have been responsible for the recent observation that even when calcium is supplemented in osteoporotic patients, a large number of those who have also been treated with fluoride still show evidence of calcium deficiency. (20) The lack of availability of calcium, either as a result of precipitation by fluoride or the formation of fluoroapatite, may result in hypocalcemia which may have other widespread and, as yet, poorly understood effects on bone formation and other regulatory mechanisms of the body.

Summary and Conclusion

The information above and the references cited illustrate that fluoride can seriously disturb the balance of enzymatically activated biochemical reactions. These effects clearly were not well-known at the commencement of fluoridation activities. However, the recent literature contains many references (e.g. 21-25) to original research results that illustrate that fluoride effects the metabolism of a number of common oral bacteria, (e.g., *Streptococcus mutans*) so that, equally clearly, this phenomenon of fluoride effects on enzymes should be as well known to the proponents of fluoridation as to anyone. Thus, while there can be no doubt that fluoridation has contributed to the reduction of dental caries in the past, there is likewise little doubt that the continuation of the fluoridation process in the light of recent evidence outlined above is inappropriate without first answering the serious and potentially health-affecting questions raised.

References

1. *Properties of Minerals*, in *Lange's Handbook of Chemistry*, Chemical Rubber Publishing Company (1960)
2. In fact, more than a few attempts have been made to suggest or prove that fluoride is a necessary nutrient in the human diet. It is now almost universally recognized that if there is an MDR for fluoride, it is so small as to border on the insignificant. The fact that fluoridation has an effect on the incidence of dental caries does not make it an essential nutrient any more than penicillin would be so-considered in view of its effects on other microbes.
3. Roberts, JD and Caserio, MW, *Basic Principles of Organic Chemistry*, A. Benjamin, Inc., (1964)
4. Emsley, J, et al., *An Unexpectedly Strong Hydrogen Bond*, J.A.C.S., **103**: 24 (1981)
5. Villee, C, *Birth Defects & Glycolysis*, N. E. J. Med., **310**: 254 (1981)
6. Chlubek, D, *Some aspects of prenatal fluoride metabolism in human studies performed during the perinatal period*, Ann. Acad. Med. Stein., 31: 1-99, (1996)
7. Karlson, P, *Introduction to Modern Biochemistry*, Academic Press, 1963
8. Edwards, SL, et al., *The crystal structure of fluoride-inhibited cytochrome c peroxidase*, J. Biol. Chem., **259**:12984 (1984)
9. Emsley, J, et al., *The Uracil-Fluoride interaction*, J.A.C.S., **104**:476 (1982)
10. The literature search for this section alone produced over four hundred references from one internet source. The vast majority of these are from well-respected, peer-reviewed, scientific journals.

11. Kanwar, KC, et al., *In vitro inhibition of testosterone synthesis in the presence of fluoride ions.*, I. R. C. S. Med. Sci., 11:813-814, (1983).
12. Wiseman, A, *Pharmacology of Fluorides*, in F. A. Smith, *Handbook of Exper. Pharm.*, Springer, (1970)
13. Lehman, IR, *DNA Synthesis*, in *Methods in Enzym.*, VI, Academic Press (1963).
14. Tsutsui, T, et al., *Cytotoxicity, Chromosome aberrations...Fluoride.*, Mutation Res., 139:193 (1984)
15. Sternweis, PC and Gilman, AG, *Aluminum: A Requirement...by Fluoride.*, Proc. Natl. Acad. Sci. (USA), 79:4888 (1982)
16. *Fluoride Inhibition of Yeast Enolase.*, Biochemistry, 20:6894 (1981)
17. Song, XD, et al., *The Effect of Sodium Fluoride.....Study.*, Fluoride, 21:149 (1988)
18. Bartholmes, P, et al., *Inhibition of Glycolytic Enzymes by Fluoride Ions.*, Dtsch. Zahnarztl. Z., 42:916 (1987)
19. Holland, RI, *Fluoride Inhibition of Protein Synthesis.*, Cell. Biol. Int. Rep. 3:701 (1979)
20. Dure-Smith, BA, et al., *Calcium deficiency in fluoride-treated osteoporotic patients despite calcium supplementation.*, J. Clin. Endocrinol. Metab., 81:269-275, (1996)
21. Bunick, FJ, and Kashket, S, *Enolases from Fluoride Sensitive...Streptococci.*, Infect. Immun. 34:856 (1981)
22. Huther, FJ, et al., *Isolation, characterization....FA-I.*, Infect. Immun., 58:1043 (1990)
23. Marquis, RE, *Diminished acid tolerance....fluoride.*, J. Dent. Res., 69:672 (1990)
24. Curran, TM, et al., *Quasi-irreversible inhibition....fluoride.*, FEMS Microbiol. Lett., 119:283 (1994)
25. Maltz, M and Emilson, CG, *Susceptibility of oral bacteria to various fluoride salts.*, J. Dent. Res. 61:786 (1982)

Hypersensitivity and "Allergic" Reactions

Some humans appear to be hypersensitive to fluoride, although there is some question about whether the reaction is allergic. At the very least, some components of the hypersensitivity appear to be allergic (i. e. an immune system effect); other portions may be related to the central nervous system or altered fluoride metabolism (pharmacokinetics). However, from the viewpoint of the sufferer of these effects, it is a moot question since the end result is virtually the same. With apologies to the sufferers and those physicians who use the terms "*allerg-(ies,ic)*" in their discussions or papers, this section refers to the manifestations of these effects as hypersensitivity.

Hypersensitivity reactions to fluoride, including fluoridated water, have been known to and reported by medical practitioners for decades (1). A search of the recent literature identified several references to occupational asthma induced by fluoride exposure in the aluminum industry (aluminum potroom asthma)(2), but no references to environmental exposures. While this absence of recent literature suggests that this is not an active area of current research, it does not invalidate the older observations.

George Waldbott, M. D., summarized both the medical literature and his own observations on the allergic reactions to fluoride in a 1964 article in the Journal of Asthma Research. He reported six cases of urticaria (hives) due to fluoridated water. The urticaria was accompanied by other fluoride associated health effects, including paresthesias, cephalgia (headaches), arthritis in the lower spine, gastrointestinal and urinary disturbances. For at least some of these patients, the association of the urticaria with fluoride was demonstrated in double blind challenge tests. These patients appeared to retain more fluoride than most individuals, putting them at higher risk of fluoride-associated health effects. In the same journal article, Waldbott also described other effects on fluoride on the skin of sensitive individuals. These included atopic dermatitis and contact dermatitis, including on the fingers of dentists after applying sodium fluoride to patients.

Summary

This paper demonstrates that there is a sub-population of adults that is hypersensitive to even low doses of fluoride such as those in water fluoridated to 1 ppm. While the size of this sub-population is unknown, there appears to be at least one Natick resident who is hypersensitive to fluoridated water.

References

1. Waldbott. G.L., *Allergic Reactions to Fluoride.*, J. Asthma Res. 2(1):51-64, 1964.
2. MEDLINE search using key words "fluoride" and "allergy" on August 22, 1997, search included "related articles" feature of PubMed

Central Nervous System: behavioral and IQ effects

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

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

Animal Toxicology Studies

Dr. Phyllis Mullenix and co-workers published a study on the neurotoxicity of sodium fluoride in rats in 1995. (1) The study used behavioral methodology that focused on behavioral repertoire, responses to novelty, and the temporal or sequential organization of spontaneous behavior. This methodology had been previously used to study alterations in CNS function and behavioral alterations including cognitive deficits (mental retardation) due to chemotherapy for childhood acute lymphoblastic leukemia (2), amphetamine induced hyperactivity, and triethyltin-induced hypoactivity. (1) Thus, the methodology used to test the sodium fluoride should be considered a validated one.

In all, 532 rats (Sprague-Dawley, male and female) were used in the study. Fluoride was given at different doses and three life stages: prenatal, weanling and adult. Prenatal doses were administered via subcutaneous injections of the pregnant dam on either gestational days (GD) 14-18 or GD 17-19. The dose schedules produced plasma fluoride peaks of 0.15 to 0.2 ppm. Weanling and adult rats were dosed via incorporation of 75 ppm, 100 ppm or 125 ppm sodium fluoride in drinking water for 6 - 20 weeks. While these drinking water concentrations are higher than would be used in water fluoridation, the cumulative exposures to fluoride, as measured by the concentrations of fluoride in plasma, are similar to plasma levels observed in humans with high end exposures. For example, the plasma level of fluoride in the rats ranged from 0.059 - 0.640 ppm. Plasma fluoride levels of 0.076-0.25 are found in humans ingesting 5-10 ppm fluoride in drinking water and plasma fluoride levels of 1.44 ppm

have been found in children 1 hour after receiving topical applications of acidulated phosphate fluoride gel.

The prenatal exposures (also referred to as *in utero* exposures) during GD 17-19 altered the behavioral outcome in male (but not female) offspring in a manner correlated with hyperactivity. There was no overt toxicity to the dams or offspring based on reduced body weight, suggesting the behavioral alterations were not secondary to another toxicity. [Note that absence of overt toxicity in the dam is a key test in defining a developmental toxicant according to EPA guidelines.] (3) Plasma fluoride concentrations at 3 and 9 weeks of age were not elevated, suggesting that the damage occurred *in utero* and as a result of a short term exposure. It should be noted that the nervous system develops throughout gestation and during the early postnatal period, and that the higher cognitive functions develop towards the end of gestation (gestation period is 20-21 days in rats) and during the postnatal period. It is also a common feature of developmental toxicants that adverse effects are observed if a toxicant is given during one period of gestation, but not during others (other adverse effects may or may not be observed at other periods).

Weanling exposures to fluoride affected the behavior of both males and females in a dose dependent manner (based on plasma fluoride levels), although the female rats were affected at lower doses. These doses also induced slight toxicity as judged by body weight gains. The behavioral changes for both sexes and at all doses were consistent with respect to the controls, and were different from the behavioral changes observed in male rats exposed prenatally. The observed behavioral changes are associated with cognitive deficits in other studies (1).

Adult rats were exposed for 6 weeks to 100 ppm fluoride in addition to the no fluoride control. No toxicity was associated with this dose based on differences in body weight. Female (but not male) rats showed behavioral changes, and these changes were similar to those observed in the weanling exposures, namely cognitive defects (1).

A study by Liu published in 1989 (4) appears to show behavioral changes associated with *in utero* exposure to fluoride, although only the abstract is available in English. Female Wistar rats were given 0, 30 or 60 ppm NaF in drinking water, apparently for 85 days before and during pregnancy. Their offspring were tested (age 33-42 days) for pain reaction and conditioned reflex. The response time for the fluoride exposed offspring was longer than for the no-fluoride control animals.

Behavioral Changes in Humans Associated with Exposure to Fluoride as Adults

Spittle (5) summarized several studies that reported central nervous system effects in humans following occupational or environmental exposures to fluoride. About 25% of workers exposed to fluoride from cryolite (a fluoride-containing mineral) who had

skeletal fluorosis also had central nervous system effects including fatigue, headache and giddiness. A similar proportion of aluminum smelter workers with skeletal fluorosis also reported psychiatric disturbances including depression, mental sluggishness and memory disturbances. Although these observations are reported for people with high fluoride exposure, the effects from occupational exposures are often used to forewarn of hazards that may also occur, but be harder to measure, at lower doses such as those that may result from environmental exposures.

There are also several studies where behavioral changes or other CNS symptoms are associated with lower fluoride exposure. Waldbott (summarized in Ref. 5) reported generalized progressive fatigue associated with a distinct decline in mental acuity in persons residing within 3 miles of an enamel factory emitting hydrogen fluoride. Waldbott also reported CNS symptoms (lethargy, memory impairment) in several patients exposed to fluoridated drinking water. Some of these studies are also described in this report in the section titled hypersensitivity. (5,6)

Rotton and coworkers (7) subjected adult volunteers to experiments that tested their attention and error rate on primary and secondary tasks (tracking objects and responding to flashing lights). The individuals were given one drop of sodium fluoride in water (0.1, 1, 10, 100 ppm) sub-lingually. The administration of sodium fluoride did not affect the primary task; tracking a moving target. However, the sodium fluoride increased the error rate (missed responses) of the secondary task and the highest two doses resulted in an increase in the latency (response time) between the secondary stimulus and the subject's response.

Decreases in IQ in humans exposed to fluoride *in utero*

Cognitive deficits due to fluoride exposure, in the form of a population-wide decrease in intelligence in children, have been reported in several different populations in China in the last few years. Two of the studies were published in English (8,9), and they are summarized here.

Zhao and co-workers (8) studied the differences in IQ in children aged 7-14 in two villages in Shanxi Province of China. The villages were similar with respect to occupations, living standards and social customs, but differ with respect to the fluoride content of drinking water. Sima has a fluoride content of 4.12 ppm, 86% of the population has clearly evident dental fluorosis, and 9% have clinically diagnosed skeletal fluorosis. Xinghua has a fluoride content of 0.91 ppm, 14% of the population has dental fluorosis and no skeletal fluorosis has been diagnosed. In each village, 160 (80 male, 80 female) randomly selected children were given standard IQ tests. The only constraint was that the children's mothers lived in the village being studied during their pregnancy. The mean IQ in the Sima (high F) and Xinghua (low F) were 97.7 and 105.2, respectively, which is a statistically significant difference. The range was also generally lower in the higher fluoride area.

Table I - IQ Ranges for Different Exposures to Fluoride (8)

Village	Mean IQ	Range
Sima (high F)	97.7	60-133
Xinghua	105.2	69-141

The IQ distribution of children in Sima was lower than in the low F village of Xinghua, leading to fewer children in the superior intelligence category and more children in the low intelligence category in the high fluoride village. There was no difference in IQ between males and females. As expected, within each village, IQ correlated with parents' educational levels.

The high F village of Sima had fluoride concentrations only just above that allowed by US standards (MCL of 4.0 ppm) and the low F village of Xinghua had a fluoride content (0.91 ppm) slightly lower than the proposed fluoridation level in Natick. However, these data do not suggest that 0.91 ppm is without effect, as no village with lower drinking water fluoride concentrations were tested. It is also important to note that the study required *in utero* exposure to these levels of fluoride.

Li and co-workers (9) conducted IQ tests on children living in four areas of the Guizhou Province of China. The areas differed with respect to endemic fluorosis caused by coal burning for grain drying, but were otherwise similar in cultural and socioeconomic aspects. Children aged 8-13 were tested (total of 907) using a Chinese IQ test for children in rural areas. Dental fluorosis was measured using Dean's scale of DMF. Urinary fluoride was also measured and correlated with the measured dental fluorosis (Table II, below).

The results of this study in terms of mean IQ scores and the distribution of IQ scores are summarized in the two tables below.

Table II**Mean IQ scores for children in areas with different prevalence of fluorosis (9)**

Degree of Fluorosis	none	slight	medium	severe
No. of children	226	227	224	230
Dental Fluorosis Index	<0.4	0.8	2.5	3.2
Urinary F (mg/L)	1.02	1.81	2.01	2.69
IQ (mean±SD)	89.9±10.4	89.7±12.7	79.7±12.7	80.3±12.9

Table III
Distribution of child IQ scores from areas of differing fluorosis prevalence (9)

Fluorosis Status	IQ Range						
	<70	70-79	80-89	90- 109	110- 119	120- 129	>129
none	2.6%	9.7%	37.1%	46.8%	3.9%	0.8%	0
slight	3.1%	15.9%	29.1%	47.1%	3.1%	1.3%	<0.4
medium	25.4%	23.7%	29.9%	20.5%	0.4%	0	0
severe	20.9%	26.6%	26.9%	25.2%	0.4%	0	0

Inspection of the first table indicates that there is a 10 point IQ drop in the medium-severe fluorosis area compared to the non-slight fluorosis areas. Inspection of the second table shows that the decrease in IQ is throughout the "bell shaped" IQ curve. There is a marked increase in the percentage of children with IQ less than 70 in the medium-severe fluorosis areas (approximately 3% to more than 21%) and a marked decrease in the percentage of children in the higher IQ ranges (for example, IQ greater than 110 decreases from approximately 5% to 0.4%, a ten-fold decrease).

No correlation was observed between IQ decrement and age of the children. As pointed out by the authors, this suggests that early exposure (in utero or early postnatal) to fluoride is critical to the production of the adverse effect.

Biochemical studies of the brain

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

Mullenix and coworkers (1) measured the concentrations of fluoride in various regions of the brain of both weanling and adult animals exposed to fluoride in their behavioral studies. They detected increased fluoride concentrations in the hippocampus of females, but not males exposed as adults, and of both females and males exposed as weanlings. This pattern of elevation of fluoride in the hippocampus is the same as the pattern of behavioral changes. Several studies have linked hippocampal damage and hyperactivity and cognitive deficits.

Alterations in the hippocampus section of the brain following ingestion of sodium fluoride in drinking water have also been reported by other researchers (10) using a different rat strain (Long Evans) and different measurement endpoints [abnormalities

in the hippocampus and alterations in biochemical reactions in the brain such as beta amyloid and IgM antibody].

Additional studies along these lines have been published in the Chinese literature, but only abstracts or other summaries are available in English. Higher concentrations of fluoride have been found in human embryonic brain tissue obtained from termination of pregnancy operations in areas where fluorosis due to coal burning was prevalent. Detailed studies of these tissues showed that differentiation of brain nerve cells was poor and brain development delays (cited in reference 9). Li (4) reports that the brain of rat pups whose mothers had been exposed to 60 ppm NaF in drinking water had higher nerve cell density in the brain and mild degeneration of organelles of the nerve cells compared to pups from control dams.

Summary and Conclusions Regarding CNS Effects

The study conducted by Mullenix et al., shows central nervous system changes in rats that are likely to be observed as hyperactivity and decreases in IQ or other cognitive (thinking) functions in humans. The observed change depends upon whether the fluoride was administered prenatally or after the pups were born. The observed changes also depended upon whether the animal was male or female. This is a very well conducted study using a previously validated test system, and fluoride doses within the range that humans receive. A lot of weight should be placed on the results of this study.

The two Chinese epidemiology studies suggest that fluoride exposure sufficient to produce moderate to severe dental fluorosis also results in substantial IQ decrements if the fluoride exposure occurs in utero or during the early postnatal period. Taken together, the studies indicate that total fluoride exposure is critical: the IQ decrements were observed due to both drinking water and inhalation exposures. These findings are quite consistent with the animal toxicology data published by Mullenix et al. (1)

Biochemical and histological studies show the accumulation of fluoride in fetal and adult brain tissue and fluoride-induced changes of the structure of brain tissue. These studies support the animal and human studies that fluoride adversely affects human behavior and cognitive function by showing that fluoride reaches brain tissue and alters its appearance.

The Chinese IQ studies (8, 9), the animal toxicity study by Mullenix et al. (1), the studies summarized by Spittle (5), and the biochemical and histological studies together very strongly support the proposition that fluoride has adverse effects on the human central nervous system. Moreover there is good evidence that fluoride is a developmental neurotoxicant (1, 8, 9), meaning that fluoride affects the IQ and behavioral patterns of the developing fetus at doses that are not toxic to the mother.

References

1. Mullenix PJ, PK Denbesten, A Schunior and WJ Kernan, *Neurotoxicity of Sodium Fluoride in Rats.*, Neurotoxicol. and Teratol. **117**(2):169-177. (1995).
2. Mullenix, PJ, WJ Kernan, MS Tassinari, A. Schunior, DP Waber, A Howes and NJ Tar bell, *An Animal Model to study toxicity of central nervous system therapy for childhood acute lymphoblastic leukemia: Effects on Behavior.*, Cancer Res **50**:6461- 6465. (1990).
3. US EPA Guidelines for Developmental Toxicity Risk Assessment. Federal Register **56**(234):63798-63826, (1991).
4. Liu, WX, *Experimental study of behavior and cerebral morphology of rat pups generated by fluorotic female rat.*, Chung-hua Ping Li Hsueh Tsa Chih. **18**(4) :290-292. (English abstract from Medline), (1989)
5. Spittle, B, *Psychopharmacology of fluoride: a review.*, Inter Nat Clin. Psychopharmacol. 9:79-82, (1994)
6. Waldbott, GL, *Allergic Reactions to Fluoride.*, J. Asthma Res. **2**(1):51-64, (1964)
7. Rotton, J, RS Tikofsky, HT Feldman, *Behavioral Effects of Chemicals in Drinking Water.*, J. Appl. Psycho. **67**(2):230-238, (1982)
8. Zhao, LB, GH Liang, DN Zhang, and XR Wu, *Effect of a high fluoride water supply on children's intelligence.*, Fluoride **29**(4):190-192. (1996)
9. Li, XS, JL Zhi, and RO Gao, *Effect of fluoride exposure on intelligence in children.*, Fluoride **28**(4):189-192, (1995)
10. Varner et al., 1995 Soc. for Neuroscience abstract; this may be out soon in Brain Research

Reproductive and Developmental Toxicity (other than nervous system)

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

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

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

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

Reproductive Toxicity

There have been a number of studies of the effects of fluoride ingestion and of water fluoridation on reproductive capability of humans and animals. In its influential 1991 review of water fluoridation, the US Public Health Service (US PHS) (2) found that fluoride may affect reproduction in animals, although some data were contradictory. [It should be noted that the US PHS report was provided to this committee by both Myron Coplan and the Natick Board of Health.]

Several laboratory studies of rodents (rats and mice) exposed to fluoride in food or drinking water showed reduced fertility. Heifers exposed to 5 ppm fluoride in water during four breeding seasons calved at a rate that was only 30% of normal. At higher fluoride doses, the effect was earlier and more severe, which is strongly indicative that the effects observed were due to fluoride and not a confounding factor (cited in 2, 7).

In screech owls, chronic dietary intake of 40 ppm sodium fluoride resulted in significantly smaller egg volume, which is considered a slight-to-moderate reproductive disorder. No gross abnormalities were apparent. (3) Pastel mink fed up to 230 ppm fluoride in their diet did not show adverse reproductive effects such as changes in breeding, gestation, whelping or lactation. However, there was only a 14% survival rate of kits whelped by females fed 385 ppm fluoride. (4)

Several animal studies have examined the effect of fluoride on sperm count, motility and other sperm quality parameters. Narayana and Chinoy (5) fed albino rats 10 mg/kg sodium fluoride for 50 days, and examined the structure and metabolism of the sperm. They observed biochemical alterations that manifest themselves in reduced sperm motility and lower sperm count. Both of these are considered adverse reproductive effects. Withdrawal of sodium fluoride reversed most, but not all of the observed alterations. Addition of ascorbic acid and calcium to the rat diet after withdrawal of the sodium fluoride produced full recovery from the adverse effects of the sodium fluoride.

Susheela and Kumar (6) fed rabbits 10 mg/kg sodium fluoride for 18 or 29 months. At the end of the exposure period, the animals were sacrificed and the structure of the testis, epididymis and vas deferens studied by microscopy. Deleterious changes were observed after 18 months, including absences of mucus droplets in the vas deferens and changes in the epithelial cells lining the lumen of various structures. Spermatogenesis ceased in animals treated for 29 months, but not 18 months, suggesting that longer exposures to fluoride result in more severe effects.

Because of the lack of any human epidemiology studies, Stan Freni, a participant in the US PHS (2) review, initiated an epidemiological study of the possible association of fluoride concentrations in community water supplies and US birth rates. (7) Freni calculated the annual total fertility rate for white women in the age range 10-49 years for the period 1970-1988 in 30 regions (somewhat equivalent to counties) in 9 states. He compared the total fertility rates with measures of fluoride concentrations in drinking water (up to 10 ppm in some individual systems, but averaged over all the drinking water in the county), the percentage of people drinking highly fluoridated (>3 ppm) water, and various socioeconomic factors that are known to affect fertility rates. After accounting for the socioeconomic and other demographic factors, Freni found an association of decreasing total fertility rate (low birth rates) with increasing water fluoride concentrations for most, but not all, of the regions examined. (7)

Developmental Toxicity (other than neurotoxicity)

This subsection describes two animal experiments that tested the effect of fluoride given to pregnant mice. The endpoints studied were various aspects of health, growth and birth defects of the fetus. However, neither study examined functional deficits, such as neurobehavioral deficits. These are fully described in the neurotoxicity section of this report.

Collins and coworkers (8) published a study on the effects of sodium fluoride in drinking water provided to pregnant rats on the health of the fetuses. In this study, they dosed pregnant female rats with drinking water containing 0, 10, 25, 100, 175 or 250 ppm NaF every day throughout gestation. The NaF did not appear to produce any change in fetal growth or affect the development of specific bones. However at

the highest dose (250 ppm NaF), there was a significant increase in the average number of fetuses with three or more skeletal variations.

Heindel and coworkers (9) evaluated the effects of sodium fluoride in drinking water provided to pregnant rats and rabbits on the health of the fetuses at the end of gestation. In this study, they dosed rats with 0, 50, 150, or 300 ppm NaF in drinking water during gestational days 6-15; rabbits were dosed on GD 6-19 with 0, 100, 200 or 400 ppm NaF in drinking water. The animals were killed and the fetuses were examined at the end of gestation (GD 20 for rats, GD 30 for rabbits). No clear signs of maternal toxicity were noted at the 150 ppm level and lower. No developmental effects, manifest as post-implantation loss, mean fetal body weight/litter, external, visceral or skeletal malformations were observed.

Summary

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

Regarding fluoride and developmental effects: These two studies do not show any fluoride associated developmental effects such as malformations (birth defects), post-implantation loss, or death of the fetus or infant at drinking water doses up to 175 ppm sodium fluoride. There may be some effects above 250 ppm sodium fluoride, particularly skeletal related effects. The experimental protocols used in these two studies do not test for cognitive or neurobehavioral changes following *in utero* exposure, such as those observed by Mullenix and coworkers described in the neurotoxicity section.

References

1. Rogers, JM, and RJ Kavlock. *Developmental Toxicology*. in Casarett & Doull's *Toxicology* 5th ed. C. Klaassen, ed., McGraw Hill, NY, (1996)
2. U.S. Public Health Service. Committee to Coordinate Environmental Health and Related Programs, (1991).
3. Hoffman, DJ, OH Pattee, and SN Wiemeyer, *Effects of fluoride on screech owl reproduction: teratological evaluation, growth, and blood chemistry in hatchlings*. *Toxicol. Let* **26**(1):19-24, (1985)
4. Aulerich, RJ, AC Napolitano, SJ, Bursian, BA Olson, and JR Hochstein, *Chronic toxicity of dietary fluorine to mink*. *J Anim Sci* **65**(6):1759-1767, (1987)
5. Narayana, MV and NJ Chinoy, *Reversible effects of sodium fluoride ingestion on spermatozoa of the rat*. *Int. J. Fertile. Menopausal Stud.* **39**(6):337-346, (1994)
6. Susheela, AK, and A Kumar, *A study of the effect of high concentrations of fluoride on the reproductive organs of male rabbits using light and scanning electron microscopy*. *J. Reprod. Fertile.* **92**(2):353-360, (1991)
7. Freni, S.C, *Exposure to high fluoride concentrations in drinking water is associated with decreased birth rates*. *J. Toxicol. and Environ. Health* **42**:109-121, (1994)
8. Collins, TF, RL Sprando, ME Shackelford, TN Black, MJK Ames et al., *Developmental toxicity of sodium fluoride in rats*. *Food Chem Toxicol.* **33**(11):951-960. (1995)
9. Heindel, JJ, HK Bates, CJ Price, MC Marr, CB Myers, BA Schwetz, *Developmental toxicity evaluation of sodium fluoride administered to rats and rabbits in drinking water*. *Fundam. Appl. Toxicol.* **30**(2):162-177, (1996)

Lead Contamination

This section discusses the impact of specific concerns about the role of fluoridation relative to the issue of lead contamination.

Lead Contamination

Lead contamination is a recognized concern in water supplies. As such, it is regulated by the EPA. This regulation stipulates that certain action(s) must be implemented if the lead concentration exceeds 15 parts per billion (ppb) which is termed the action level. The 1986 Safe Drinking Water Act Amendment prohibits the use of lead pipes and limits the lead content in brass plumbing components to 8%. Older facilities are likely to have a larger amount of lead used throughout the water distribution system.

Although the lead level in ground and surface water supplies may be low, the level of lead can increase to the action level (as specified by the EPA) depending on chemical and physical factors. The factors affecting the amount of lead contamination are: 1. The corrosiveness of the water which is dependent on pH, alkalinity (or buffering capacity), and mineral content, 2. Age of lead-soldered joints and other lead components, 3. Quantity and surface area of lead materials, 4. Time and temperature of water in contact with lead surfaces. (7)

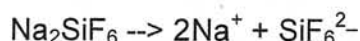
The health effects of lead can be as severe as coma and possible death at high blood levels (exceeding 80 ug/dL). Severe effects are unlikely to occur from drinking water. Low level lead exposure (as determined by blood lead levels greater than 10 ug/dL) is more likely to occur from drinking water. Low level lead exposure is associated with adverse effects on the central nervous system such as decreased intelligence and impaired neurobehavioral development. (7)

Corrosion is one of the mechanisms by which lead contamination increases in the water supply. The fluoridating agents Natick intends to use, fluorosilicic acid and sodium fluorosilicate, are both corrosive in certain conditions. As stated in the Water Fluoridation Manual for Plant Operators (1):

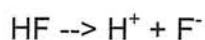
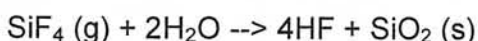
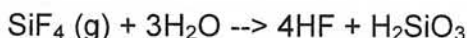
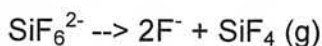
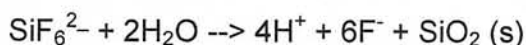
".....Under certain water-quality conditions, a small increase in the corrosivity of potable water that is already corrosive may be observed after treatment with alum, chlorine, fluorosilicic acid, or sodium fluorosilicate. This increase in corrosivity is caused by a depression of pH resulting from these treatments and occurs in potable water with a low buffering capacity. The increase in the corrosivity of potable water as a result of the addition of the fluorosilicic acid or sodium fluorosilicate is negligible for most water systems, but where it is significant, it can be reduced by adding small amounts of lime or caustic soda".

An example of the relationship between increased lead levels associated with fluoridation occurred in 1992 in Tacoma, WA. Data from the City of Tacoma water treatment plant show water sampling parameters from the same neighborhood before and after the fluoridation equipment broke down (2). The pH was identical at 6.6 in both cases, yet with fluoridation 20% of the homes exceeded the EPA action level for lead, whereas 10% of the homes exceeded the level without fluoridation.

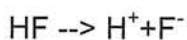
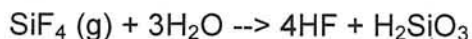
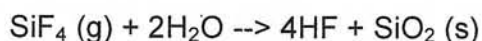
An important aspect of this corrosivity effect requires a consideration of the dissociation of sodium fluorosilicate, which takes place as follows: (1, 5)



Further dissociation of fluorosilicate ion takes place utilizing different pathways:



Similar to sodium fluorosilicate dissociation, fluorosilicic acid dissociation occurs as follows:



These reactions do not occur at equal rates, however the end products are fluoride ion (F^-), silicon compounds, and hydrogen ion (H^+ ; hydrated). The rates of reaction are of concern when considering corrosion effects, since the initial reaction will release some hydrogen ions which will lower the pH, increasing the acidity. The initial pH drop can be neutralized at the water plant; however, the slower breakdown of intermediate products like silicon tetrafluoride will happen gradually, quite likely after leaving the plant. As a result of this delayed hydrolysis of the silicon compounds, increased acidity will be experienced throughout the water distribution system. (6)

A results of a study of water fluoridation agents (if any) and the venous blood lead levels of children of ages 0 to 4 years is shown in Table I, below. (3, 4, 8, 9)

These data illustrate that the reported blood levels exceeded the limit of 10 ug/dL in 0.75% of the children in non-fluoridated communities while more than twice as many (1.53%) of the children in the fluorosilicic acid-fluoridated communities exceeded the recommended limit. (3) The communities represented are comparable in size to Natick, ranging from 15,000 to 50,000.

Table I
Fluoridation and Venous Blood Lead Levels in MA Children Aged 0-4 Yrs.

Percent of Children with Venous Blood (VB) Levels Greater than 10
Micrograms/Deciliter (3,4,8,9)

Number of Communities	Total Population	Number Screened	Number with VB > 10 ug/dl	Incidence n/N	Fluoridation Agent Used
	(thousands)	N	n	%	
36	882.8	40669	306	0.75	None
20	416.0	17441	181	1.04	NaF
30	865.3	36804	564	1.53	H ₂ SiF ₆

Summary

Fluorosilicic acid and sodium fluorosilicate are acknowledged to have corrosive abilities. Even when maintained diligently, an increase in lead levels should be anticipated at point-of-use in homes after exposure to the distribution lines where lead solder and valves are in contact with water. The use of fluorosilicic acid and sodium fluorosilicate poses a specific risk since they have been associated with increased blood lead levels.

References

1. U.S. Department of Health and Human Services, *Water Fluoridation - A Manual for Engineers and Technicians*, (1986).
2. City of Tacoma Water Quality Sampling Parameters, (1992).
3. Bailey, A, et al., *Poisoned landscape: the epidemiology of environmental lead exposure in Massachusetts children 1990-1991*, Soc. Sci. Med., **6**:757, (1994).
4. Masters, R, Research supported by a grant from the Mass. Health Research Institute, Contract H64/CCH105095-03 from the Centers for Disease Control and Prevention and by grant MCJ-330597 from the Maternal and Child Health Bureau.
5. American Water Works Association, *Water Fluoridation Principles and Practices*, 3rd Edition, (1988).
6. Coulton, E, *Fluosilicic Acid*, J. Chem. Ed., **10**:563, (1958).
7. U. S. Department of Health and Human Services, *Preventing Lead Poisoning in Young Children*, 1991
8. Centers for Disease Control, *Fluoridation Census*, (1992)
9. Coplan, MJ, *The Fluoridation-Lead Connection*, Unpublished, (1997)

Non - Health Issues

Although the Committee was not specifically asked to address any other issues related to fluoridation, the members felt that certain of the following matters would/should bear upon the decision of the Natick Board of Selectmen vis-à-vis the overall question of fluoridation.

Forced Medication/Freedom of Choice

It has been said that **"....fluoride, at the levels recommended by profluoridationists for reducing tooth decay is not an essential nutrient; is not a natural substance for babies or for most adults; is not a compulsory medication; but is an expensive-to-avoid medication with an uncontrolled dose; and is harmful to some people."** (1) As a result of these characteristics, the issue of fluoridation cannot be properly discussed without a concomitant discussion about the ethical issues involved. (1-3). In spite of this, the position taken by most profluoridationists is that the questions involved are of a scientific nature only and therefore should be addressed only by those well-versed in these issues. (4-6) However, a powerful case can be made that there are indeed ethical questions involved and that **perhaps these issues should be addressed first and not merely by the technically elite.** The obligation that this imposes upon any government official, including the Board of Selectmen, cannot be minimized. It is therefore appropriate that the Committee recommend that a careful reading of reference (1) below should precede **any action** taken in the resolution of the Natick fluoridation issue.

Economic Issues of Fluoridation

The issue of economic costs in a massive program such as the fluoridation of a public water supply is difficult to treat exhaustively. Moreover, the Committee has no great degree of financial expertise. Nevertheless, we would be remiss if we failed to identify those costs which come to mind. These are treated in the following sections.

Direct Process Costs

The Natick water department expects to pump approximately 1.5 **billion** gallons of water this year to Natick consumers. Of this amount, approximately 60 **thousand** gallons will be used **weekly** (0.208 per cent) in the preparation of food and for drinking purposes. (7) Fluoridation of 1.5 billion gallons of water to the 1 ppm level will require 15,720 lbs. of H_2SiF_6 . (7) The projected annual cost for fluoridation of the Natick wells is believed to be a minimum of \$35,000 per year. (8) Other estimates range from \$30,000 to \$50,000 per year. Moreover, it is not clear whether these costs

take into consideration such factors as training of personnel, amortization costs, repairs and replacement of both privately owned and town-owned equipment due to increased corrosion (unrelated to lead issues) which is known to occur. (9) From these facts, two interesting pieces of data emerge. These involve the issues of environmental impact and the reputed cost effectiveness of the fluoridation effort.

Cost-effectiveness of Fluoridation

Public health officials have always considered fluoridation of public water supplies to be a cost effective approach to giving children fluoride. (10) However, the economics have changed in the last few decades. We feel that it deserves a closer look and we note some concerns below.

Two important factors need to be taken into consideration when considering the 'savings' attributable to reduction in caries caused by fluoridation. First is the fact that modern DMFS scores (explained in Appendix D) are much lower in all communities than was the case when fluoridation was first started. (11) This means that there are fewer cavities in the population and that any percentage reduction in the incidence of cavities involves many fewer incidences than was previously the case. The second salient fact is that 55% of the children in communities having unfluoridated water are cavity free. (12) Clearly, fluoridation is not cost effective for this segment of the population.

The calculation of cost effectiveness of fluoridation is very complex. One has to consider the savings due to (possibly) fewer cavities in some children and the cost to treat those children. However, it is also true that there will be increased treatment costs due to dental fluorosis (between 10-30% of children in communities that fluoridate develop some form of dental fluorosis-see section on dental fluorosis). Although these costs are not borne by the community at large, they should be considered in any assessment of cost-effectiveness. (13)

It is beyond the scope of this committee to make such complicated calculations but it seems clear that there will be a greater increase in fluorosis than there will be a reduction in cavities.

Indirect Costs

The committee has also identified indirect costs that should be included in the cost effectiveness calculations. These include the costs borne by individual Natick residents who choose not to drink fluoridated water and individual Natick residents who may incur medical or dental costs due to drinking fluoridated water. Finally, there are other costs to the town such as amortization, repair, etc., of equipment necessary

to the program. These cost include (but are not limited to) the following identifiable items:

- Increased dental costs (not covered by insurance) to treat fluorosis
- Purchase of unfluoridated water from other sources (\$3-4 per week)
- Purchase of fluoride removal equipment
- Increased medical costs
- Legal costs to the town to defend against lawsuits (see below)
- Increased plumbing costs resulting from corrosion. (9)

Liability Ramifications

If the town fluoridates its public water supply, there is a possibility that legal culpability may result from any number of sources. For example, if continued research into the correlation between fluoride and diminished IQ (or other factors) substantiates the research results described above (see the sections on Central Nervous System effects and Lead Contamination), the town may well be held liable. (That the town in this case would have unlimited company may be of little consolation!) In addition, in spite of the best efforts of the town, a hazardous spill may occur, as has already occurred in several other communities. In at least one of these incidents, multiple lawsuits have been filed. (14)

Environmental Impact

In the process of fluoridation of Natick's water supply, 15,680 pounds of fluoride per year will enter the environment (assuming 50% fluoride retention within the human body). This fluoride will be dispersed via a number of mechanisms into a variety of paths including incorporation into locally grown foods and locally raised livestock. It is easy to see how the presence of fluoride has become so endemic that many researchers have postulated that it is no longer possible to determine whether fluoride in public water supplies has any value, without considering how to quantify this effect. (13)

In order to place this issue in proper perspective, it is insightful to consider that the effect of having the entire United States being served with fluoridated water will result in the dumping of at least 100 million pounds of fluoride annually within the United States alone! Needless to say, for any other material, beneficial or otherwise, a far greater public outcry would be raised.

Political Ramifications of Referenda and Plebiscites

The town of Natick appears to be deeply divided on the subject of fluoridation. The issues of a "binding" vs. a "non-binding" referendum; the **staledatedness** of a popular

vote and the question of whether a small majority of a larger number of voters is more valid than a larger majority of a smaller number of voters in a plebiscite are questions that interest all of the parties concerned, including the members of the Committee. However, it seems clear that since neither the Committee nor the various parties in the fluoridation issue will resolve these questions, we will not speculate on the legal opinions which may be rendered thereon. On the other hand, certain valid points can be made.

First of all, the information and research about fluoridation and the effects of fluoride has grown tremendously in the past few years. When the first vote on fluoridation was taken in Natick, much less was publicly known about the possible negative effects of fluoride and the decreasing impact of fluoridation of drinking water. For this reason we feel that more attention should be paid to the latest vote in Natick in which the voters failed to support the fluoridation of Natick drinking water.

Secondly, even if there is a possible reduction of caries in Natick due to fluoridation, this has to be weighed against the possible harm caused to some number of residents of the town due to increased fluoride in their diets. It has been argued that fluoridation of the drinking water is the most cost effective method of getting additional fluoride into the diet of children. However, this cost savings (if indeed there is a savings) has to be weighed against the increased cost of medical care for those who may be negatively effected by an increase in fluoride.

Summary of Non-Health Issues

Fluoridation of the Natick water supply has multiple implications beyond the risk vs. benefit considerations that were the primary focus of this report. There are environmental impacts, unquantifiable potential costs, liability and political ramifications that must be addressed. Further, the issue of cost-effectiveness must be more fully explored before an intelligent decision can be rendered. To varying degrees, all of the matters addressed in this section of the report would tend to argue against fluoridation in Natick. That is to say that all of these factors, quite aside from the main issue of the benefits of fluoride, do not cast the matter in a positive light.

References

1. Diesendorf, M, *How science can illuminate ethical debates - A case study on water fluoridation.*, Fluoride, **28**(2):87-104, (1995)
2. Colquhoun, J, *Why I changed my opinion about water fluoridation*, in Perspectives in Biology and Medicine, (in press)
3. Waldbott, GL, et. al., *Fluoride - The Great Dilemma*, Coronado Press (1978)
4. Koop, CE, Chem. & Engin. News, **28**:2 (1988)
5. Breda, DJ, et al., *Letter from the Natick Board of Health to J. Moran*, Feb. 10, 1997
6. (Authorship unknown), List of National and International Organizations that Endorse or Support Water Fluoridation, (undated)
7. *Natick Annual Town Report.*, p 37., (1996). Drinking/cooking gallonage estimated using the values of five gallons per household per week and 12000 households. The calculation for the annual amount of fluorosilicic acid required to fluoridate Natick's water is: $\{(1.5 \times 10^9 \text{ gal})(3785 \text{ g/gal.})/10^6/456 \text{ g/lb}\}/0.792$ The quantity in brackets [] is the required number of pounds of F⁻ and the value 0.792 is the number of pounds of F⁻ per pound of fluorosilicic acid
8. Conley, F, Natick Town Administrator via Ball, J, Clerk/Selectman
9. Sargent, E, (Superintendent of Water Supplies, Wilmington, MA), *Letter to townspeople of Wilmington*, Feb. 28, 1962
10. Ripa, L., *A Half-century of Community Water Fluoridation...Review and Commentary*, J. Pub. Hlth. Dent., **53**; 17 (1993)
11. Brunelle, JA and Carlos, JP, *Recent trends in dental caries in US children and the effect of water fluoridation*, J. Dent. Res., **69**:723-727 (1990)
12. Szpunar, SM and Burt, BA, *Dental Caries, fluorosis, and fluoride exposure in Michigan schoolchildren*, J. Dent. Res., **67**:802-806 (1988)
13. Lewis, DW and Banting, DW, *Water fluoridation: current effectiveness and dental fluorosis*, Community Dent. Oral Epidemiol., **22**:153-158 (1994)
14. Bevis, M, *10 Years of Fluoride leaks, spills and overfeeds in the U.S.*, National Fluoridation News, p.3, Nov. 1981

Question 2 The Establishment of an Appropriate Dosage

2. If your answer to question 1 is "No" -- i.e. you believe Natick's water should be fluoridated -- do you believe that steps should be taken to establish the appropriate dosage before such fluoridation begins?

Discussion

Conclusion and Committee Response to Question 2

In view of the Findings, Conclusion and Recommendations given on p. 3, this is not an easy question to answer. One of the most troublesome aspects of this entire investigation involves the determination of total fluoride intake within the community. Moreover, both the sources and the quantities of ingested fluoride may be all but impossible to determine except in the simplest cases. In fact, it is precisely this inability to identify and quantify the uncontrollable sources of fluoride, particularly in the young, that argue against adding a so-called "controlled" dose of fluoride into any public water system.

It has been suggested that the increased incidence of dental fluorosis is, to a large degree, the result of this "uncontrollable" fluoride ingestion. Nevertheless, Appendix C has been included to provide some insight and guidelines with regard to recommended maximum fluoride intakes for various age groups.

Question 3 The Advisability of a Dental Survey

3. *If your answer to question 2 is "Yes", do you believe that an outside organization should be engaged to examine Natick school children and determine their DMFS (decayed, missing and filled surfaces) levels as an aid to selecting an optimum fluoridation level?*

Discussion

Conclusion and/or Committee Response to Question 3

From a pragmatic view, the debate on fluoridation in Natick may continue regardless of either the conclusions reached in this report or the actions taken by the Board of Selectmen. If Natick decides to go ahead with fluoridation, then those who are opposed will probably continue to lobby for the cessation thereof and if Natick decides not to fluoridate then the profluoridationists will probably continue to lobby for fluoridation.

Nevertheless, it is patently clear that all of the past and current debate has taken place without any hard data about the incidence of dental caries in Natick. It seems equally clear that unless we know whether there is any real need for fluoridation in Natick the question of benefit vs. risk, whether perceived or real, can never be answered. For this reason we recommend that a study of the incidence of dental caries and the signs of fluorosis in the youth of Natick be undertaken regardless of whether the water is fluoridated or not. Without this baseline we will never know the possible benefits to fluoridation in Natick.

Question 4 Source of the Survey and Probable Costs

4. *If your answer to question 3 is "Yes", what organizations (identify at least two) are qualified to conduct such a survey, and what are preliminary estimates of the costs involved?*

Discussion

Conclusion and Committee Response to Question 4

The Committee consensus is that this question cannot be easily answered without additional further study. Some have suggested that such a study could take as long as the time taken to produce the current document. The Committee recommends that if such a study is undertaken, a neutral organization should be selected and appropriate oversight provisions should be made.

Appendix A Source Materials

During the planning stage of this report, the committee had considered including copies of all of the supporting documents received. During the ensuing weeks, it has become clear that such an approach would merely ensure that this report would be less useful to the Selectmen as well as the community at large. Therefore, this section is comprised of a list of those materials provided by each requested party. A separate binder(s) containing all of the materials provided or referenced will also be prepared and this will be provided to the Morse Library, as will a bound copy of this report.

As can be seen from the following lists, proponents and opponents of the fluoridation issue have provided (often substantially) more than had been requested by the committee. These materials are listed below. [Note: All of the concerned parties also submitted additional materials later in the study which are not listed below.] Since the committee, in addition, reserved the right to search the literature on their own, no attempt was made to limit the selections provided. However, "propaganda" pieces such as those characterized by hysteria and/or polemics, and in particular, those documents which contained no referential material generally were less favorably received by the committee than were peer-reviewed and/or clearly unpartisan attempts to summarize the field. The numbers assigned to any of the provided materials listed below should not be construed to indicate correspondence with the references noted in any of the sections of this report. They are merely used to enumerate the documents provided.

Materials Provided by the Board of Health

1. Wade, R., Cover memo to Fluoride Study Committee
2. Ripa, L., *A Half-century of Community Water Fluoridation....Review and Commentary*, J. Pub. Hlth. Dent., 53; 17 (1993)
3. Authorship Uncertain, *Health Effects of Ingested Fluoride: Executive Summary* (undated)
4. Flynn, J. P., *Letter from the Town Counsel to the Bds. of Health & Selectmen*, May 8, 1997
5. Harte, D. B., *Letter from the Mass. Dental Society to Board of Selectmen*, Feb. 10, 1997
6. Taddeo, A. C., et al., Legal Notice May 28, 1988
7. Mass. Dept. of Health, *Sources of Fluoride Factsheet* (1996)
8. National Research Council (NRC/NAS), *Drinking Water Concerns Unwarranted*, Press Release Aug. 18, 1993
9. *List of National and International Organizations that Endorse or Support Water Fluoridation*, (authorship unknown; undated)
10. American Association of Public Health Dentistry, *Resolution on Community Water Fluoridation*, J. Pub. Hlth. Dent., 53: 59 (1993)
11. National Osteoporosis Foundation, *FDA Advisory Committee Recommends Approval of Sodium Fluoride Treatment for Osteoporosis*, News Release, Nov. 17, 1995
12. Manley, A. F., *Surgeon General Statement on Community Fluoridation*, USPHS, Dec. 14, 1995
13. American Dental Association, *Fiftieth Anniversary of Water Fluoridation*, Press Release, Apr. 1995

14. Todd, J. S., *Letter from the A. M. A. to T. J. Ginley*, Apr. 30, 1990
15. Consumer Reports, *A Two-part Report on Fluoridation*, Jul., Aug. 1978
16. *Community Water Fluoridation*, Mass. Dept. of Public Health Brochure, (undated)
17. Center for Disease Control and Prevention, *Fluoride: The Benefits Can Last a Lifetime*, Press Release (?), (undated)
18. American Dental Association, *Communication Points Regarding Community Water Fluoridation*, Apr. 1995
19. Taddeo, A. C., et al. *Letter from the Natick Board of Health to E. Dlott*, Mar. 21 1996
20. Breda, D. J., et al., *Letter from the Natick Board of Health to J. Moran*, Feb. 10, 1997
21. Natick Board of Health, Press Release, Mar. 11, 1997
22. Natick Board of Health, Press Release, Apr. 29, 1997
23. Delli Colli, P. A., *Letter from the Natick Board of Health to the Natick Bulletin*, Mar. 18, 1997
24. Various other materials presented at a later date. Though not enumerated here, this material will be included in the reference package to be provided to the Morse Library.

Materials Provided by Shirley Brown

1. Hileman, B, *Flouridation of Water*, Chem. & Eng. News, **66**:26, (1988) (several copies)
2. Agency for Toxic Substances and Disease Registry U.S. Public Helath Service, *Toxicological Profile for Fluorides, Hydrogen Fluoride, and Fluorine*, TP-91/17, (1993) (selected pages)
3. Glass, G, *Water Fluoridation: The Enhanced Toxicity Factor?*, The Australian Fluoridation News, 31:6, (1995)
4. Connett, P, and Connett, E, *The fluoridation of drinking water: a house of cards waiting to fall.*, Waste Not, (1996)
5. Mullenix, PJ, et al., *Neurotoxicity of sodium fluoride in Rats*, Neurotoxicology and Teratology, **17**:169, (1995)
6. Li, XS, et al., *Effect of fluoride exposure on intelligence in children*, Fluoride, 28:4, 189, (1995)
7. Spittle, BJ, *Psychopharmacology of fluoride: a review*, International Clinical Psychopharmacology, 9:79,(1994)
8. Marcus, W, *Memorandum on fluoride conference to review the NTP draft fluoride report*, US EPA, (1990)
9. Diesendorf, M, Colquhoun, J, Spittle, BJ, Everingham, DN, and Clutterback, FW, *New Evidence on Fluoridation*, Aus. NZ J. Pub. Health, 21(2):187-190, (1997)
10. Lindsay, R, *Fluoride and Bone - Quality versus Quantity*, N. E. J. Med., 322(12):845-846, (1990)
11. Anonymous, *Fluoride Frenzy - Rats, Research, Politics & Propaganda*, Sacramento News & Entertainment Weekly, 9(4):16-19, (1997)
12. Miscellaneous, Numerous News Releases, News Articles, Fax, Letters & Memoranda
13. Walker, GSR, *Fluorides and Birth Defects*, The Australian Fluoridation News, 31:5, (1995)
14. Waldbott, GL, *Fluoridation: The Great Dilemma*, Coronado Press, p. 189, (1978) (2 copies)
15. Yiammouyiannis, J, *Fluoride, the Aging Factor*, Health Action Press, Delaware, OH, (1986) (2 copies)
16. Our Rights, Our Water, *Facts about Fluoride*, (1977)
17. Kranzfelder, K, *Letter to Wini Silko*, Jan 4, 1991
18. Center for Health Action, *Misc. quotations and references* (undated)
19. Dietrich-Goetz, W, *Drinking water Contraception*, (Letter) Fluoride, **28**:121 (1995)
20. Steelink, C, *Fluoridation Controversy* (Letter) Chem. and Eng. News, **70**:2 (1992)
21. Micromedex, Inc., *Hazardous Substance Database*, **20**:(2 unnumbered pages), (1994)
22. American Family Physician, *Special Medical Reports*, **53**:7, (1996)
23. Health Alliance International, *Water Fluoridation* (press release) May 5, 1997
24. Various other materials presented at a later date. Though not enumerated here, this material will be included in the reference package to be provided to the Morse Library.

Materials Provided by Myron Coplan

1. Coplan, M. J., *Cover letter to the Natick Fluoride Study Committee*, July 10, 1997
2. Coplan, M. J., *The Massachusetts Lead/Fluoridation Connection in 1992*, (unpubl.)
3. Coplan, M. J., *Fifty Years of Tooth Decay and Fluoridation*, (unpubl.) (1997)
4. Report of the Ad Hoc Committee, *Review of Fluoride Benefits and Risks*, USPHS, (1991). (Only 89 pages of this document were provided.)
5. DePaola, P., et al., *Changes in Caries Prevalence of Massachusetts Children over Thirty Years*, (paper presented at 1981 IADR)
6. Diesendorf, M., *The mystery of declining tooth decay*, Nature, 322:125(1986)
7. Shell, E. R., *An Endless Debate*, Atlantic Monthly, Aug. 1986, p26
8. Emsley, J., et al., *An unexpectedly Strong Hydrogen Bond....Systems*, J. A. C. S., 83, 24 (1981)
9. Diesendorf, M., et al., *New Evidence on Fluoridation*, Aus. & NZ J. Pub. Hlth. 21, 187 (1997)
10. Various other materials presented at a later date. Though not enumerated here, this material will be included in the reference package to be provided to the Morse Library.

Materials Provided by NFSC Members

These materials are listed throughout this report. A collection of these references is being prepared and will be provided to the Morse Library.

Appendix B The Difficulty of Research on Fluoridation

This committee encountered many problems in conducting a literature search on the subject of fluoridation. The topic has become a very emotional one and proponents on both sides of the issue (profluoridationists and antifuoridationists) are guilty of not giving full credit to research from their opponents. However, since the 'power' and money lies mostly within the profluoridation side of the issue, (the larger, powerful organizations such as the AMA and ADA historically have been in favor of fluoridation) the antifuoridationists have been given 'short shrift' due their inability to get approval and funding for studies which lead to, or may lead to, conclusions that are in opposition to fluoridation. In fact, some profluoridation reviews do not even record or address these opposing views. It is therefore very important that when one looks into the literature regarding fluoridation, one keep in mind the biases that exist on this issue. This matter has been addressed in Hileman's review of fluoridation (which can fairly be described as non-partisan). We have included an excerpt from this review to give the reader some insight into the problems of research on the fluoridation issue. (1)

"Ever since the Public Health Service (PHS) endorsed fluoridation in 1950, detractors have charged that PHS and the medical and dental establishment, such as the American Medical Association (AMA) and the American Dental Association (ADA), have suppressed adverse scientific information about its effects.

Some of those who generally support fluoridation make similar charges. For example, Zev Remba, the Washington Bureau editor of AGD Impact, the monthly publication of the Academy of General Dentistry, wrote last year that supporters of fluoridation have had an "unwillingness to release any information that would cast fluorides in a negative light," and that organized dentistry has lost "its objectivity - the ability to consider varying viewpoints together with scientific data to reach a sensible conclusion."

The dozen or so scientists C&EN was able to contact who have done research suggesting negative effects from fluoridation agree on this aspect. They all say that fluoridation research is unusual in this respect.

If the lifeblood of science is open debate of evidence, scientific journals are the veins and arteries of the body scientific. Yet journal editors often have refused for political reasons to publish information that raises questions about fluoridation. A letter from Bernard P. Tillis, editor of the New York State Dental Journal, written in February 1984 to Geoffrey E. Smith, a dental surgeon from Melbourne, Australia, says: "Your paper ... was read here with interest," but it is not appropriate for publication at this time because "the opposition to fluoridation has become virulent again." The paper poses the question: Are people ingesting increasing amounts of fluoride and can they do so with impunity?

Sohan L. Manocha, now a lawyer, and Harold Warner, professor emeritus of biomedical engineering at Emory University medical school in Atlanta, received a similar letter in 1974 from the editor of AMA's Archives of Environmental Health. The editor rejected a report Manocha and Warner submitted on enzyme changes in monkeys who were drinking fluoridated water because of reviewers' comments such as: "I would recommend that this paper not be

accepted for publication at this time" because "this is a sensitive subject and any publication in this area is subject to interpretation by anti fluoridation groups."

These papers were subsequently published in prestigious British journals, Science Progress (Oxford) and Histochemical Journal. Many other authors have reported similar difficulties publishing original data that suggest adverse effects of fluoridated water. Most authoritative scientific overviews of fluoridation have omitted negative information about it, even when the oversight is pointed out. Phillips Grandjean, professor of environmental medicine at Odense University in Denmark, wrote to the Environmental Protection Agency in June 1985 about a World Health Organization study on fluorine and fluorides: "Information which could cast any doubt on the advantage of fluoride supplements was left out by the Task Group. Unless I had been present myself, I would have found it hard to believe."

In his 1973 Ph. D. thesis on the fluoridation controversy, Edward Groth, III, a Stanford biology graduate student at that time, concluded that the vast majority of reviews of the literature were designed to promote fluoridation, not to examine evidence objectively. Groth also noted a number of anti fluoridation reviews that were equally biased.

According to Robert J. Carton, an environmental scientist at EPA, the scientific assessment of fluoride's health risks written by the agency in 1985 "omits 90% of the literature on mutagenicity, most of which suggests fluoride is a mutagen."

Several scientists in the U.S. and other countries who have done research or written reports questioning the benefits of fluoridation or suggesting possible health risks were discouraged by their employers from publishing their findings. After their paper had been rejected by the editor of Archives of Environmental Health, Manocha and Warner were told by the director of their department not to try to publish their findings in any other U.S. journal. NIDR had warned the director that the research results would harm the cause of fluoridation. Eventually, Manocha and Warner were granted permission to publish their work in a foreign journal.

In 1982, John A. Colquhoun, former principal dental officer in the Department of Health in Auckland, New Zealand, was told after writing a report that showed no benefit from fluoridation in New Zealand that the department refused him permission to publish it.

In 1980, Brian Dementi, then toxicologist at the Virginia Department of Health, wrote a comprehensive report on "Fluoride and Drinking Water" that suggested possible health risks from fluoridation. This 36-page study has been purged from the department's library even though it is the only one the department has prepared on the subject. According to current employees, no copy exists anywhere in the department. Spokesmen say the report was thrown away because it was old but also say the department will be preparing another report on the subject soon.

An ADA white paper written in 1979 states: "Dentists' nonparticipation [in fluoridation promotion] is overt neglect of professional responsibility." An ADA spokesperson says this is still the association's official policy. In recent years, several dentists who have testified on the anti fluoridation side have been reprimanded by their state dental officers.

ADA and PHS also have actively discouraged research into the health risks of fluoridation by attacking the work or the character of the investigators. As part of their political campaign, they have over the years collected information on perceived anti fluoridation scientists,

leaders, and organizations. Newspaper articles about them are stored in files, as are letters about them from various proponents of fluoridation. Little or no effort has been made to verify the accuracy of this information. It is used not only in efforts to counteract arguments of the anti-fluoridationists, but also to discredit the work and objectivity of U.S. scientists whose research suggests possible health risks from fluoridation.

One example is the false information about the late George L. Waldbott, founder and chief of allergy clinics in four Detroit hospitals, that ADA disseminated widely to discredit the validity of his research. Rather than deal scientifically with his work, ADA mounted a campaign of criticism based largely on a letter from a West German health officer, Heinrich Horning. The letter made a number of untrue statements, including an allegation that Waldbott obtained his information on patients' reactions to fluoride solely from the use of questionnaires. ADA published Horning's letter in its journal in 1956 and distributed a news release based on the letter. ADA later published Waldbott's response to this letter. But the widely disseminated original news release was not altered or corrected, and continued to be published in many places. As late as 1985, it was still being quoted. Once political attacks effectively portrayed him as "anti fluoridation," Waldbott's work was largely ignored by physicians and scientists.

In November 1962 and 1965, ADA included in its journal long directories of information about anti-fluoridation scientists, organizations, leaders, and others known to be opposed to fluoridation. Listed in alphabetical order were reputable scientists, convicted felons, food faddists, scientific organizations, and the Ku Klux Klan. Information was given about each, including quotes from newspaper articles, some of which contained false data. The information was published for use by proponents of fluoridation in local fluoridation referenda.

John S. Small, information specialist at the National Institute of Dental Research, is quite willing to talk about the files he keeps on anti fluoridation organizations and their leaders. **"Of course, we gather information," he says. "These people are running all over the country opposing fluoridation. We have to know what they are up to."** Consumer advocate Ralph Nader has a different view of this activity. He calls it an **"institutionalized witchhunt."**

It is easy to understand why research on risks of fluoridation has never been more vigorously pursued. Most of the individuals and agencies involved have been promoting fluoridation publicly for nearly 40 years. Research that suggests possible harm threatens them with a loss of face. For example, PHS has historically been the principal source of funds for fluoride research; but ever since June 1950, PHS has been officially committed to and responsible for promoting fluoridation. Thus, the agency has a fundamental conflict of interest.

Colquhoun, now teaching the history of education at the University of Auckland, offers another explanation for what appears to be the suppression of research. He notes that the editorial policy of scientific journals has **"generally been to not publish material which overtly opposes the fluoridation paradigm."** Scientific journals employ a referee system of peer review. But when the overwhelming majority of experts in an area from which the referees are selected are committed to the shared paradigm of fluoridation, Colquhoun notes, the system lends itself to preservation and continuation of the traditional belief that fluoridation is safe and effective. This results in **"single-minded promotion, but poor quality research, and an apparent inability to flexibly reassess in the presence of unexpected new data,"** he says....." (1)

Reference

- (1) Hileman, B, *Fluoridation of Water*, Chem. & Eng. News, 66:26, (1988)

Appendix C Recommended Dosage of Fluoride

The following table provides the currently recommended fluoride supplementation for children living in a community with fluoride level in the ranges shown. (1) These doses were revised downward in 1995.

Table I
Recommended Fluoride Supplementation (mg/day) for Children

Range of Child's Age			Fluoride Concentration of Drinking Water (ppm)		
Child Age	PPM				
From	To	Units	mg/day	mg/day	mg/day
0	6	months	0	0	0
6	36	months	0.25	0	0
3	6	years	0.5	0.25	0
6	1	years	1.0	0.5	0

For example, from the shaded box above, one can determine that the fluoride supplement suggested above for a 6-36 month-old child living in a community with public water fluoride concentration below 0.3 ppm, amounts to 0.25 mg per day.

Table II below illustrates the estimated tap water intake in milliliters per day (ml/d) for each of the age groups shown related to the fluoride intake (mg/d) for 1 ppm fluoride and for 0.7 ppm fluoride in the tap water.

Table II Estimated Tap Water and Fluoride Intake for Children

Daily F & H ₂ O Intake			Water Consumption		F Intake (1.0 ppm)		F Intake (0.7 ppm)	
Child Age			Mean*	90 pct*	Mean*	90 pct*	Mean*	90 pct*
From	To	Units	ml/day	ml/day	mg/day	mg/day	mg/day	mg/day
0	6	mos	?	?	?	?	?	?
6	36	mos	470	890	0.47	0.89	0.33	0.62
3	6	yrs	550	930	0.55	0.93	0.38	0.65
6	16	yrs	700	1160	0.70	1.16	0.49	0.81

- The "Mean" value is equivalent to the arithmetic average value. "90 pct" refers to the 90th percentile. In the above example, 10 per cent of the children in the 6-36 month-old group will ingest more than 0.89 mg of Fluoride per day. The hypothetical average child in this age range will ingest about 0.47 mg/day.

The shaded boxes in Table II under Water Consumption indicate **that the same 6-36 month old child** who drinks 470 ml/d or the upper 90th percentile child who drinks 890 ml/d will have received 0.47 and 0.89 mg/d of fluoride if the water is fluoridated at the level proposed for Natick. Clearly such a child **will have ingested between about 2-3.5 times as much fluoride as recommended by the American Academy of Pediatrics, American Dental Association and the American Academy of Family Physicians.**

Reference

- (1) American Academy of Pediatrics, Committee on Nutrition, Pediatrics, 95:777, (1995); Also endorsed by the American Dental Association and the American Academy of Family Physicians.

Appendix D The Measurement of Fluoridation Parameters

The following material is provided so that persons unfamiliar with the literature of the fluoridation issue can gain some insight into the methods used by workers in the field to express dental caries prevalence and reductions thereof. (1,2) The process used and the specification of the results obtained are described below. The second section of this appendix describes the method used to arrive at a measure when comparing the prevalence of dental fluorosis.

The Dental Caries Reduction Measurement

Measurements are taken by trained examiners using specified lighting and dental instruments to examine the teeth of subjects for evidence of:

- untreated decay ("D" or "d"),
- tooth lost due to decay ("M" or "m"),
- filling in place ("F" or "f").

Capital letters are used when the tooth is permanent and lower case is used when the tooth is deciduous. Most scores do not mix the two types of teeth in a common number. However, sometimes "d" appears without "m" because it is not always possible to know after the fact if a missing baby tooth was carious when it was lost. Initially, the scoring system used to relate an individual score was determined by the number of decayed, missing and filled teeth found, so "DMFT" (or "dmft") was the term used. Later on, counting the number of tooth surfaces implicated in a carious tooth became more popular, so "DMFS" (or "dmfs") appears more often in the literature after 1975.

For epidemiologically statistical purposes, the scores of all the individuals in a group are added and the sum is divided by the number of individuals to give a group score. Note however, that this says nothing about the distribution of poor teeth within the group as a whole. In addition, it has been widely recognized that fluoridation is responsible for the phenomenon of delayed dentition. Szilag has evaluated the "dental age" of a number of children and has reported that not only is there evidence of delayed dentition but that the greater the dosage of fluoride taken up during development, the greater was the retardation of dentition. (3) Opponents of fluoridation have pointed out that when due consideration is given to this fact, the reported DMFT and DMFS scores fail to reflect a significant difference between fluoridated and unfluoridated areas. For example, Coplan states:

"....When the basic scoring system was adopted, a small filling or decayed tooth area added one unit to a DMFT score with the same weight as a tooth seriously enough involved to have required extraction or major repair. Thus, a

badly decayed extracted molar added no more to a score than a small filling in a cuspid. Since this lack of discrimination could mask real effects, the practice was adopted of weighting decay severity by counting implicated surfaces, not just implicated teeth.

A molar extracted due to decay adds five units to a score, a small filling adds one unit, a two-surface filling on a cuspid adds two units, etc. This may leave some room for judgment calls but it definitely makes DMFS scores numerically larger than DMFT scores for the same actual caries status and has the numerical consequence that $DMFS = 1.6 \times DMFT$ as a general rule." (2)

As the incidence of caries has declined, however, and DMFS scores have fallen into the low single-digit region, the interpreter of group DMFS values should be cautioned to be wary of the potential for subtle distortions. For example, a 10% difference in group DMFS scores in the region of approximately 3.0 can mean any number of things, not the least of which may be due to slight variations in examining protocol. Possible sources of such differences may actually be due to variations in treatment schemes from time to time, dentist to dentist or location to location. (2)

As an example, decisions to extract a decayed first molar because it seems to be interfering with the eruption of other teeth, but which might have been saved by filling, or to decide whether a particular tooth gets a 3-surface crown or some less complicated repair will obviously have some impact on these scores. Waldbott (1) has commented on this variability as follows:

"...Another crucial factor to be considered in evaluating caries statistics is the variability and possible bias of the examiner. One investigation demonstrated, for example, that repeated examinations of the same tooth by the same examiner yielded widely varying caries scores from one examination to another.(58) In a different study, when each of the 33 patients was examined by three of eight different dentists, a deviation of 89% in the number of cavities was recorded.(59) In one case two of the dentists found 12 cavities, while the third found only five. In another case one dentist found 13 cavities, the second found six, and the third found only five. Overall, the average difference in assessment for the 33 patients was 4.2 carious teeth and 5.8 carious surfaces. With such large and glaring discrepancies, it is obvious that any conclusions based on differences of only two or three DMF teeth, as is often the case in fluoridation studies, has only marginal value at best. Realistically speaking, such conclusions are highly questionable...."

Socioeconomic factors may also play a decisive (albeit unapparent) role, especially in comparisons of areas wherein these factors may vary greatly, obviating otherwise identical factors. In such an area, how much does DMFS depend on diet, willingness to seek medical help, ability to pay, delay in getting proper, early and regular

professional attention? When one considers that close to half of the student population in current studies is caries-free, one is forced to recognize that "...the poor dentition resides in a smaller segment of the population, probably starts very early and benefits little if at all from fluoridated water." (2)

Nevertheless, with DMFS values as low as 2.3, what is the significance of a 10 % reduction in practical terms? In a school system with DMFS value of 2.8 averaged across 5-17 year-olds, the statistically average child who comes into the school system with no cavities will leave with a DMFS of 7.01 (fluoridated public water supply) vs. 8.59 (unfluoridated public water supply), a difference of only 1.5 treated surfaces over the 12-year school experience. (4) An 18% reduction in DMFS as far as the student is concerned is less than one cavity and therefore can hardly be called significant.

The Quantification of Dental Fluorosis

This measurement, also termed the Dental Fluorosis Index, was postulated and defined as a means to respond to the problem of determining the optimal fluoride concentration in a public water supply that would produce the soundest teeth without the disfigurement of mottling. The parameters for this metric are illustrated in Table I.

Table I Degrees of Dental Fluorosis (5,6)

Category	Description of Aberration	Weighting Factor
None (Normal)	Enamel smooth, glossy, pale creamy white translucency	0.0
Questionable	Slight aberrations from translucency with occasional white fleck or spots	0.5
Very Mild	Small, opaque, paper-white areas involving less than 25% of the surfaces of the two most affected teeth; may acquire brown stains in adulthood	1.0
Mild	More extensive dull white opacities involving less than 50% of the surfaces of the two most affected teeth; Brown staining often present	2.0
Moderate	All enamel surfaces affected; distinct brown staining frequent	3.0
Severe	Teeth show marked hypoplasia, attrition and pitting; brown or black staining widespread	4.0

According to Dean, the preferable community index should not exceed 0.4 and at 0.6 "...it begins to constitute a public health problem." Waldbott comments (5) on both the weighting factors used and the justification thereof by stating:

".....Although in theory such calculations are attractive, in reality they are misleading. The community index of dental fluorosis does not accurately represent the true state of mottling in a community. It gives the same weight to eight questionable (0.5) cases as to one severe case (4.0); it counts three mild (2.0) or six very mild (1.0) cases as equal to two moderate (3.0) ones For the individual with an unsightly degree of mottling, it is of no comfort to know that the community index of dental fluorosis is below 0.6 or even below 0.4! This dilemma was clearly perceived by Cox, who first explicitly advocated fluoridation, when he wrote: **'...With the threat of the Scylla and Charybdis of dental caries and mottled enamel, great caution must be observed in the means of administration of fluorides and in the control of such procedures as may be adopted'...**"

References

1. Waldbott, GL, *Fluoridation: The Great Dilemma*, Coronado Press, p. 189, (1978)
2. Coplan, MJ, *Fifty Years of Tooth Decay and Fluoridation*, (unpubl.), (1997)
3. 3) Szelag J, *Evaluation of the effects of various fluoride concentrations in drinking water and atmospheric air on permanent teeth eruption in children aged 12 years.*, Czas. Stomatol., **43**(3):154-159, (1990) (Abstract from Medline; article in Polish)
4. Brunelle, JA and Carlos, JP, *Recent trends in dental caries in US children and the effect of water fluoridation.*, J. Dent. Res., **69**:723-727, (1990)
5. Dean, HT, *Classification of Mottled Enamel Diagnosis*, J. Am. Dent. Assoc., **21**:1421, (1934)
6. Dean, HT, *Investigation of Physiological Effects by the Epidemiological Method*, in *Fluorine and Dental Health*, Moulton, F. R., Ed., p. 23, (1942)
7. Waldbott, GL, *op. cit.*, p. 179

Members of the Committee

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Jason Kupperschmidt, B. S.	17 Greenwood Road, Natick, MA
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*The committee wishes to acknowledge the participation of Dr. Benedict J. Gallo and to recognize his contributions during the course of this study. Although Dr. Gallo resigned from the committee during the final preparation of this report, he remained in complete agreement with the Findings, Conclusion and Recommendations of the Natick Fluoridation Study Committee.

Curricula Vitae of the Committee Members

The following material is being provided at the specific request of the Board of Selectmen of the town.

Curriculum Vitae of Benedict J. Gallo

BENEDICT J. GALLO

72 Washington Street
Natick, MA 01760

EDUCATION

- Ph.D., University of Michigan, Ann Arbor, Michigan, 1977.
Major, Botany; Minor, Chemistry.
- M.S., Eastern Michigan University, Ypsilanti, Michigan, 1970.
Major, Biology; Minor, Education.
- M.S., University of Michigan, Ann Arbor, Michigan, 1962.
Major, Geology; Minor, Zoology.
- B.A., University of Connecticut, Storrs, Connecticut, 1958.
Major, Geology; Minor, Zoology.

EMPLOYMENT

1979 – Retirement 1996

Research Microbiologist at the U.S. Army Research, Development and Engineering Center, Natick, Massachusetts 01760-5020. Conducted basic research involving the search and development of inexpensive sources of microbial enzymes for use in Chemical Defense and munitions biodegradation.

1976 – 1979

Research Microbiologist, National Research Council Research Associate at the U.S. Army Research, Development and Engineering Center, Natick, Massachusetts 01760- 5020. Conducted basic research in the bioconversion of ligno-cellulosic urban wastes and agricultural residues into the power fuel ethanol.

1970 – 1976

Teaching Fellow and Research Associate, University of Michigan, Ann Arbor, Michigan. I instructed degree candidates in Botany, Genetics and Microbiology and conducted independent research involving the genetics, biochemistry and microbiology of several microbial enzyme systems.

1960 – 1970

Instructor, Biology, Central Connecticut University, New Britain, CT;
Secondary School Science Teacher at St. John School, Jackson, Michigan and
Bentley Senior High School, Livonia Public School System, Livonia, Michigan,
Pharmaceutical Sales, Westerfield Labs, Cincinnati, OH

PUBLICATIONS

Journal papers, DOD reports and U.S. Patents.

AWARDS

1989 USANRDEC Silver Pin for Research,
1989 Soldier Sciences Directorate Outstanding Project Officer Award.

Curriculum Vitae of Jason Kupperschmidt

Jason Kupperschmidt

17 Greenwood Road
Natick, MA 01760

Education

University of Illinois, Urbana-Champaign, IL

B. S., Chemical Engineering, May 1992

Northeastern University, Boston, MA

M. S. Student, Environmental Engineering, September 1995 to Present

Professional Affiliations

Commonwealth of Massachusetts Engineer-in-Training, Certificate # 16409

American Institute of Chemical Engineers, Member

Experience

Armstrong Pharmaceuticals, Boston, MA

September 1994- Present

Process Engineer

- Enhance existing manufacturing processes through optimization of critical steps.
- Interact with the other department supervisors to ensure production under GMP guidelines.
- Oversee personnel training, preventive maintenance, and calibration programs.
- Implement process changes in chemical aspects of production.
- Maintain all support equipment including: refrigeration, pumps, and tanks.

General Chemical Corporation Framingham, MA

November 1993 - January 1995

Environmental Chemist

- Analyzed hazardous waste primarily for chlorinated solvents and PCB contamination.
- Evaluated whether chlorinated solvent waste can be reclaimed.

U.S. Army Natick Research, Development and Engineering Center, Natick, MA

May - August 1990, May - August 1991, July 1992 - February 1994

Biochemistry Research Assistant

- Conducted and analyzed biochemical experiments for food research applications.
- Designed diagnostic thermal processing devices to optimize heat exposure.
- Utilized analytical instrumentation and computer software in data analysis.

Dupont/Merck Pharmaceutical Company, Billerica, MA

March - August 1994

Contract Assignment

- Production Engineer Technician
- Regulated and disposed of radioactive waste.
- Generated the radiopharmaceuticals used as imaging agents.

Curriculum Vitae of Norman R. Mancuso**Norman R. Mancuso, B.S., M.S., Ph.D.**

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Dr. Norman R. Mancuso possesses a broad and diversified background in Engineering and the Sciences. Trained and educated at such institutions as St. Bonaventure University, State University of New York at Buffalo and the Massachusetts Institute of Technology, he has over thirty years of in-depth, hands-on experience in a wide range of Chemical, Engineering and Computer related projects. He is the author or co-author of over one hundred Chemical, Scientific and Engineering publications and has extensively served both the domestic and European high-tech communities.

Educational Background

Postdoctoral Fellow
Massachusetts Institute of Technology
Ph.D., Chemistry, SUNY at Buffalo, Buffalo, NY.
M.S., Chemistry, St. Bonaventure University, Olean, NY.
B.S., Chemistry/Mathematics, St. Bonaventure Univ. Olean, NY.

Academic and Industrial Honors

Dupont Teaching Fellow
National Institutes of Health Postdoctoral Fellow
Product Innovation Award - Dennison Mfg. Co., Inc.

Academic and Industrial Positions

NORMAN R. MANCUSO ASSOCIATES, Framingham, MA, Consulting Engineer
AVERY DENNISON, INC., Imaging Systems Division, Hopkinton, MA, Group Leader
DENNISON MFG. CO., INC., Corporate R&D, Framingham, MA, Senior Engineer
INFOREX, INC., Advanced Development Group, Burlington, MA, Senior Engineer
MASSACHUSETTS INSTITUTE OF TECHNOLOGY, Dept. of Chem., Cambridge, MA

- Postdoctoral Fellow and Research Staff Member
- Director of the Computer Facility at the NIH Mass Spectrometry Center

NORTHEASTERN UNIVERSITY, Boston, MA, Lecturer/SOA Engineering Program

Dr. Mancuso pioneered the use of high-integration embedded microprocessors, high density programmable logic and field programmable gate arrays at Dennison and introduced various CAE tools into the Dennison R & D environment.. He also planned, implemented, and directed an Automatic Test facility for PCB testing. He received a Corporate Productivity Award for the design and development of high integration embedded microprocessor PCB's used in several product lines. He also developed various product/process specifications, including technical documentation for a number of engineering companies. A strong proponent of continuing education, he was instrumental in arranging and administering employee Professional Development courses and other programs increasing technical employee involvement, productivity and morale.

As an Apollo Program Project Scientist, Dr. Mancuso was responsible for the development of an interlock system enabling the organic analysis of lunar samples while maintaining and protecting the integrity of the terrestrial biosphere. Other analytical instruments developed include a laser-based web flaw detector for the Dunn Paper Co. as well as Comparator/Spectrophotometers and real-time data acquisition systems for the measurement of Mass Spectrometric photographic plates. While serving as a consultant to Karolinska Institute (Stockholm) he developed a real-time data acquisition systems incorporating multi-channel non-coincidence amplifier systems.

Dr. Mancuso is a member of the Metrowest Chamber of Commerce, the Institute of Electrical & Electronic Engineers, the Committee of Concerned Engineers and the American Chemical Society. He also served on the Natick Underground Storage Tank Removal Committee.

Curriculum Vitae of Alfred J. Murray

Alfred J. Murray
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Education:

- Bridgewater State College, 1960. B.S.
- Major; Mathematics and Chemistry
- Colby College, 1973. M.S.T.
- Major; Chemistry

Massachusetts Teaching Certification:

- Secondary school principal, mathematics, chemistry and general science

Work Experience:

- 1991-1997 Dean College. Instructor of Chemistry
- 1963-1997 Natick High School, Teacher and Dept. Chairman
- 1990,91,94 U.S. Army Natick Labs. Research
- 1966-1971 Framingham Union Hospital, Lab Technician
- 1962-1964 Lawrence General Hospital, Lab Technician
- 1963-1964 Longwood Hospital, Lab Technician
- 1960-1962 United States Army, Clinical Lab Technician

Publication:

Mental Deficiency, *Dwarfism and Decreased Segmentation of Neutrophilic Leucocytes*; Journal of Mental Deficiency Research, 11(4) December 1967

Awards:

- U.S. Army Special Act Award (as a food chemist)
- Edison Citation for Distinguished Service
- Certificate of Honor, Westinghouse Science Search
- Tandy Outstanding Educator Award (1993 & 1994)

Curriculum Vitae of Harlee S. Strauss

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Dr. Strauss is the President of H. Strauss Associates, Inc. (HSAI), a consulting firm she founded in 1988. Dr. Strauss works on a broad range of projects, from site specific human health risk assessments, to in-depth evaluations of the toxicity of individual chemicals, to the development of risk assessment methodology. She has conducted projects related to identifying gender biases in risk assessment, how to apply risk assessment methodology to childhood cancer, and how to establish risk assessment frameworks with respect to microorganisms, including bioremediation. Dr. Strauss initiated and, for its first year, lead a multi-million dollar study to investigate the potential links between the environment and breast cancer on Cape Cod, Massachusetts.

Dr. Strauss has been a member of the Society for Risk Analysis since 1987. She served on the Management Committee for the residential exposure assessment project and on the Advisory Committee for SRA Workshop "Key Issues in Carcinogen Risk Assessment Guidelines." Dr. Strauss is a long time member and former president of the New England Chapter of SRA. She initiated the SRA-NE monthly newsletter, "Back of the Envelop" and was its editor for several years. Dr. Strauss received an Outstanding Service to Society award from the SRA in 1996.

Dr. Strauss's other activities include serving her second, two-year term on the Army Science Board (ASB). She was a member of the ASB work group on Management and Abatement of Lead Based Paint at Army Sites and is currently a member of two ASB study panels: 1) Evaluation of the Effectiveness of Existing Groundwater and Soil Treatment Systems in the US Army and 2) a study related to Chemical/biological Weapons Defense.

Harlee Strauss earned a Ph.D. in molecular biology from the University of Wisconsin-Madison (1979) and an A.B. in chemistry from Smith College (1972). She was a postdoctoral fellow in biology at MIT (1979-81), sponsored by the NIEHS) and a Congressional Science Fellow sponsored by the Biophysical Society (1981-83). Dr. Strauss has also held the positions of special assistant for government affairs at the American Chemical Society (1983-84), special consultant at ENVIRON Corporation (1984), research associate at the MIT Center for Technology, Policy and Industrial

Development (1985-86), senior associate at Gradient Corporation (1986-88), and executive director of Silent Spring Institute (1994-95).

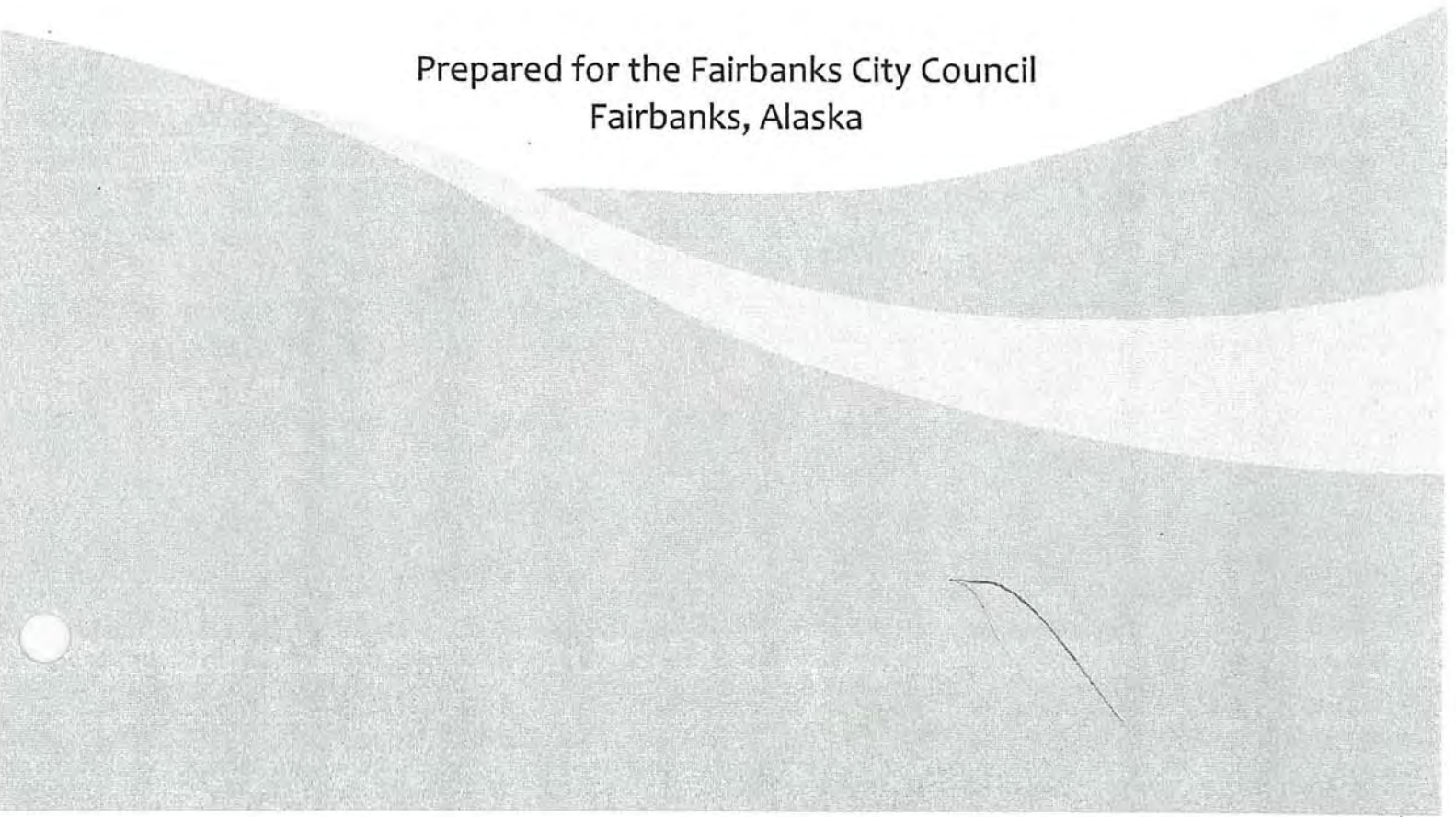
Errata

<u>Page</u>	<u>Correction</u>
8	"...Dosage of Fluoride..."
11	"...Selectmen with regard..."
12	"...normal usage, direct..."
	"...this usage...."
12, 16	"...profluoridation..."
14, 15	"...antifluoridationists..."
15, 16	"...profluoridationists..."
	"...a.k.a...."
16	"...desirability..."
18	"...pre-eruptive..."
18	"...e. g., lifestyle...."
18	"...desirable...."
24	"...dysfunction...."
	"...insecticides...."
	"...minimum...."
25	"...independent..."
27	"...mgs of fluoride per kg of..."
	"...nationwide..."
29	"...Agency's...."
33	"...postmenopausal...." (2x)
36	"...carcinogenicity...."
43	"...enzymes (17)...."
48	"...triethyltin-induced...."
52	"...than 110...."
53	"...prenatally..."
	"...observed...."
	"...findings are...."
56	"...epidemiological study..."
60	"...negligible...."
	"...occurred in 1992...."
61	"...sodium fluorosilicate are...."
76	Definitions corrected and clarified.
82	"...MA"
89	"...murray@meol.mass.edu"
	"...Army...."

Report of the Fairbanks Fluoride Task Force

April 25, 2011

Prepared for the Fairbanks City Council
Fairbanks, Alaska



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Chapter 1

Introduction

In response to concerns expressed by community members, on February 8, 2010, the Fairbanks City Council passed a resolution (Appendix A) establishing a committee charged with the responsibility to examine evidence related to fluoridation of public water supplies and to provide the City Council with a report containing analysis and recommendations. The committee was to obtain documentation provided by both proponents and opponents of fluoridation and to supplement this documentation with information from other appropriate sources. The committee was to make its final report to the City Council by early July, 2010, but the committee was unable to meet this deadline due to the complexity of the assignment and the schedules of the committee members.

The committee, referred to in this report as the Fairbanks Fluoride Task Force (FFTF), is composed of the following members:

Paul Reichardt, Ph.D. (Chair)
Professor of Chemistry Emeritus
University of Alaska Fairbanks

Bryce Taylor, D.D.S.
Dentist
Fairbanks

Richard Stolzberg, Ph.D.
Professor of Chemistry Emeritus
University of Alaska Fairbanks

Joan Braddock, Ph.D.
Professor of Microbiology Emeritus
University of Alaska Fairbanks

Rainer Newberry, Ph.D.
Professor of Geochemistry
University of Alaska Fairbanks

Beth Medford, M.D.
Tanana Valley Clinic
Fairbanks

The FFTF met for the first time on March 4, 2010, and continued to hold public meetings approximately twice a month through March 8, 2011. At the invitation of the FFTF, both proponents and opponents of fluoridation of the Fairbanks water system (operated by Golden Heart Utilities) made presentations at the March 16, 2010, meeting. Public testimony was received at each of the ten public meetings during the period March 16, 2010, through June 22, 2010. Numerous comments and pieces of information were submitted to the FFTF electronically. Members of the FFTF supplemented this information with relevant articles from the professional literature and results of personal interviews and research.

All documents and information received by the FFTF during the period in which public testimony was being accepted are cited on the References section of the FFTF website (www.ci.fairbanks.ak.us/boardscommissions/fluoridetaskforce/fluoridetaskforcereferencematerials.php). While FFTF members considered the entire body of information submitted and collected, only some of the materials listed on the References website were used in preparing this report. Those materials are listed as references at the end of this report. There is a massive amount of relevant information on this topic. For example,

in 2008, C. A. Yeung did a review of the efficacy and safety of fluoridation that began with over 5,000 relevant citations. The approach the FFTF took to assessing and using this information was to rely on reviews and studies published between 2000 and 2008 to assess the evidence for and against fluoridation of drinking water as it existed up to 2008 and to supplement this body of literature with key professional articles published in the last several years.

Although the FFTF examined all aspects of water fluoridation, it focused most of its review of the literature on exposure of individuals to fluoride, the efficacy of fluoridated water in caries prevention, and the risks associated with consumption of fluoride. While the task force's major concerns were about populations exposed to 0.7 to 1.2 parts per million (ppm) fluoride in their water supplies, it did examine and consider evidence related to populations receiving both higher and lower concentrations of fluoride in their drinking water. The FFTF's review and analysis of relevant information was organized around the topics that became the chapters of this report. After a series of discussions and work sessions in which all members voiced their observations and concerns about each of the topics, assignments were made to individual task force members for lead responsibility in producing an initial draft of each chapter. The entire task force was subsequently engaged in the process of chapter revision that led to a draft report, which went out for public review and comment. After consideration of comments submitted electronically as well as at two public hearings (March 29 and 31, 2011), the task force made corrections and edits at its meeting on April 5, 2011. The subsequent final report (including recommendations) will be submitted to the City Council.

Some technical terms and abbreviations are used throughout this report. In an attempt to make the report more readable for the general public, a few key definitions are given below:

concentration: the relative content of a component, often expressed as amount in a given volume (e.g., ppm)

DMFS: decayed, missing, and filled surfaces in permanent teeth

DMFT: decayed, missing, and filled permanent teeth

dmft: decayed, missing, and filled deciduous (baby) teeth

dose: measured quantity of an agent to be taken at one time

g (gram): 0.001 kg

kg (kilogram): a basic unit of mass and weight equal to 2.2 pounds

mg (milligram): 0.001 g

L (liter): a basic unit of volume equal to about a quart

LD₅₀ (lethal dose, 50%): dose of a toxin required to kill 50% of a group of test organisms

ppm (parts per million): a unit of concentration, defined for this report as one mg/L

Chapter 2

Recommendations

The Fairbanks Fluoride Task Force makes a set of four recommendations. We anticipate that the community's focus will be on Recommendation #1, but as a committee we feel strongly that Recommendations 2, 3, and 4 should be implemented along with Recommendation #1 as part of a cohesive plan to address dental health issues in our community.

1. Primarily because (1) the ground water used for Fairbanks public water contains an average of 0.3 ppm fluoride, and (2) higher concentrations of fluoride put non-nursing infants at risk, the task force recommends that supplemental fluoridation of the Fairbanks public water supply be terminated. The task force further recommends that the Fairbanks community be informed of possible dental health implications from not fluoridating the water.

Rationale: Not fluoridating Fairbanks water will reduce the fluoride content from 0.7 ppm to 0.3 ppm, which is the fluoride concentration of the raw water used by Golden Heart Utilities (GHU). This will reduce, but not eliminate, the risk of significant incidence and severity of fluorosis, especially fluorosis associated with the use of GHU water to prepare infant formula. Doing so will also address ethical concerns raised during the task force's public testimony. However, the effect of this reduction in fluoride concentration on the caries rate in the Fairbanks community, while most likely small, is unknown and unpredictable. Those who depend on 0.7 ppm fluoride in tap water for their dental health need to be informed of the possible adverse consequences to their dental health caused by reducing the fluoride content of Fairbanks tap water from 0.7 ppm to 0.3 ppm and of the measures that can be taken to address these possible adverse consequences.

The task force has made this recommendation to terminate fluoridation of GHU water with full knowledge of and respect for the positions of the American Dental Association (ADA), the Centers for Disease Control and Prevention (CDC; part of the U.S. Department of Health and Human Services), the World Health Organization, and the Alaska Department of Public Health in support of fluoridation of public water supplies. While the task force members agree that water fluoridation may be an important element of an effective dental health program in many communities, the majority of members are not convinced that it is necessary in Fairbanks because of the fluoride content of the city's ground water and the alternate sources of fluoride available in the community. Five task force members, with various degrees of conviction, support this recommendation, while one member (Dr. Taylor) supports continuing fluoridation at 0.7 ppm.

2. The Fairbanks City Council's decision-making process on fluoridation should involve representatives of the Fairbanks North Star Borough government.

Rationale: At least 25% of area residents who receive GHU water reside outside the city limits.

3. Local dentists and physicians should be encouraged to provide their patients with up-to-date information on the benefits and risks associated with fluoride.

Rationale: If nothing else, the recent notice that the secretary of the U.S. Department of Health and Human Services has proposed a new recommendation on fluoridation of public water supplies

indicates that the citizenry should be informed about the state of contemporary research findings and analysis related to the role of fluoride in dental health. All of the members of the task force went into this project with incomplete and in some cases incorrect information about the issue. We suspect that we are not unique in that respect.

4. The Fairbanks City Council should encourage the local school system to review and modify, as appropriate, its approach to promoting good dental health practices.

Rationale: The local schools have an excellent opportunity to help all families in the community to learn about and to implement good dental health practices, which can include optional opportunities at school for topical fluoride treatment (in the form of rinses and tooth brushing, for example) as well as techniques for minimizing unnecessary and/or unwanted exposure to fluoride.

Chapter 3

History of Fluoridation of Public Water Supplies

Fairbanks

A version of Fairbanks City Code dated July 1, 1959, contained a section (Article III, Section 10.301) that authorized and directed the Municipal Utilities System to develop and implement a fluoridation plan that fulfilled the requirements of the Alaska Department of Health. A slightly rewritten version of Article III, Section 10.301 of the City Code was adopted on January 12, 1960, and on August 21, 1962, the mandated fluoridation of city water was implemented in the city of Fairbanks. In 1996, the city water plant was sold by the Municipal Utilities System to Golden Heart Utilities (GHU). The fluoridation program continued under the auspices of GHU, and in 1999 the rewritten Fairbanks General Code (FGC 82-1) continued the mandate for fluoridation under the administration of Golden Heart Utilities. The present version of the Fairbanks City Code retains the language of Section 82-1 as it existed in 1999.

The only formal attempts to discontinue the fluoridation program took place in 2008. On February 25 of that year a proposed ordinance to prohibit the addition of fluoride to the GHU water supply failed in a vote of the City Council. In July 2008, a city resident submitted an application for an initiative proposing that FGC 82-1 be repealed and reenacted to read:

Fluoride should not be added to City community water systems. Water utilities that own or operate community water distribution systems in the City shall not add fluoride, in any form, to the water system. All water utilities owning or operating community water systems in the City shall conduct periodic water quality testing.

The required signatures were not submitted by the deadline of August 12, so the initiative did not go on the October ballot. The city took no additional action on the fluoridation issue until February 8, 2010, when the City Council passed Resolution No. 4398, establishing a task force to research issues related to the fluoridation of the municipal water supply.

United States

In the early 1900s, research, largely by dentist Frederick McKay and Dr. G. V. Black of the Northwestern University Dental School, documented that many residents in several areas of the western U.S. had mottled teeth and, in severe cases, brown stains ("Colorado brown stain") on their permanent teeth. McKay also noticed that the mottled teeth were resistant to decay. By the 1930s it had been determined that these conditions (today known as fluorosis) were caused by high concentrations of fluoride (ca. 4–14 ppm) in drinking water. In the ensuing years, Dr. H. Trendley Dean conducted a series of epidemiological studies and reported that (1) fluoride concentrations of up to 1.0 ppm in drinking water did not cause the more severe forms of dental fluorosis and (2) a correlation existed between fluoride levels in drinking water and reduced incidence of dental decay

(Dean et al., 1941). Dean's work led Dr. Gerald Cox and associates to publish in 1939 the first paper in which fluoridation of public water supplies was proposed (Cox et al., 1939).

In the 1940s, four classic, community-wide studies were carried out to evaluate the addition of sodium fluoride as a caries-reduction strategy in Grand Rapids, MI; Newburgh, NY; Brantford, Ontario; and Evanston, IL. Based on the overwhelmingly positive evaluations of these pilot studies by scientists and dental professionals, water fluoridation programs were instituted in a number of large U.S. cities in the following two decades. In addition, alternative methods of administering fluoride to combat caries were developed, the most notable being the introduction of fluoridated toothpaste in 1955.

However, as water fluoridation programs spread, so did opposition to the practice. In 1965, the first lawsuit in the U.S. contesting the legality of fluoridation of public water supplies was settled by the New York State Supreme Court, which denied the plaintiff's case "at least until some proof is advanced that fluoridation has harmful side effects" (Graham and Morin, 1992, p. 215). In the ensuing years a number of lawsuits contesting fluoridation of public water supplies have been pursued, but in no case have the plaintiffs been successful in stopping the practice (see Legal/Ethical Issues, chapter 4).

The relevant federal, state, and professional organizations have endorsed and promoted the fluoridation of public water supplies for the past fifty years. As a result, in 2008, forty-six of the country's fifty largest cities provided fluoridated water, and approximately 60% of the U.S. population consumed fluoridated water (Fagin, 2008). The U.S. Public Health Service (USPHS) has set a goal of "at least 75% of the U.S. population served by community water systems should be receiving the benefits of optimally fluoridated water by the year 2010" (U.S. Department of Health and Human Services [HHS], 2000, p. 205). However, the actions of communities on this front are mixed. One summary (Juneau Fluoride Study Commission, 2006) indicates that from 1998 to 2005 approximately two hundred communities in the U.S. moved to fluoridated water or decided to retain it while approximately one hundred chose to discontinue the practice. The situation in Alaska, where the fluoridation of public water systems is encouraged by the Alaska Department of Public Health (www.hss.state.ak.us/dph/targets/ha2010/PDFs/13_Oral_Health.pdf), roughly mirrors the national picture. In 2006, 64% of the Alaska population received fluoridated water, up from 47% in 1993 (Whistler, 2007). However, today's statewide figure may be below that of 2006 because Juneau discontinued its fluoridation program in January 2007.

International

According to the British Fluoridation Society (British Fluoridation Society, 2010), over 400 million people in sixty countries were served by fluoridated public water supplies in 2004. Countries and geographic regions with extensive water fluoridation programs include the U.S., Australia, Brazil, Canada, Chile, Columbia, Ireland, Israel, Malaysia, New Zealand, Hong Kong, Singapore, Spain, and the United Kingdom. However, especially during the period of 1970 to 1993, Japan and a number of European Countries (Federal Republic of Germany, Sweden, Netherlands, Czechoslovakia, German Democratic Republic, USSR, and Finland) discontinued water fluoridation programs. In 2003, Basel, Switzerland, ended its water fluoridation program, and in 2004 Scotland rejected plans to fluoridate water supplies.

In most or all of these situations, dental health continued to improve following cessation of water fluoridation (Ziegelbecker, 1998), presumably due to factors including enhanced dental hygiene programs, fluoride-containing table salt, fluoridated toothpaste, and improved diets. There are data to support the contention that in recent years caries rates in many areas have declined irrespective of the concentrations of fluoride in water supplies. World Health Organization (WHO) data (Peterson, 2003: Fig. 7) indicate substantial declines in DMFT among twelve-year-olds in developed countries (from about 4.7 to about 2.5) during the period 1980 to 1998 but little change among this age group in developing countries (from about 1.8 to about 2.3). Nevertheless, the World Health Organization continues to consider community water fluoridation to be an effective method to prevent dental caries in adults and children. However, it recognizes that other approaches, including fluoridated salt and milk fluoridation, have “similar effects” (www.who.int/oral_health/strategies/cont/en/index.html). It also recognizes the value of fluoridated toothpaste and fluoride-containing mouth rinses and gels.

For Alaska communities, perhaps the most relevant international situation is that in the neighboring country of Canada. According to the Health Canada website (www.hc-sc.gc.ca), each Canadian municipality retains the authority to decide on fluoridation of its water supply; in 2005, 43% of the Canadian population was served by fluoridated water supplies (Federal-Provincial-Territorial Committee on Drinking Water, 2009). The Guidelines for Canadian Drinking Water Quality set a maximum allowable fluoride concentration of 1.5 ppm in drinking water, a level at which Health Canada believes there are no undue health risks (Health Canada, 2010). Although Canadian provincial and territorial governments regulate the quality of drinking water in their jurisdictions, Health Canada has recommended to communities wishing to fluoridate their water supplies that “the optimal concentration of fluoride in drinking water to promote dental health has been determined to be 0.7 mg/L” (Health Canada, 2010).

The Controversy

From the very beginning of efforts to implement water fluoridation programs in 1945, there has been controversy (Connett et al., 2010). By the 1950s the sides were pretty well drawn. On one side were dentists and scientists from government and industry, who promoted the addition of fluoride to drinking water as a protection against dental decay. On the other side were mostly activists who contended that water fluoridation was essentially compulsory mass medication, thus a violation of individual rights, and that the risks of fluoridation had not been studied adequately. The advocates of fluoridation won the argument, in part by ridiculing the unlikely arguments of some of the opponents (e.g., the John Birch Society, which contended that fluoridation was a communist plot to poison the citizens of the USA).

A series of court cases from the mid-1960s through the mid-1980s established that local and state governments have the constitutional authority to implement fluoridation programs. These decisions were based largely on the principle that the “government interest in the health and welfare of the public generally overrides individual objections to health regulation” (American Dental Association [ADA], 2005, pp. 47–49). In light of these decisions, the argument against “compulsory mass medication” has emphasized ethical rather than legal issues (see, for example, Bryson, 2004).

During this same period, a number of scientific investigations into potential adverse effects of drinking fluoridated water were undertaken. None of these studies produced results that were generally accepted as demonstrating serious adverse health effects of water containing “optimal levels” of fluoride ion (0.7 to 1.2 ppm). However, a number of them raised significant questions about potential risks by showing some adverse health effects at fluoride concentrations of greater than 2 ppm (for example, Kurttio et al., 1999; Freni, 1994).

Around the turn of the century, a comprehensive review of the scientific literature related to water fluoridation was undertaken under the auspices of York University in the United Kingdom. The report from this review (McDonagh et al., 2000), often referred to as the *York Report*, noted the generally poor quality of the evidence for both beneficial and adverse effects of fluoridation. The resulting uncertainties about the benefits and risks of consuming fluoridated water fueled the controversy in that it allowed each side to discount the opposition’s arguments because of the “poor quality” of the evidence on which positions were based. While there are many examples of the arguments put forward by the two sides, two representative accounts are an anti-fluoridation article by Colquhoun (1998) and a pro-fluoridation article by Armfield (2007).

Another key review of the effects of fluoride in drinking water was published by the U.S. National Academy of Sciences in 2006 (National Research Council, 2006). This review and associated recommendations were focused on EPA standards for drinking water (Maximum Contaminant Level, MCL, of 4 ppm and Secondary Maximum Contaminant Level, SMCL, of 2 ppm) and did not directly address the USPHS regulations on the lower concentrations in fluoridated public water supplies in the U.S. (0.7 to 1.2 ppm). Nevertheless, the report contains information and data relevant to the safety of fluoridated water. Evidence in the scientific literature led the review committee to conclude that water containing 4 ppm fluoride “puts children at risk for developing severe enamel fluorosis” and was “not likely to be protective against bone fracture” (National Research Council, 2006, p. 2). This review also contains analyses of a number of other adverse health effects that have been alleged to be related to fluoride ingestion, but the authors found that these allegations were either not supported by good evidence or required further study before any meaningful conclusions could be drawn. As with the *York Report*, the uncertainties about the risks of fluoride-containing water (compounded, in this case, by uncertainties about how conclusions based on consideration of fluoride concentrations of 2 ppm or higher relate to lower concentrations) have given both advocates and opponents of fluoridation data and arguments that they have selectively employed in supporting their opposing positions.

As time has gone on, particularly since the publication of the *York* and National Research Council reports, a number of professionals with expertise in dental health and toxicology have joined the opposition to fluoridation. They include dental researchers who were originally supporters of fluoridation (e.g., Colquhoun, 1998; Limeback, 2000), dentists (e.g., Osmunson, 2010a), and EPA employees (e.g., Thiessen, 2006, 2009a, 2009b, 2010; Hirzy, 2000). A “Professionals’ Statement to End Fluoridation” (www.fluoridealert.org/prof_statement.pdf) had over three thousand signers as of July 2010 (although many of the signers are not identified with respect to their areas of expertise, so it is not clear that all these “professionals” have expertise in relevant areas). However, professional and governmental organizations remain supportive of water fluoridation, and to our knowledge, the majority of dental health practitioners in the United States continue to support it.

There is no shortage of information; the literature search for a recent review of the efficacy and safety of fluoridation turned up over five thousand citations. However, after application of exclusion/inclusion criteria related to the quality of the research and after review of the full text of each remaining article, the author of the review selected just seventy-seven citations for inclusion (Yeung, 2008). Why has so much of the fluoridation literature been deemed to be of less than high quality? There are at least four difficulties inherent in these studies:

1. as with all epidemiological studies, those focused on the safety and efficacy of water fluoridation are complicated by a multitude of confounding variables (e.g., Taubes, 2006), not the least of which is the tremendous variability in water consumption and related fluoride dose of individuals (EPA, 2004);
2. in many cases the data cannot be interpreted without the application of sophisticated statistical methods, and even then statistical correlations do not necessarily imply causative relationships (e.g., Sigfried, 2010);
3. some of the alleged adverse effects of fluoride are associated with very rare conditions (e.g., osteosarcoma), making it difficult to detect small, but potentially significant, differences in study populations;
4. the results from studies with laboratory animals are often not complicated by confounding variables, but their relevance to humans and the concentrations of fluoride in public water supplies is often difficult to determine (Hayes, 2008, pp. 330–332).

In recent years, the difficulties associated with critical evaluation of research findings and associated conclusions have been exacerbated by the widespread use of the internet as a medium for distributing information and opinions. The opponents of fluoridation in particular have used the internet to advance their arguments and point of view. Although many of these sites contain useful information and cogent arguments, the sites and the information on them are not uniformly of high quality. In many instances it is difficult to evaluate the quality of material posted on websites focused on fluoride and fluoridation without a fairly thorough knowledge of the peer-reviewed literature.

While these scientific issues continue to be debated, it appears that within the general public the major concern is related to ethics, not quality of the research on benefits and adverse effects of water fluoridation. Thus, many opponents of water fluoridation would remain opposed to “mass medication” even if the safety and efficacy of the practice were clearly documented. So, today the controversy continues unabated. The situation is described quite well in a recent journal article:

Plans to add fluoride to water supplies are often contentious. Controversy relates to potential benefits of fluoridation, difficulty in identifying harms, whether fluoride is a medicine, and the ethics of a mass intervention. We are concerned that the polarised debates and the way that evidence is harnessed and uncertainties glossed over make it hard for the public and professionals to participate in consultations on an informed basis. (Cheng et al., 2007, p. 699)

Findings

Throughout the United States, and in many countries around the world, the incidence of tooth decay has decreased significantly over the past several decades. Although claims have been made that adding fluoride to drinking water has been one of the main reasons for this decline, the data indicate that in many countries and communities progress in preventing caries has been made without fluoridated water.

For many years professional organizations and federal, state (including Alaska), and local governments in the United States have promoted the fluoridation of public water supplies, and these organizations and relevant government agencies still strongly support the practice. However, there has also been opposition to the practice since its inception in the 1940s. Although it appears that most dental practitioners and researchers still support fluoridation of municipal water supplies, it also seems that the number of practitioners and researchers who oppose the practice has increased. At this time the claims most often cited by opponents of fluoridation of water supplies are:

- lack of definitive evidence for efficacy,
- evidence indicating risk of adverse effects, and
- ethical issues related to mass medication.

Chapter 4

Legal and Ethical Issues

As indicated by testimony to the Fairbanks Fluoride Task Force, legal and ethical issues are perhaps the biggest concerns of the local residents who are opposed to fluoridation of Fairbanks' public water supply. The testimony received by the task force was overwhelmingly against fluoridation. During the ten task force meetings at which public testimony was invited, sixty-two testimonies were presented by thirty individuals (at the extremes eighteen individuals presented testimony just once, and one individual submitted testimony on six different occasions). The positions of the testifying individuals, as described by themselves or ascertained by the task force from the nature of the testimonies, were twenty-six against fluoridation, three in favor, and one with no clearly stated opinion. The major concerns voiced by the opponents of fluoridation were:

1. toxic and harmful effects of fluoride;
2. lack of high-quality evidence that fluoride in public water supplies effectively prevents dental caries;
3. unethical aspects of "mass medication," including lack of informed consent;
4. fluoridation of public water supplies interferes with freedom of choice, infringes on individual rights, and results from an overreach of governmental powers; and
5. the risk that fluoridation of public water supplies may do more harm than good.

While testimony and evidence on all five of these concerns were presented to the task force, concerns 3, 4, and 5 were highlighted for the task force by both the frequency and passion of testimonies related to them. They have also been voiced in the larger debate over water fluoridation. The "mass medication" argument is that fluoridation of public water supplies administers medication to an unaware and in some cases, unwilling public (see, for example, www.fluoridedebate.com/question34.html; Cross and Carton, 2003). The "individual rights" concern (#4) is related to the previous concern in that it questions governmental authority to implement the "mass medication" (Cross and Carton, 2003). The concern that water fluoridation may do more harm than good brings into the argument the "first, do no harm" precept of medical ethics. This precept basically says that in a given situation it may be better to do nothing if the action to be taken may cause more harm than good.

The legal concerns brought to the task force were considered in light of a rather lengthy history of legal challenges to fluoridation of public water supplies (Graham and Morin, 1999). Although fluoridation has been challenged numerous times in at least thirteen states, and while cases decided primarily on procedural grounds have been won and lost by both proponents of and opponents to fluoridation, no final ruling in any of these cases has stopped a proposed fluoridation program or ruled in favor of elimination of an existing program (Block, 1986; ADA, 2005; Pratt et al., 2002). In the process, the U.S. Supreme Court has declined to review fluoridation cases at least thirteen times (ADA, 2005).

In contrast to the legal question, which has repeatedly been addressed by the courts, the ethical issues remain problematic. On the one hand, opponents of fluoridation cite concerns about the propriety of forced "mass medication" and the integrity of at least some of the individuals and organizations that promote the practice (see, for example, Bryson, 2004; Cheng et al., 2007; Connett et al., 2010). On

the other hand, some proponents have argued that those who potentially have the most to gain from fluoridation of public water supplies—the economically and educationally disadvantaged and those with limited access to proper health care—do not have a voice in the development of health policies and practices unless those in power are looking out for their interests (McNally and Downie, 2000). Cohen and Locker (2001), observe that the conflict between beneficence of water fluoridation and autonomy remains unresolved and that “there appears to be no escape from this conflict of values, which would exist even if water fluoridation involved benefits and no risks” (p. 578). Further, they argue that although recent studies indicate that water fluoridation continues to be beneficial, critical analysis indicates that the quality of evidence provided by these studies is generally poor. Thus, they argue that from an ethical standpoint, past benefits of fluoridation cannot be used to justify continuation of the practice, and they call for new guidelines that “are based on sound, up-to-date science and sound ethics” (p. 579).

Chapter 5

Exposure

Fluorine, which exists in its elemental form as fluorine gas, is one of the most reactive elements. Its chemical reactivity is characterized by its propensity to accept electrons and to undergo reduction to the fluoride ion. While elemental fluorine is found in just one form, the fluoride ion exists in a number of compounds, including the common minerals fluorite and especially fluorapatite. Fluorine is also found in a group of compounds called “organic fluorides,” compounds in which fluorine is chemically bonded to carbon. Some pharmaceuticals, consumer products, and pesticides are organic fluorides.

Concerns about the safety and efficacy of artificially fluoridated water revolve around one species, the fluoride ion—often referred to in this report as fluoride. Fluoride is easily absorbed in the human alimentary tract, is distributed to most—if not all—tissues, and is cleared from the blood and tissues by uptake into bone and by excretion (Whitford, 1996; National Research Council, 2006). It is capable of inhibiting certain enzymes (Scott, 1983, p. 166; National Research Council, 2006) and of affecting bacterial metabolism, including reducing the capability of plaque-forming bacteria to produce acid (Featherstone, 2000; Jones et al., 2005), which is the bacterial product responsible for caries. Given that fluoride has these biochemical properties, it is not surprising to find that it is toxic. The acute toxic dose of fluoride is 5 to 10 grams for a 155-pound person (Hodge and Smith, 1965; ADA, 2005). More precise determinations of toxicity have been performed with pure chemicals and laboratory rats, and these studies indicate, for example, that sodium fluoride is about ten times less toxic than sodium cyanide and about fifty times more toxic than sodium chloride (table salt).

The fluoride-containing compound of most interest in the Fairbanks situation is sodium fluorosilicate, the compound that Golden Heart Utilities (GHU) uses to fluoridate the water it distributes. Sodium fluorosilicate is toxic; for rats its LD50 is 125 mg/kg (that is when laboratory rats were given single doses of 125 mg of sodium fluorosilicate per kg of body weight, 50% of the test animals died). According to the National Institute of Health’s TOXNET website (<http://toxnet.nlm.nih.gov/cgi-bin/sis/search/a?dbs+hsdb:@term+@DOCNO+770>), the acute toxic dose of sodium fluorosilicate for a human is between 3.5 and 35 grams. However, the low concentration of this compound in treated water (around 1.5 mg per liter) ensures that there is no acute toxicity threat associated with the treated GHU water. Nevertheless, concerns have been voiced about risks related to the use of sodium fluorosilicate in water fluoridation programs. In particular, a correlation was reported between use of sodium fluorosilicate to fluoridate water in various locales in the state of New York and levels of lead in the blood of children residing in these communities (Masters and Coplin, 1999; Masters et al., 2000). However, this correlation was not verified in a subsequent study (Macek et al., 2006). Furthermore, a causative link between the use of sodium fluorosilicate and elevated lead levels in blood of children who consume the fluoridated water would require that sodium fluorosilicate incompletely dissociates when it dissolves in water, a proposition put forward by Westendorf (1975) but which is inconsistent with the best contemporary evidence (Urbansky, 2002).

Because fluoride is found in a number of common minerals, it is not surprising to find that it is naturally present in water. The concentration of fluoride in the oceans is approximately 1.3 ppm (Turekian, 1969). In the United States, fluoride concentrations in wells, lakes, and rivers range from below detection to 16 ppm (National Research Council, 2006). For example, Lake Michigan's fluoride level is 0.17 ppm, wells in Arizona have concentrations up to 7 ppm, and groundwater in Bauxite, Arkansas, has up to 14 ppm fluoride (ADA, 2005). In Alaska, a voluminous DEC data sheet (Alaska Department of Environmental Conservation, 2010) demonstrates that although many natural water systems around the state have undetectable levels of fluoride, one area (Wales) has 2 ppm fluoride in groundwater, and several sources of groundwater in the Fairbanks area have from 0.1 to 0.3 ppm fluoride. Several independent studies of domestic, commercial, and monitoring wells in the greater Fairbanks area show that fluoride is present at concentrations ranging from 0.1 to 1.6 ppm (Fig. 5.1; USGS, 2001; Mueller, 2002; Verplanck et al., 2003).

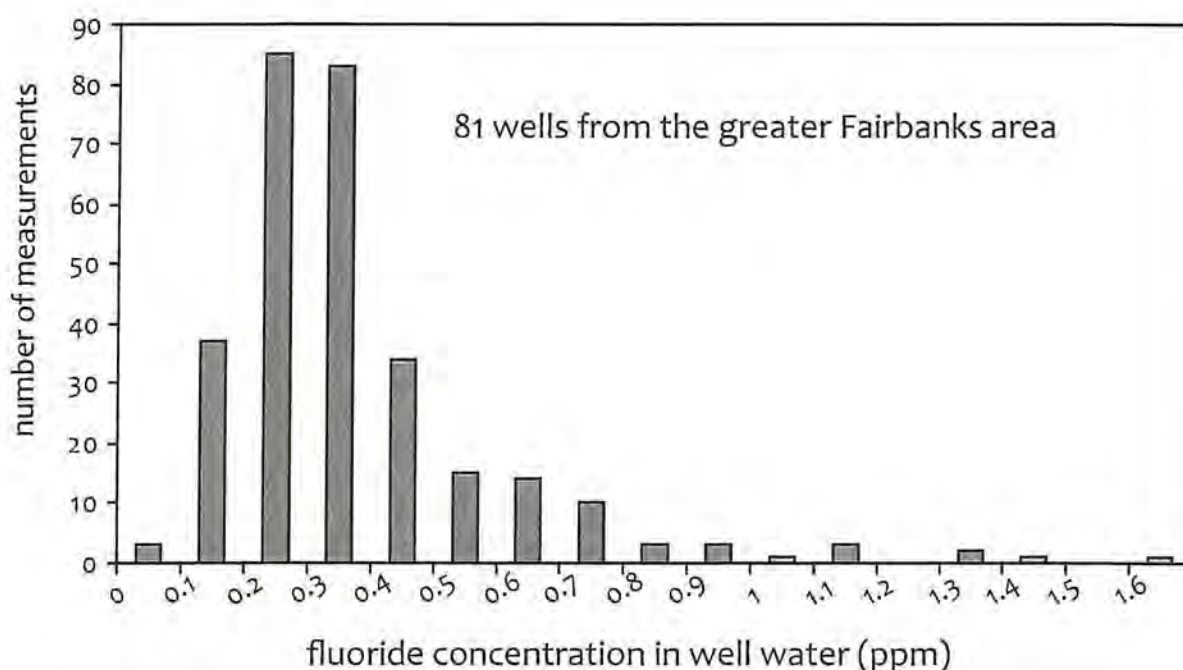


Figure 5.1. Histogram of fluoride concentrations in 81 wells in the Fairbanks area. The median value is between 0.2 and 0.3 ppm, and the bulk of values are between 0.1 and 0.7 ppm. Wells in metamorphic rocks contain the higher fluoride concentrations; those tapping the sedimentary aquifer have values of 0.2 to 0.4 ppm. Data from USGS, 2001; Mueller, 2002; Verplanck et al., 2003; and Alaska Department of Environmental Conservation, 2010).

Wells employed for Fairbanks city water are at depths greater than 100 feet below the surface and tap the sedimentary aquifer of the Fairbanks floodplain. The several hundred feet of sediment is essentially uniform in mineralogy and mineral compositions, hence, by reaction with groundwater it creates water with an essentially constant composition. The fluoride content of raw water from these wells has been tested numerous times between 1987 and 2008 yielding an average fluoride concentration of 0.34 ± 0.1 ppm (Fig. 5.2). Given the constant substrate for groundwater in the Fairbanks floodplain, there is every reason to consider this fluoride concentration to be the same for a very long time to come.

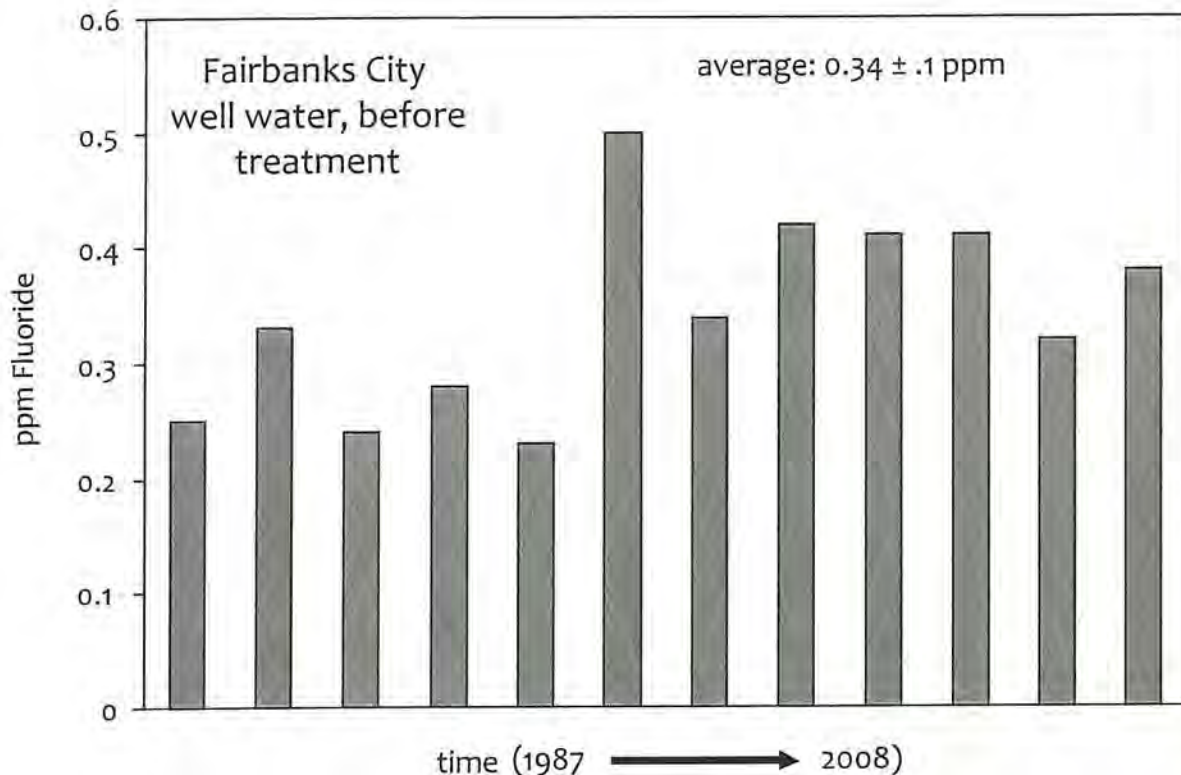


Figure 5.2. Fluoride concentrations in Fairbanks municipal raw well water prior to treatment and fluoridation. Each bar represents a single measurement. Based on checks of fluoride standards, the uncertainty of a given measurement is approximately 0.05 ppm. Data supplied by GHU.

A major source of exposure to fluoride for many Americans, including those who receive GHU water, is drinking water. While this exposure is clearly related to the concentration of fluoride in the water, it is important to distinguish between concentration and dose. The amount of fluoride (dose) an individual receives from drinking water depends on the concentration of fluoride in the water and the amount of water consumed. Thus an individual who drinks one liter of water containing 0.5 ppm fluoride receives the same dose of fluoride as another individual who drinks two liters of water containing 0.25 ppm. Various surveys have found that the amount of drinking water consumed by individuals varies considerably. For example, an EPA report (2004) states that the results from surveys done in the 1990s indicate that very young children consume an average of about 0.3 liter of drinking water per day and adults about 1 liter, as opposed to earlier EPA and WHO estimates of 1 liter and 2 liters, respectively. More importantly, the ranges of consumption are enormous: among the study subjects, infants less than one year old had water consumptions ranging from 0.03 liter to 1.5 liters, and the range among adults was from 0.1 liter to over 4 liters. The situation is further complicated by the fact that certain metal ions present in many water supplies can react with fluoride ions (before consumption) in a way that alters the uptake of fluoride from drinking water by humans (Institute of Medicine, 2000; Urbansky, 2002). For example, in seawater about one-half of the total fluoride is actually present as the MgF^+ complex ion (Bethke, 1996). Therefore, it is very difficult to determine how much fluoride any individual actually consumes from drinking water on a daily basis. Furthermore, “average consumption” is meaningful for a relatively small segment of the population (see Fig. 5.3 for one representation of the situation).

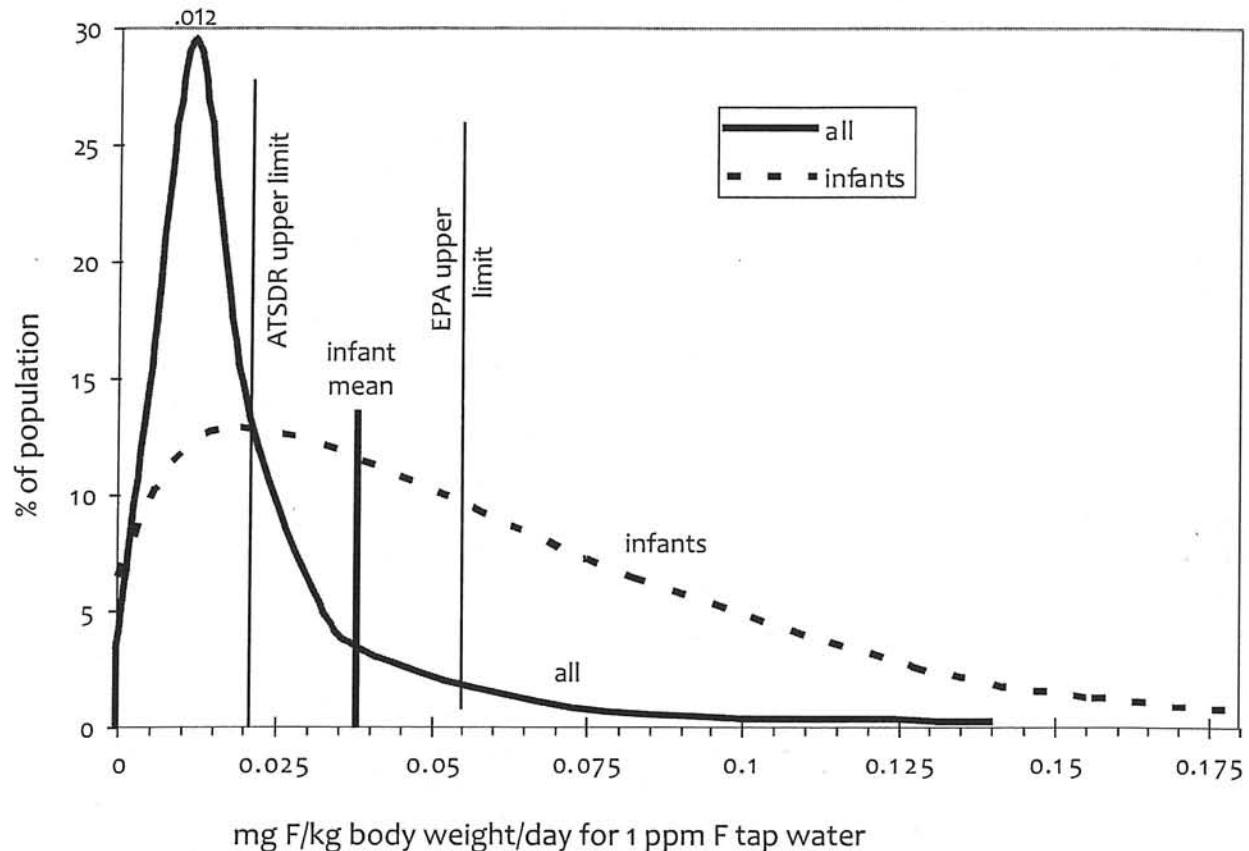


Figure 5.3. Fluoride consumption from tapwater distribution for total population (dark, solid curve) and for infants (dashed curve). Note that only a small proportion of the population receives the target dose from tap water and that a large proportion of infants receive a dose considerably higher than the target dose. Graph constructed from data in EPA (2000).

Agencies of the U.S. federal government, taking into account information that documents the adverse effects of human consumption of large doses of fluoride, have issued regulations and recommendations on the concentrations of fluoride ion in drinking water. The U.S. Environmental Protection Agency (EPA) has set a maximum contaminant level (MCL) for fluoride at 4 ppm and a secondary maximum contaminant level (SMCL) of 2 ppm (to provide a margin of safety against development of fluorosis from exposure to fluoride in drinking water—see Chapter 7). In 1962 the U.S. Public Health Service adopted standards that call for fluoride concentrations between 0.7 ppm and 1.2 ppm in public water supplies that have been “artificially fluoridated” or have “adjusted” levels of fluoride. This range of concentrations was selected based on estimates of water consumption that take into account differences based on climate and the assumption that people in warmer climates drink more tap water than do residents in cooler climates.

In January 2011, just as the Fairbanks task force was finalizing the first draft of its report and recommendations, two federal agencies initiated formal processes to change policy and regulations related to fluoride exposure. In early January, the secretary of the U.S. Department of Health and Human Services (HHS) issued a notice that HHS was seeking public comment on a proposed new recommendation that communities that are fluoridating or choose to fluoridate their public water

supplies adjust the fluoride concentration to 0.7 ppm (http://www.hhs.gov/news/press/2011pres/01/pre_pub_frn_fluoride.html). This recommendation is based on the considerations that (1) scientific evidence indicates that water fluoridation is effective in preventing dental caries, (2) fluoride in drinking water is now just one of several sources of fluoride, (3) the prevalence and severity of dental fluorosis has increased in recent years, and (4) the water consumption of children and adolescents is independent of ambient temperatures. At this writing, the HHS action is limited to initiating the public comment period and does not constitute a formal change in the HHS recommendation. A few weeks later, the EPA initiated a “Registration Review” of the pesticide sulfuryl fluoride. This chemical, used for controlling insect pests in a variety of stored agricultural products, breaks down during application to release fluoride ions. Although the fluoride residue from sulfuryl fluoride contributes negligibly to the fluoride exposure of individual humans, this proposal is based on the EPA’s assessment that “aggregate fluoride exposure is too high for certain identifiable subpopulations in the United States, in particular children under the age of seven who live in areas with higher fluoride concentrations in drinking water resulting from natural background sources” (EPA, 2011; Office of Pesticide Programs, 2011). Under the Federal Food, Drug, and Cosmetic Act, EPA must withdraw sulfuryl fluoride under these circumstances, and the action initiated at this point (invitation for public comment) is the first step in the withdrawal process.

The exposure of a given individual in the Fairbanks area to fluoride from drinking water is very difficult to assess because of the various sources of drinking water available in the area. However, for the purposes of this report, we will focus on individuals who are served by the Golden Heart Utilities water system. This distribution includes about 30,000 people (approximately 6,500 hookups) in the city of Fairbanks and an additional 10,000 to 25,000 individuals (approximately 2,200 hookups, including several water delivery services) in the surrounding area served by College Utilities. Until January of 2011 the drinking water supplied to these individuals contained, on average, 1.0 ppm fluoride. The GHU records examined by the task force demonstrated that over an extended period of time, the range of fluoride concentration in the distributed water was from 0.8 to 1.1 ppm. The variability in the concentration of fluoride was probably due to measurement uncertainties and to the fluctuation in fluoride concentration in the feed water for the GHU process—averaging 0.3 ppm but ranging from 0.2 to 0.4 ppm (Fig. 5.2). In response to the HHS action described in the previous paragraph, in January of 2011 GHU reduced the concentration of fluoride in distributed water from 1.0 ppm to 0.7 ppm. Thus the GHU fluoridation process presently raises the fluoride concentration from about 0.3 ppm in the groundwater to 0.7 ppm in the distributed water.

The process used by GHU to produce water containing 0.7 ppm fluoride is one of the two most common approaches used elsewhere in the United States. A calculated amount of sodium fluorosilicate (SFS) is added to the raw water in a rather sophisticated treatment process. The SFS originates at KC Industries in Mulberry, Florida, where it is manufactured and purified as a byproduct from the domestic phosphate fertilizer industry. Each lot of SFS is analyzed and verified as meeting or exceeding American Water Works Association standards of purity before it is shipped. The material used by GHU is shipped from Florida by truck and container ship to Univar in Anchorage then by truck to Fairbanks. Univar has on record the certificates of assurance for the purity of each lot of SFS that it receives (R. Holland, personal communication). A member of the Fairbanks Fluoride Task Force conducted a laboratory analysis of a sample of SFS provided by GHU and found it to be impressively pure (Table 5.1) relative to typical laboratory chemicals. When used in the fluoridation

process, the calculated concentrations of metal ions added from the SFS are in the parts per trillion range, well below limits set by the EPA. While there are no guarantees against accidents in which fluoride levels in distributed water could rise to a dangerous point, the GHU fluoridation process is well run and has controls in place to provide a high level of assurance of safe operation. Each year since 2006 GHU has received a “Water Fluoridation Quality Award” from the Alaska Oral Health Program (Alaska Division of Public Health). The fluoride concentration in drinking water is measured three times each day, and the concentrations of eleven metals and radionuclides are analyzed on schedules that range from every three to nine years.

Table 5.1a. Major elemental components of a random sample of KC Industries’ sodium fluorosilicate^a

Element	Weight %	Element	Weight %
Silicon	14.8	Fluorine	60.3
Sodium	24.9	Chlorine	0.24

Table 5.1b. Trace elements in a random sample of KC Industries’ sodium fluorosilicate^a

Element	ppm	Element	ppm
Aluminum	25	Arsenic	<4
Barium	<5	Bromine	132
Cobalt	<1	Chromium	<1
Copper	<5	Iron	35
Iodine	35	Nickel	<2
Phosphorous	34	Lead	<1
Antimony	<5	Thorium	<0.5
Vanadium	<1	Tungsten	<2
Zinc	<2		

Table 5.1c. Approximate concentrations of elements added to Fairbanks water after the fluoride concentration has been adjusted to 0.7 ppm

Element	ppm	Element	ppm
Silicon	0.1	Fluorine	0.4
Sodium	0.2	Chlorine	0.002
Element	ppt ^b	Element	ppt ^b
Aluminum	21	Arsenic	<4
Barium	<4	Bromine	11
Cobalt	<1	Chromium	<1
Copper	<4	Iron	28
Iodine	28	Nickel	<1
Phosphorous	28	Lead	<1
Antimony	<4	Thorium	<0.4
Vanadium	<1	Tungsten	<1
Zinc	<2		

a. Analysis by XRF at the University of Alaska Fairbanks, Advanced Instrumentation Lab; R. Newberry, analyst

b. ppt = parts per trillion

Exposure of individuals to fluoride from dental products was not an issue when fluoridation of public water supplies was first introduced in the 1940s. Fluoridated toothpaste became commercially available in 1955, and it rapidly became widely accepted as an agent for caries prevention. However, inadvertent intake of fluoride from toothpaste can be a problem, especially with children who may have poor control of the swallowing reflex. Detailed studies of fluoride ingested by children from swallowing toothpaste have led to ingestion estimates ranging from 0.1 to 0.4 mg per brushing (Ophaug et al., 1985; Levy and Zarei-M. 1991; Rojas-Sanchez et al., 1999). A USPHS report (Institute of Medicine, 2000) summarized the findings by concluding that an average of about 0.3 mg of fluoride is introduced with each episode of tooth brushing in young children. Additional, and highly variable, amounts of fluoride may be ingested by individuals who take fluoride supplements (e.g., drops) or receive topical fluoride application by dental professionals.

Many foods and beverages contain detectable amounts of fluoride. The USDA National Fluoride Database on the fluoride content of a wide range of beverages and foods (USDA, 2004) contains an extensive list. Some representative entries from the USDA database are displayed in Table 5.2.

Table 5.2. Fluoride concentrations in selected foods and beverages available in the United States. Adapted from USDA National Fluoride Database of Selected Beverages and Foods (2004) and Lalumandier and Ayers (2000).

Food or Beverage	Mean (ppm)	Standard Deviation	Range (ppm)
Dairy Products	0.25	0.38	0.02–0.82
Grain and Cereals	0.42	0.40	0.08–2.01
Potatoes	0.49	0.26	0.210–0.84
Leafy Vegetables	0.27	0.25	0.21–0.84
Fruits	0.06	0.03	0.02–0.08
Sugar and Substitutes	0.28	0.27	0.02–0.78
Tea (brewed)	3.7	0.6	2.6–5.3
Soda Pop or Cola	0.5	0.1	0.05–0.8
Bottled Water ^a	NA	NA	0.02–0.94

a. An analysis of bottled water available in Scotland found some European bottled waters to contain nearly 6 ppm (MacFayden et al., 1982).

Part of the variation in fluoride concentrations in foods reflects differences in plant metabolism (for example, tea leaves seem to sequester higher concentrations of fluoride than do the leaves of lettuce or kale). However, one notable aspect of the range of fluoride concentrations in prepared foods is what is called the “halo effect”—the result of the use of fluoridated water to prepare foods and beverages (Griffin et al., 2001). Thus, the fluoride content of processed foods and beverages reflects, in large part, the fluoride concentrations in the water used in their processing.

While the halo effect is manifested in a variety of products, perhaps the most obvious is bottled water, a product of special interest to residents of communities with fluoridated water supplies because it provides an alternative to tap water. The fluoride content of bottled water is regulated by law (see National Research Council, 2006), and it can contain up to 2.4 ppm fluoride with no requirement for a statement of fluoride content on the label, unless fluoride has been added. The large range of

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

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

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

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

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

a. Assuming all water, tap plus other, at 1.0 ppm

b. NRC (2006), Table 2-9

c. NRC (2006), Table 2-11

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

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

a. Calculated from Table 5.3, assuming all water, tap plus other, at 0.7ppm NRC (2006)

b. NRC (2006), Table 2-9

c. NRC (2006), Table 2-11

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

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

a. Calculated from Table 5.3, assuming all water, tap plus other, at 0.3ppm

b. NRC (2006), Table 2-9

c. NRC (2006), Table 2-11

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

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

a. Calculated from Table 5.3, assuming all water, tap plus other, at 1.0ppm

b. NRC (2006), Table 2-9

Table 5.7. Estimated fluoride exposure (mg/kg body weight/day) of U.S. populations on water with 0.7 ppm fluoride, based on water intakes estimated by EPA in 2004

Population	water ^a	toothpaste ^b	background food ^b	pesticides & air ^b	total exposure	% from water
Nursing infant	.012		.0046	.0019	.019	63
Non-nursing Infant	.039		.0114	.0019	.052	75
1–2 year old	.020	.0115	.0210	.0020	.055	36
3–5 year old	.018	.0114	.0181	.0012	.049	37
6–12 year old	.012	.0075	.0123	.0007	.033	36
13–19 year old	.010	.0033	.0097	.0007	.024	42
20–49 year old	.013	.0014	.0114	.0006	.026	50
50+ year old	.013	.0014	.0102	.0006	.025	52

a. Calculated from Table 5.4, assuming all water, tap plus other, at 0.7ppm

b. NRC (2006), Table 2-9

Table 5.8. Estimated fluoride exposure (mg/kg body weight/day) of U.S. populations on water with 0.3 ppm fluoride, based on water intakes estimated by EPA in 2004

Population	water ^a	toothpaste ^b	background food ^b	pesticides & air ^b	total exposure	% from water
Nursing infant	.0051		.0046	.0019	.012	43
Non-nursing Infant	.017		.0114	.0019	.030	57
1–2 year old	.0087	.0115	.0210	.0020	.043	20
3–5 year old	.0078	.0114	.0181	.0012	.039	20
6–12 year old	.0051	.0075	.0123	.0007	.026	20
13–19 year old	.0042	.0033	.0097	.0007	.018	23
20–49 year old	.0054	.0014	.0114	.0006	.019	28
50+ year old	.0054	.0014	.0102	.0006	.018	30

a. Calculated from Table 5.5, assuming all water, tap plus other, at 0.3 ppm

b. NRC (2006), Table 2-9

Several things must be kept in mind when interpreting the data in these tables:

- The average intakes of water are based on two different estimates of water consumption (NRC, 2006; EPA, 2004). The following pairs of tables allow direct comparison of the overall estimated exposures based on the differences in estimates of water intake: Tables 5.3 and 5.6, Tables 5.4 and 5.7, Tables 5.5 and 5.8.
- The range of water intakes among individuals is quite large.
- For simplicity of calculation, the estimated intake of fluoride from water assumes that all water has the fluoride concentration indicated in each table. This clearly is not the case for someone who uses several sources of water (for example, well, public system, and bottled) on a regular basis. This assumption, coupled with the range of fluoride concentrations in commercial bottled water, injects quite a bit of uncertainty into the results of these calculations.
- The estimated amounts of fluoride ingested by individuals from toothpaste are for individuals who regularly brush twice daily with fluoridated toothpaste and who have control over swallowing.
- Estimates of intakes from food (and beverages) are really just educated guesses because of variability in diets and in the magnitude of the halo effect.

Despite the limitations on the validity of the estimates of exposure, the data in the tables can be evaluated in light of recommendations made by relevant organizations of health professionals. There

have been a number of recommendations through the years, and the situation is complicated by the fact that some recommendations are in terms of mg per individual per day and others in terms of mg per kg per day. In the opinion of the task force, the key recommendations on fluoride are:

- Adequate daily intake (Institute of Medicine, 1997):
 - 0.0014 mg/kg/day for infants 0–6 months
 - 0.06 mg/kg/day for infants 7–12 months
 - 0.05 mg/kg/day for other children and all adults
- Upper limits:
 - Agency for Toxic Substances and Disease Registry (ATSDR): 0.023 mg/kg/day
 - Environmental Protection Agency (EPA, 2010): 0.06 mg/kg/day
 - Institute of Medicine tolerable upper intake (Institute of Medicine, 1997):
 - 0.1 mg/kg/day for newborns through age 8
 - 0.15 mg/kg/day for ages 9 through adult

The ATSDR limit (MRL, minimal risk level) is an estimate of the daily human exposure to sodium fluoride that is likely to be without appreciable risk of adverse noncancer health effects (set, in the case of sodium fluoride, by the lowest level of fluoride judged to be correlated with increased bone fracture rates and then divided by a “safety factor” of ten). The ATSDR “upper limit” of 0.023 mg/kg/day for fluoride cited in this report takes into account the fluoride content of sodium fluoride for which the ATSDR has set an MRL of 0.05 mg/kg/day. The EPA limit (“reference dose”) is based on a “no observed adverse effect level” for mottling of the teeth. The Institute of Medicine limits (tolerable upper intake limits, or UL’s), which were also endorsed by the American Dental Association in 1994 and the American Dietetic Association in 2000, are set to minimize the risk of dental fluorosis but are at or near those that have been associated with mild (Institute of Medicine, 1997) or even crippling (National Research Council, 1993) skeletal fluorosis. While these upper limit recommendations have been used in formulation of a number of public health programs, the opponents of fluoridation have often critiqued and questioned the propriety of the recommendations and have called for lower limits for exposure to fluoride (see, for example, Connett et al., 2010). The problems associated with using these guidelines to develop public policy is perhaps best illustrated by the observation that the adequate daily intakes recommended by the Institute of Medicine for individuals greater than six months of age are equal to or greater than upper limits recommended by the ATSDR and the EPA.

The relationships between estimated fluoride exposures of several subpopulations of Fairbanks residents consuming drinking water with 0.7 or 0.3 ppm fluoride can be analyzed with the aid of Figs. 5.4 and 5.5 (derived from Tables 5.7 and 5.8, respectively). In analyzing these data, it is important to keep in mind that the numbers represent “average” individuals and that the consumption of drinking water varies widely among individuals (Fig. 5.1). In the existing scenario (0.7 ppm fluoride in drinking water, Fig. 5.4), it is apparent that nursing infants (NI) are estimated to be exposed to daily fluoride doses well below those established by ATSDR, EPA, and IOM; those over twenty years of age (20+ YR) have exposure well below EPA and IOM upper limits and about at the limit recommended by ATSDR. However, non-nursing infants (NNI) and one to five year-olds receive daily doses significantly above the ATSDR recommendation, marginally below that recommended by EPA, and significantly below that recommended by IOM. In contrast, while drinking water with 0.3 ppm fluoride does place non-nursing infants and one to five year-olds at risk of exceeding ATSDR upper limits, the exposure of other age groups remains below the ATSDR recommendation. Furthermore, no age group risks exposure greater than the recommended upper limits of the EPA or IOM (Fig. 5.5).

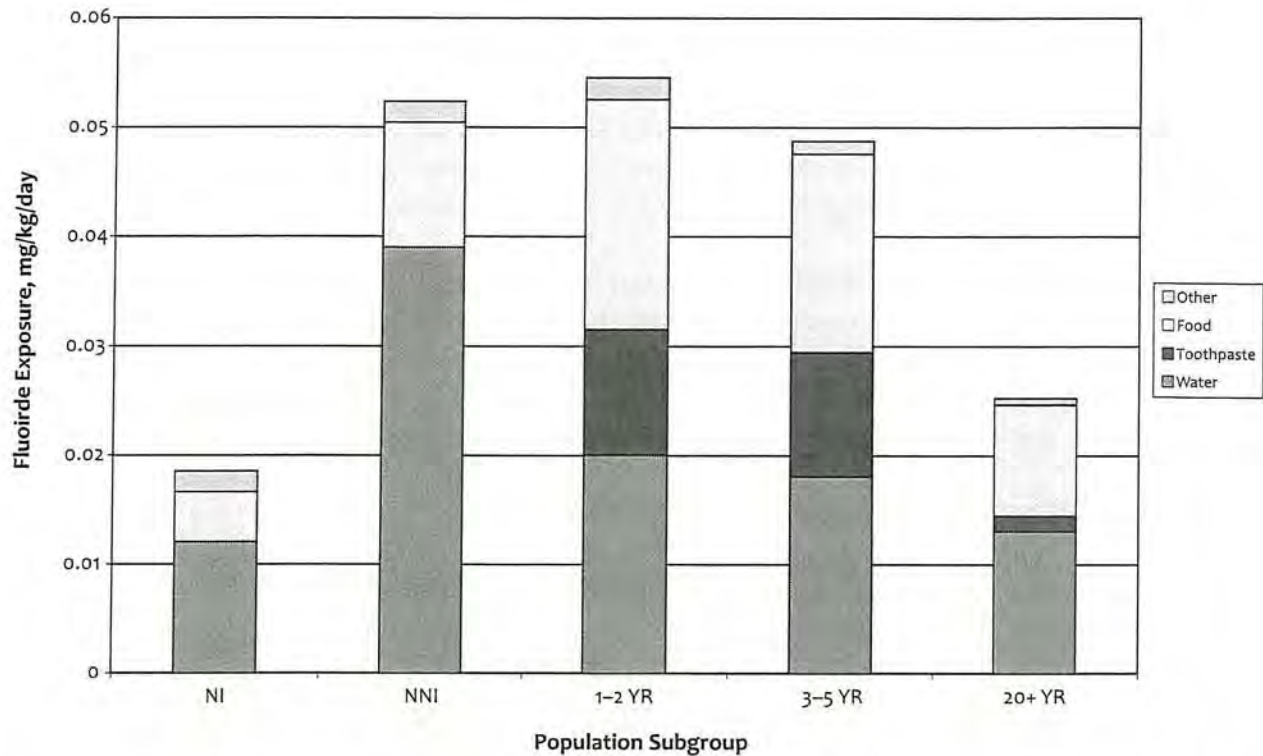


Figure 5.4. Estimates of fluoride exposure of individuals with 0.7 ppm fluoride in drinking water (data from Table 5.7)

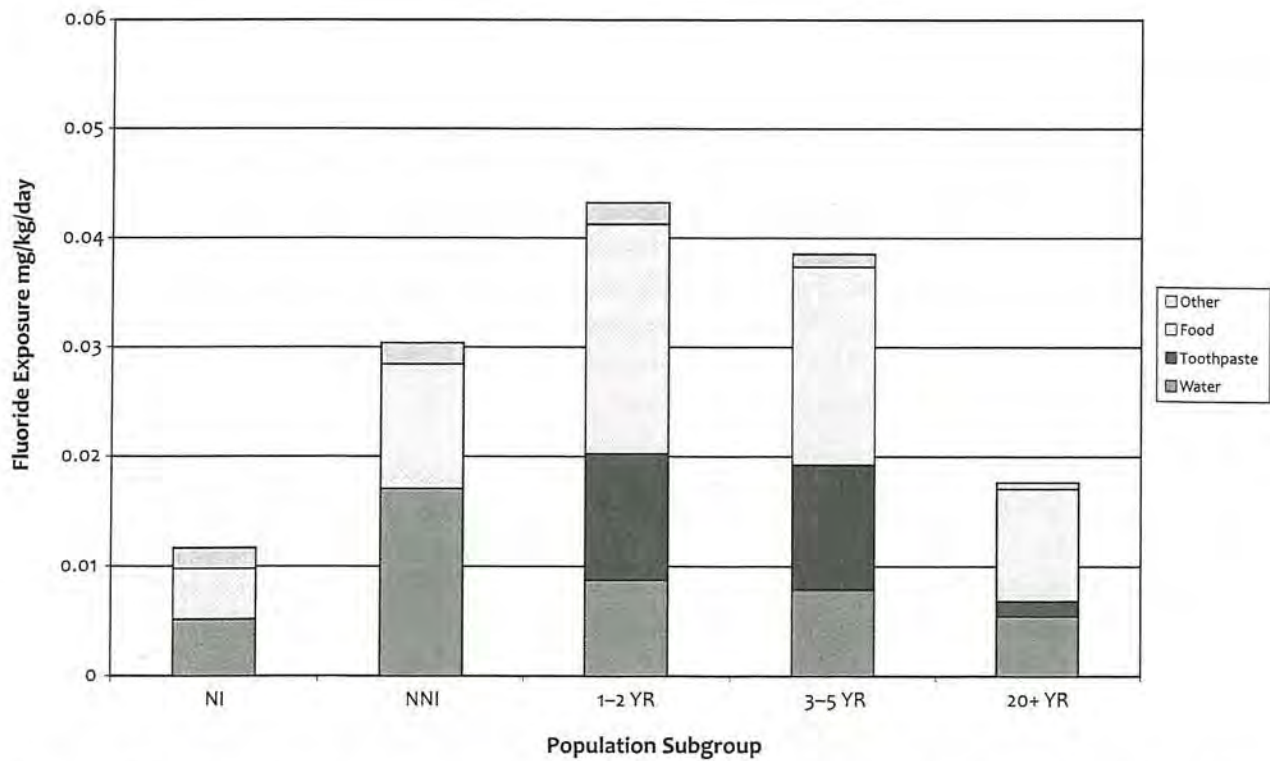


Figure 5.5. Estimates of fluoride exposure of individuals with 0.3 ppm fluoride in drinking water (data from Table 5.8).
NI = nursing infant, NNI = non-nursing infant

In addition to the officially defined upper and lower limits for exposure to fluoride, there has been a widely accepted “optimal intake” of fluoride of 0.05 to 0.07 mg/kg/day. The optimal intake was thought to be a narrow range of doses that provide protection from caries but do not cause dental fluorosis. However, recently the concept of an “optimal” intake has been called into question because of (1) the overlap in fluoride intakes of groups of children who are caries-free and groups of children diagnosed with fluorosis and (2) the high variability in individual fluoride intakes (Warren et al., 2009).

Because the Fairbanks Fluoride Task Force had concerns about exposure of infants to fluoride and about the uncertainties associated with estimates of drinking water consumption, we performed some independent calculations. The results of the calculations for infants are displayed in Table 5.9. While the values in Table 5.9 are not identical with corresponding entries in Tables 5.3 through 5.5, the task force judges that they are sufficiently consistent, given the uncertainties and assumptions involved.

Table 5.9. Average fluoride intake per day by non-nursing infants (mg/kg/day)

Age	1 ppm F in water	0.7 ppm F in water	0.3 ppm F in water	upper limit
Birth	0.164	0.115	0.049	0.023,*0.10 ^b
1 mo.	0.161	0.113	0.048	0.023,*0.10 ^b
2 mo.	0.179	0.125	0.054	0.023,*0.10 ^b
4 mo.	0.130	0.091	0.039	0.023,*0.10 ^b
8 mo.	0.089	0.064	0.027	0.023,*0.10 ^b
10 mo.	0.070	0.049	0.021	0.023,* 0.10 ^b
12 mo.	0.065	0.045	0.019	0.023,* 0.10 ^b

a. ATSDR

b. IOM (1997)

Findings

1. The problematic relationship between fluoride concentration in drinking water and “fluoride dose,” due to varying amounts of water consumed by individuals and to other sources of ingested fluoride, severely complicates attempts to determine both health risks and benefits associated with 0.7 ppm fluoride in drinking water. In particular, commonly available foods and beverages contain from high (greater than 2 ppm) to negligible levels of fluoride, and fluoridated toothpaste is variably used and swallowed. We believe that these factors grossly complicate interpretation of drinking water studies and explain why the numerous studies conducted have come to a variety of conclusions that, in some cases, are quite different.
2. The concentration of fluoride in raw Fairbanks city water averages 0.3 ppm and is adjusted to 0.7 ppm in the treatment process. Because removing the fluoride from the raw water is impractical, the City of Fairbanks does not seem to have a realistic option for “fluoride free” city water (for a discussion of fluoride-removal processes see Fawell et al., 2006). Whatever benefits and detriments are caused by fluoride in drinking water will continue to a smaller degree if Fairbanks city water is no longer fluoridated.
3. Fluoride concentrations in Fairbanks area well water vary from 0.1 to greater than 1.0 ppm. Thus, some well water in the Fairbanks area contains more fluoride than fluoridated city water.

4. Fluoridation of Fairbanks city water has ramifications throughout the surrounding area because of the distribution of GHU water by College Utilities and several suppliers of trucked water.
5. The practice of fluoridation as carried out in Fairbanks has sufficient safeguards to protect public health beyond whatever health effects are associated with 0.7 ppm fluoride. The chemical employed is of sufficient purity and the manner in which it is added and monitored meets or exceeds standard practices.
6. An analysis of the estimates in Tables 5.3 through 5.8 and Figures 5.4 and 5.5 indicates that two segments of the Fairbanks area population must be considered separately with respect to professional recommendations on upper limits of fluoride exposure: (1) the average consumer of GHU water (fluoride concentration of 0.7 ppm) who is greater than five years of age is projected to consume less than the daily upper limits set by the EPA and IOM and just about at the upper limit set by ATSDR, and (2) children less than six years of age (with the exception of nursing infants) are projected to have total fluoride exposures that remain below the upper limits set by IOM and EPA but exceed those of ATSDR. It appears that drinking water with a fluoride concentration of 0.3 ppm would bring total fluoride exposure for those over 20 years of age well below even the most stringent of the recommendations of upper limits (ATSDR) and would significantly reduce concerns about overexposure of infants and young children. However, due to the tremendous variability in amount of drinking water consumed by individuals, the fluoride exposures of significant portions of the population are not adequately represented by the average values.
7. Nevertheless, the estimates of Table 5.9 highlight additional concerns about fluoride exposure of non-nursing infants in their first year. The use of fluoridated water to make up infant formula leads to levels of fluoride consumption that exceed recommended upper limits. While the magnitude of the problem obviously declines with a decline in fluoride concentration in the water used to make up formula, the most conservative of the upper limits of fluoride exposure would be approached or exceeded even when using GHU well water (fluoride concentration averaging 0.3 ppm) to which no fluoride has been added. While bottled water would seem to be the water of choice, the data of Table 5.2 indicate that not all bottled waters available in the United States would provide this level of protection. The use of bottled water for this purpose is further complicated by the absence of information about fluoride content on the labels of most bottled water. The only certainty for consumers seems to be that the distilled water sold in supermarkets has an undetectable concentration of fluoride.

Chapter 6

Efficacy of Community Water Fluoridation

Evaluation of Efficacy Before 2000

The addition of fluoride was effective in reducing caries in those municipalities that were the subject of reports in the primary dental literature during the mid-twentieth century. The Ft. Collins report gives the historical background that led to widespread fluoridation of public water systems:

In 1901, a Colorado Springs dentist recognized that his patients with teeth with a brown stain or mottled dental enamel also had a very low prevalence of cavities (also called caries) (Centers for Disease Control and Prevention [CDC], 1999b). At this time in history, extensive dental caries were common, so this observation and its subsequent correlation with high amounts of fluoride ion in the water supply (2.0–12.0 milligrams per liter, mg/L) proved to be significant. Another dentist, H. T. Dean, DDS, took this information and conducted a survey of dental caries in relation to natural concentrations of fluoride in drinking water of 21 U.S. cities (Committee to Coordinate Environmental Health and Related Programs, USPHS [USPHS], 1991, pp. 18–19; CDC, 1999a, p. 934). Dean observed that at a concentration of 1 mg/L, fluoride would significantly reduce caries while causing a low incidence of mottled enamel, now called fluorosis, of the mostly very mild type. Beginning in 1945 and 1946, community trials were conducted over 13–15 years in four pairs of cities in the U.S. and Canada. These studies found a 50–70% reduction of caries in children following addition of fluoride (in the form of sodium fluoride) to community water supplies at 1 mg/L. The incidence of mild fluorosis remained low (CDC, 1999a, p. 936). Some of the early studies were criticized for lacking appropriate controls, not applying randomization, and not controlling for potential examiner bias (Sutton, 1960). However, the large effect sizes in these trials, along with replication of these findings in subsequent studies, led to the acceptance of community water fluoridation as a public health approach to caries prevention. (Fluoride Technical Study Group, 2003)

Many reviews and meta-analyses, which combine the results of several studies that address a set of related research hypotheses, support the hypothesis that water fluoridation reduces the incidence of caries. The York Report (McDonagh et al., 2000) is a systematic review made to assess the evidence of the positive and negative effects of population-wide drinking water fluoridation strategies to prevent caries. It is a summary of 254 studies published from the mid-1960s to mid-1999, which were chosen for relevance from over 3,000 studies identified in the literature. The authors of the York Report identified five objectives to make their assessment.

Their first objective was to answer the question: “What are the effects of fluoridation of drinking water supplies on the incidence of caries?” Of the 254 studies, twenty-six were relevant to this question. They are optimistic about the caries reductions caused by water fluoridation, yet cautious.

The best available evidence suggests that fluoridation of drinking water supplies does reduce caries prevalence, both as measured by the proportion of children who are caries free and by

the mean change in dmft/DMFT score. The studies were of moderate quality (level B), but of limited quantity. The degree to which caries is reduced, however, is not clear from the data available. The range of the mean difference in the proportion (%) of caries-free children is -5.0 to 64%, with a median of 14.6%. . . . The range of mean change in dmft/DMFT score was from 0.5 to 4.4, with a median of 2.25 teeth. . . . It is estimated that a median of six people need to receive fluoridated water for one extra person to be caries-free. . . . The best available evidence from studies following withdrawal of water fluoridation indicates that caries prevalence increases, approaching the level of the low fluoride group. Again, however, the studies were of moderate quality (level B), and limited quantity. The estimates of effect could be biased due to poor adjustment for the effects of potential confounding factors. (McDonagh et al., 2000, p. xii)

Their second objective was to answer the question: "If water fluoridation is shown to have beneficial effects, what is the effect over and above that offered by the use of alternative interventions and strategies?" Of the 254 studies, nine conducted after 1974 were relevant to this question. Again, their summary statement is positive toward the extra benefits of water fluoridation in the presence of other sources of fluoride:

In those studies completed after 1974, a beneficial effect of water fluoridation was still evident in spite of the assumed exposure to non-water fluoride in the populations studied. The meta-regression conducted for Objective 1 confirmed this finding. (McDonagh et al., 2000, p. xii).

A summary of observed effects of fluoridation on caries in children is presented in Figs. 6.1 and 6.2 (McDonagh et al., 2000, pp. 12–13).

An examination of twenty-one studies, half of which were published between 1990 and 2000, came to a similar conclusion, although without as many caveats: "According to *Community Guide* rules of evidence, strong evidence shows that CWF (community water fluoridation) is effective in reducing the cumulative experience of dental caries within communities" (Truman et al., 2002, p. 28; see <http://www.thecommunityguide.org/index.html> for more about Community Guide).

A meta-analysis of twenty studies concluded that fluoride prevents caries among adults of all ages (Griffin et al., 2007). Some details are worth noting. Water fluoridation was responsible for preventing 27% of the caries. Self- and professionally applied topical fluoride was responsible for the remaining 73% reduction. For studies published after 1980, fluoride from all sources annually averted 0.29 carious coronal and 0.22 carious root surfaces per person. The authors point out the value of all types of fluoride for low-income adults and the elderly, who may not be receiving routine dental care. Note that the York Report (McDonagh et al., 2000) does not support this conclusion.

An epidemiological study in the United Kingdom addressed the question of differences in effect of water fluoridation over a range of socioeconomic groups (Riley et al., 1999). They conclude that water fluoridation reduced dental caries more in materially deprived wards than in affluent wards. In addition, the introduction of community water fluoridation substantially reduced inequalities in dental health. This conclusion is supported to an extent in the York Report (McDonagh et al., 2000, p. xii), although with considerable caution due to the low quality of the evidence and the general lack of variance

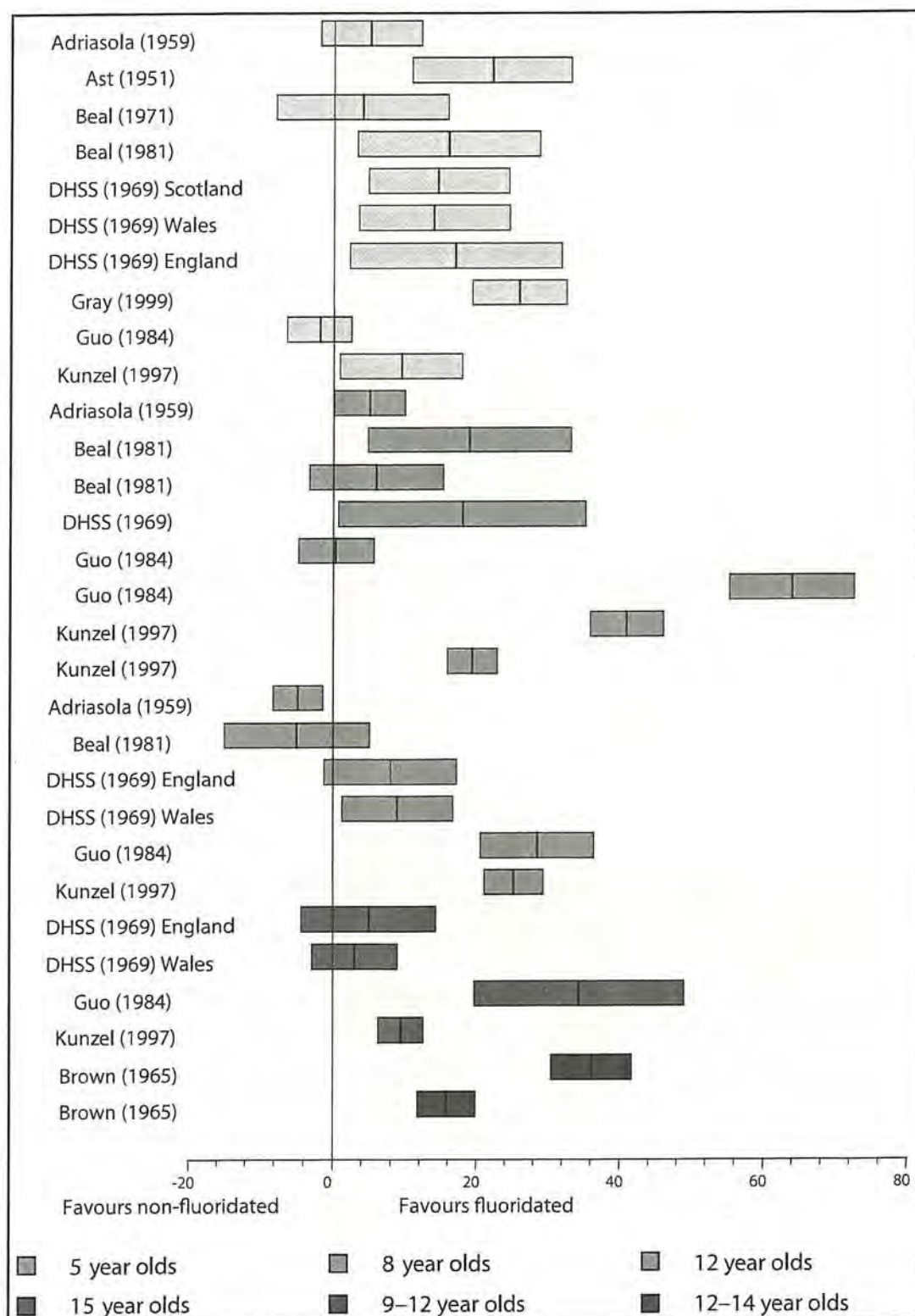


Figure 6.1. The mean difference of the change in the proportion (%) of caries-free children in the exposed (fluoride) group compared with the control group (low fluoride), for all ages extracted (color coded by age), for studies in which fluoridation was initiated after the baseline survey (McDonagh et al., 2000, p. 12)

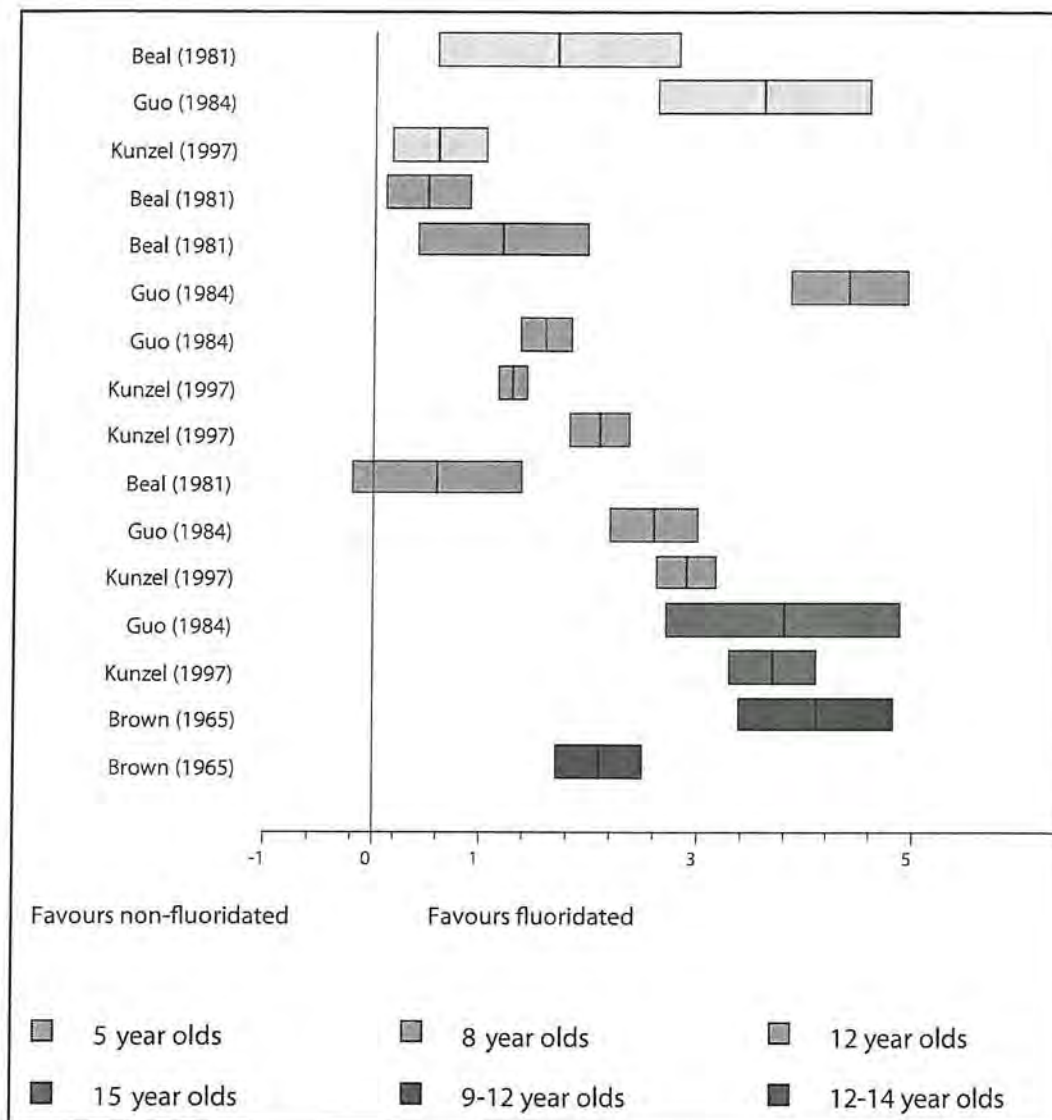


Figure 6.2. Change in dmft/DMFT Score (mean difference and 95% CI) (McDonagh et al., 2000, p. 13)

estimates in the fifteen studies. To objective 3, “Does water fluoridation result in a reduction of caries across social groups and between geographical locations, bringing equity?”, their response was

There appears to be some evidence that water fluoridation reduces the inequalities in dental health across social classes in 5 and 12 year-olds, using the dmft/DMFT measure. This effect was not seen in the proportion of caries-free children among 5 year-olds. The data for the effects in children of other ages did not show an effect. The small quantity of studies, differences between these studies, and their low quality rating, suggest caution in interpreting these results. McDonagh et al., 2000, p. xii)

It is apparently difficult to design and execute good studies to test the hypothesis that fluoridation of public water systems decreases the incidence of caries. Questions have been raised on a regular basis about the design and analysis of studies investigating the efficacy of municipal water fluoridation for the reduction of caries incidence. Concerns about experimental design and examiner bias were raised long ago (Sutton, 1960). The York Report (McDonagh et al., 2000), a meta-analysis of 214 studies published before 2000, presented relatively positive results for efficacy, with many caveats. In particular, they note the general lack of analysis, lack of control for potentially confounding factors, and the lack of any measure of variance for the estimates of decay. The difficulties of an accurate analysis and interpretation of data from a large and carefully designed longitudinal trial have been pointed out, with the observation made that “our analysis shows no convincing effect of fluoride-intake on caries development” in the permanent first molars in children between 7 and 12 years of age (Komárek et al., 2005, p. 145).

Equally important to the critical evaluation of the efficacy of water fluoridation to prevention of caries is “The Mystery of Declining Tooth Decay,” which was reported in the journal *Nature* (Diesendorf, 1986). He notes in summary that “large temporal reductions in tooth decay, which cannot be attributed to fluoridation, have been observed in both unfluoridated and fluoridated areas of at least eight developed countries over the past thirty years” (p. 125). The magnitude of the reductions observed in unfluoridated areas were generally comparable with those observed in fluoridated areas over similar periods. In his discussion of the why’s of the reductions, the author emphasized the literature that suggests changes in diet, immunity, and perhaps topical fluoride exposure with time are more likely candidates than fluoridated municipal water. The magnitude of the decrease in tooth decay is demonstrated in World Health Organization data, which was put into graphical form (Fig. 6.3) for the antifuoridation Fluoride Action Network (FAN) (Osmunson, 2010b).

The European experience has been one of generally decreasing DMFT scores. This is reported for fluoridated regions, nonfluoridated regions, and regions where fluoridation has been discontinued. In East Germany, the introduction of water fluoridation in Spremberg and Zittau brought about caries reduction averaging 48%. Surprisingly, caries levels for the twelve-year-olds of both towns significantly decreased following the cessation of water fluoridation (Kunzel et al., 2000). In Spremberg, DMFT fell from 2.4 to 1.4 (~40 %) and in Zittau from 2.5 to 2.0 (~20%). In Tiel (The Netherlands), where water fluoridation was discontinued in 1973, DMFS scores varied somewhat less consistently. The mean DMFS score increased between 1968/1969 and 1979/1980 from 10.8 to 12.7 (+18%) and then decreased to 9.6 (–26%) in 1987/1988. Overall the mean DMFS score decreased by 11% from 1968/1969, when water was fluoridated, to 1987/1988, when the town water had been

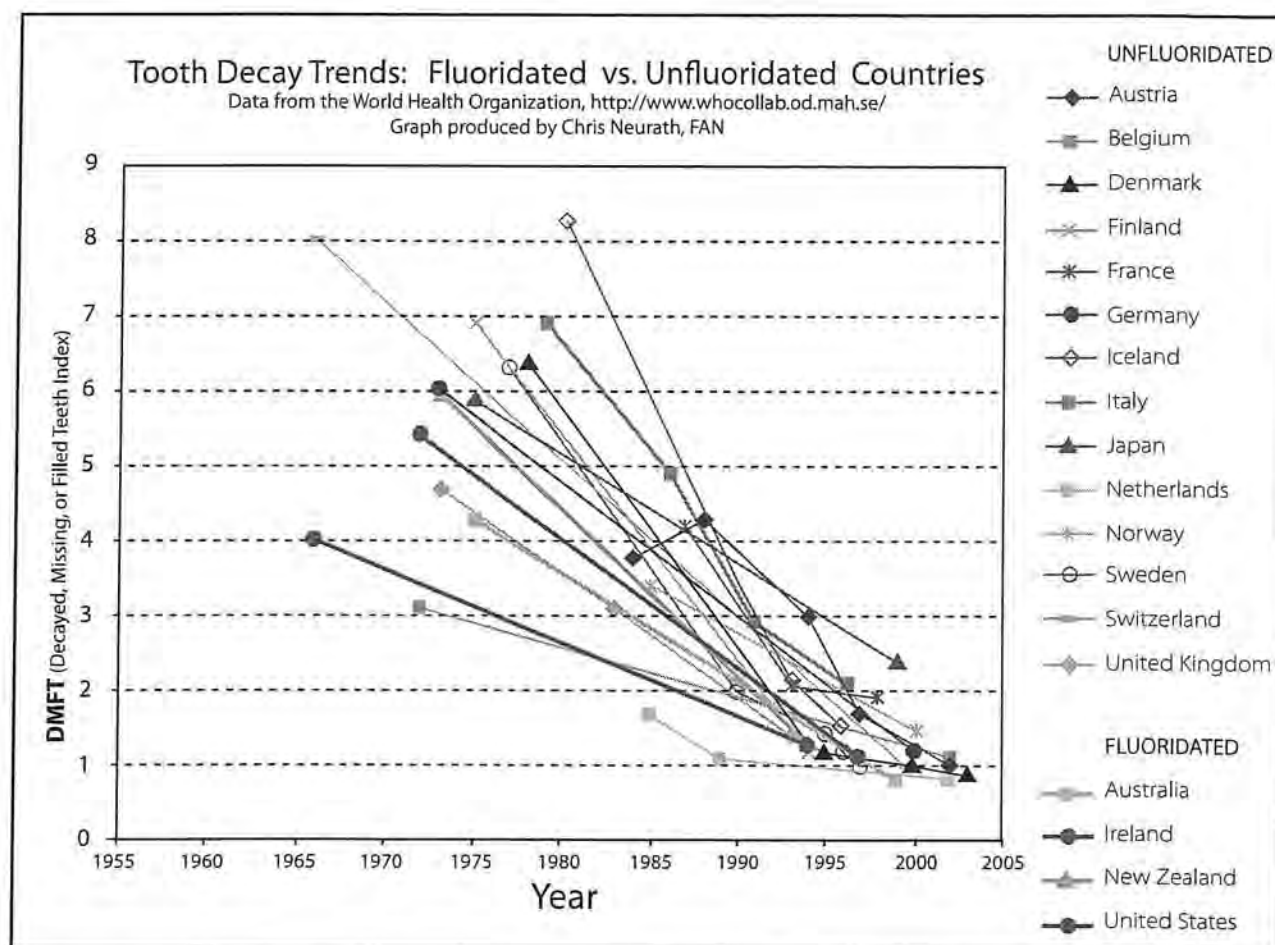


Figure 6.3. Tooth decay trends internationally in countries with fluoridated vs. unfluoridated water

unfluoridated for fourteen years. In Culemborg, where the water was never fluoridated, the mean DMFS score decreased from 27.7 in 1968/1969 to 7.7 in 1987/1988. This decrease of 72% occurred with no fluoridation of the public water supply (Kalsbeek et al., 1993). Presuming the application of existing preventive measures, the question as to whether water fluoridation would have had an additional effect if it had been continued cannot be answered, because no communities in The Netherlands now fluoridate water.

Evaluation of Efficacy After 2000

A recent review of community water fluoridation and caries prevention considers only recent data (Pizzo et al., 2007). Using MEDLINE as the primary database, the authors reviewed articles published from January 2001 to June 2006. They conclude that community water fluoridation is not necessary for caries prevention in modern, industrialized societies. Because the primary cariostatic action of fluoride occurs after tooth eruption, the use of topical fluoride is a more effective approach in communities where caries levels have become low. This line of thought is noted in a recent analysis published in the *British Medical Journal* (Cheng et al., 2007). The average number of decayed, missing, and filled teeth in twelve-year-old children in a number of European countries is near 1.5, and half of children have no cavities. There is no correlation in the downward trends with degree of

water fluoridation. Pizzo and coworkers are cautious, however, and these cautions may be germane in Fairbanks. They state that “water fluoridation may still be a relevant public health measure in populations where oral hygiene conditions are poor, lifestyle results in high caries incidence, and access to a well-functioning oral health care system is limited” (p. 192).

An evaluation of three reviews culled from fifty-nine publications published between 2000 and 2008 resulted in positive support for the effectiveness of water fluoridation in prevention of dental caries (Parnell et al., 2009). Two of the reviews have been discussed previously and they include mostly older literature (McDonagh et al., 2000; Griffin et al., 2007). The third review (National Health and Medical Research Council, 2007) identified one systematic review (Truman et al., 2002) and one cessation study (Seppa et al., 2000) published since the York Report (McDonagh et al., 2000). As noted above, the Truman study was strongly positive toward water fluoridation. In contrast, the Seppa study showed no evidence of increased caries when a previously fluoridated town reverted to nonfluoridated water. Parnell et al. concluded that the two new studies do not change the findings of the York Report that “the existing body of evidence strongly suggests that water fluoridation is beneficial at reducing dental caries” (p. 143).

A recent, somewhat indirect, study makes an association between lack of water fluoridation and inclusion of Nevada youth in the high caries prevalence group (Ditmyer et al., 2010). For adolescents in the study group (the 30% highest DMFT scores, DMFT > 4.0), 27.3% lived in a water-fluoridated community. For the control group (caries free, DMFT score = 0), 64.7% lived in a water-fluoridated community. Thus, participants living in nonfluoridated communities were almost twice as likely to be in the highest DMFT group as those living in fluoridated communities.

Discussions of efficacy may sometimes revolve around the mode of action of fluoride in optimally fluoridated water. The theoretical mechanism by which fluoride prevents caries has undergone significant revision since the introduction of community water fluoridation. The original systemic theory was that fluoride had to be ingested to incorporate into tooth mineral during its development (Dean et al., 1942). By the 1970s, doubts emerged regarding the exclusively pre-eruptive effect of fluoride. Numerous clinical studies suggested that fluoride action is predominantly post-eruptive (topical). While there are conflicting results, most recent epidemiological and laboratory studies indicate that topical application of fluoride plays the dominant role in caries prevention (CDC 2001; Hellwig and Lennon, 2004).

Fluoride's effect depends on its being in the right amount in the right place at the right time. It works primarily after teeth have erupted, especially when small amounts are maintained constantly in the mouth, specifically in dental plaque and saliva. The fluoride in saliva aids in enamel remineralization in enamel lesions by inducing apatite formation from calcium and phosphate ions present in saliva (Fejerskov et al., 1981). The effectiveness of toothpaste in decreasing the prevalence of caries is particularly clear. When introduced into the mouth, fluoride in toothpaste is taken up directly by dental plaque and demineralized enamel. Brushing with fluoride toothpaste increases the fluoride concentration in saliva 100- to 1,000-fold for one to two hours. Some of this salivary fluoride is taken up by dental plaque. The ambient fluoride concentration in saliva and plaque can increase during regular use of fluoride toothpaste (CDC, 2001).

In its recommendations, the CDC (2001) makes a strong argument supporting the topical mode of action in caries prevention. That said, they report that people living in communities with optimally fluoridated water who also use topical fluoride on a regular basis have a lower incidence of caries than people who use only optimally fluoridated drinking water or who only use topical fluoride. Thus the mode of action has been established in the modern literature as predominantly topical. Yet the epidemiological evidence, at least as reported a decade ago by CDC, still shows an empirical effect for fluoride in drinking water. Drinking fluoridated water prevents caries.

When fluoridated water is the main source of drinking water, a low concentration of fluoride is routinely introduced into the mouth. Some of this fluoride is taken up by dental plaque; some is transiently present in saliva, which serves as a reservoir for plaque fluoride; and some is loosely held on the enamel surfaces. Frequent consumption of fluoridated drinking water and beverages and food processed in fluoridated areas maintains the concentration of fluoride in the mouth. (CDC 2001)

Thus, although the mode of action for fluoride in drinking water was initially thought to be systemic, its true action is predominantly topical in caries prevention, as is the action of the fluoride present in toothpaste, supplements, mouth rinse, and professionally applied gels and varnishes.

Publications and a federal proposal made even in the past year show that the jury is very much 'out' with respect to questions about the efficacy of community water fluoridation at 1 ppm fluoride and about the benefit-to-risk assessment.

- A proponent of community water fluoridation has recently written of the existing uncertainties associated with the efficacy of community water fluoridation (Newbrun, 2010). These include the effect of reducing the concentration of fluoride below 1 ppm, the expected result of discontinuing community water fluoridation in a community, and the role of socioeconomic factors in the importance of continuing water fluoridation.
- On January 7, 2011, the U.S. Department of Health and Human Services (HHS) announced a proposal recommending that water systems practicing fluoridation adjust their fluoride content to 0.7 ppm, as opposed to the previous temperature-dependent optimal levels ranging from 0.7 ppm to 1.2 ppm (<http://www.hhs.gov/news/press/2011pres/01/20110107a.html>, accessed January 27, 2011).
- An opponent of community water fluoridation has noted the 15% difference in the proportion of caries-free children reported in the York Report and the 20% to 40% reduction in tooth decay reported by the American Dental Association (Thiessen, 2009a). She has no apparent objection to the numerical accuracy. However, she does put these values in context: "which would translate to < 1 decayed, missing, or filled permanent tooth (DMFT) in older children and adolescents (based on U.S. data from CDC 2005). Is this adequate justification for imposing inadequately characterized risks?" (Thiessen, 2009a, p. 3).

Findings

1. There has never been a double blind, randomized, long-term study of the effectiveness of community water fluoridation on decreasing the incidence of caries. Nor has there been a comparable study on the effect of discontinuing water fluoridation on the incidence of caries.

2. The degree of caries reduction due to community water fluoridation was large and significant in the first decades that it was done. In recent decades, the degree of caries reduction attributed to community water fluoridation has decreased as other sources of fluoride have come into common use and as effective dental health measures have become more prevalent. The relative importance of water fluoridation is currently much smaller, more variable among populations, and perhaps unknowable.
3. The problematic relationship between fluoride concentration in drinking water and “fluoride dose” (due to varying amounts of water consumed by individuals and to other sources of ingested fluoride) severely complicates attempts to determine both health risks and benefits associated with 1 ppm fluoride in drinking water. In particular, at this time commonly available foods and beverages range from high (greater than 2 ppm) to negligible fluoride content, and fluoridated toothpaste is variably swallowed. We believe that these factors grossly complicate interpretation of drinking water studies and explain why the numerous studies conducted have come to a variety of different conclusions.
4. Studies of the relative effectiveness of community water fluoridation among socioeconomic groups give contradictory results. Dietary habits, dental hygiene, and intervention by health/dental providers are independent factors that confound the investigation of the efficacy of fluoridation of water on caries prevalence.

Chapter 7

Adverse Effects

Introduction

Fluoride can clearly lead to adverse health effects in humans. However, as for most chemicals, the dose that one is exposed to is a critical factor in determining the effect(s). For example, many drugs with therapeutic benefit are toxic at higher-than-recommended doses. Further, some drugs may have a very narrow window of therapeutic benefit. That is, the dose at which the drug provides benefit may be only slightly lower than the dose leading to ill effects. We focused primarily on studies that examined the effects on humans of drinking water with fluoride concentrations of less than 2 ppm (or 2 mg/L).

In Fairbanks (Golden Heart Utilities), the water is fluoridated to a concentration of 0.7 ppm. One challenge in understanding possible adverse effects is that, depending on water consumption and other possible sources of fluoride exposure (such as toothpaste or heavy tea consumption), individuals may be exposed to widely different doses of fluoride. Another challenge is that the average expected dose may also vary by age (an infant receiving most nutrition from formula reconstituted with fluoridated water vs. an infant who is breast fed), health (for example, patients with kidney problems vs. people with normal kidney function), or other confounding factors.

In this section we rely heavily on several comprehensive review studies. Notably, we frequently cite the 2006 National Research Council (NRC) report by the Committee on Fluoride in Drinking Water, *Fluoride in Drinking Water: A Scientific Review of EPA's Standards*. Although the purpose of this well-researched report was to determine if the Environmental Protection Agency's drinking water standard of 4 ppm maximum allowable concentration for fluoride protects the public from harmful effects of fluoride, the report also provides valuable information about possible effects of drinking water containing lower concentrations of fluoride, such as those found in Golden Heart Utilities water. We supplemented information from this report with other comprehensive reviews and with refereed literature, particularly those papers published since the NRC report came out in 2006.

Dental Fluorosis

Dental fluorosis, a mottling and/or pitting of the tooth surface due to fluoride exposure, develops in children during tooth formation when exposure to excess fluoride leads to disruption of the crystalline-enamel structure. Fluoride has a strong affinity for developing pre-eruptive enamel, leading to integration of fluoride into the crystal lattice. Teeth appear to be most susceptible to fluorosis at early maturation stages, which vary for different tooth types. For example, central incisors of the upper jaw are most susceptible at age 15 to 24 months for boys and age 21 to 30 months for girls (Fluoride Recommendations Work Group, 2001).

Infants primarily ingesting formula reconstituted with fluoridated water, even at concentrations recommended for municipal systems, may receive doses of fluoride that could lead to more than mild fluorosis or possibly other adverse health effects from fluoride. For example, a recent study (Levy et

al., 2010) found that participants with fluorosis of permanent incisors (generally rated as mild) had significantly greater intake of fluoride from reconstituted powdered infant formula or other beverages with added water than those without fluorosis. The clinical implication suggested by the authors is that avoiding ingestion of formula or other drinks mixed with fluoridated water can reduce the likelihood of fluorosis.

Due to the increased risk of fluorosis for non-nursing infants, in 2007 the American Dental Association (ADA) made an interim recommendation that infant formula be reconstituted with water that is fluoride-free or containing low levels of fluoride (ADA, <http://www.ada.org/1767.aspx>). In January 2011, the ADA rescinded the interim recommendation and issued a new recommendation based on research by the ADA's Council on Scientific Affairs (Berg et al., 2011). The new recommendations "for infants who consume reconstituted infant formula as the main source of nutrition" are (1) "Continue use of liquid or powdered concentrate infant formulas reconstituted with optimally fluoridated drinking water while being cognizant of the potential risk for enamel fluorosis" and (2) "Use ready-to-feed formula or liquid or powdered concentrate formula reconstituted with water that is either fluoride-free or has low concentrations of fluoride when the potential risk for enamel fluorosis is a concern." These "evidence-based" recommendations were ranked by the ADA as being "based on lower levels of evidence" (ADA, http://ebd.ada.org/contentdocs/ADA_Evidence-based_Infant_Formula_Chairside_Guide.pdf).

The results of fluoride exposure on developing teeth range from mild discoloration to highly stained and pitted teeth, depending on the concentration of fluoride and to a certain degree the susceptibility of the individual (NRC, 2006; Fagin, 2008). Severe enamel fluorosis characterized by pitting results in teeth that are very susceptible to dental caries. Severe fluorosis is estimated to occur at a rate of about 10% among children drinking water at the current EPA maximum allowable fluoride concentration (4 ppm) (NRC, 2006). The incidence of severe dental fluorosis is near zero where fluoride in water is below 2 ppm (NRC, 2006). But fluoride ingestion at levels commonly used to fluoridate water (1 ppm) can lead to mild to moderate levels of fluorosis. In its mildest form, fluorosis leads to opaque areas on the teeth. Estimates in the literature on the incidence of fluorosis vary, but it can be expected that at least 30% of school-aged children who consume water with between 0.7 and 1.2 ppm fluoride will have very mild or more severe dental fluorosis (Heller et al., 1997). A more recent study reported that the incidence of fluorosis has increased since the 1980s, and an analysis of data from 1999 to 2004 found that the prevalence of dental fluorosis in adolescents aged 12 to 15 is 41% (Centers for Disease Control and Prevention, 2010b). This condition has not been linked to other adverse health effects (Fagin, 2008). However, even mild fluorosis is considered by some to be of cosmetic concern. Since fluorosis cannot be reversed, treatment requires costly cosmetic dentistry where teeth are coated to hide the effects.

For slightly older children (16 to 36 months), fluorosis risk increases with higher fluoridated toothpaste ingestion. To avoid fluorosis, it is recommended that ingestion of toothpaste should be reduced through parental supervision and using only a small smear of toothpaste when brushing (Levy et al., 2010).

There are challenges to determining the relationship between fluorosis and dental caries. One challenge is consistent diagnosis of mild dental fluorosis, which is subjectively rated using various rating scales. Another challenge is that there is some evidence that fluoride delays the eruption of permanent teeth, thus affecting studies comparing caries rates in children of different age groups

exposed to varying fluoride concentrations (NRC, 2006). A final challenge that affects all studies linking water fluoridation to both positive and negative health effects is that the concentration in water can lead to widely different individual doses, depending on water consumption and exposure to other sources of fluoride.

Bone Effects and Skeletal Fluorosis

Since about 50% of ingested fluoride not excreted is deposited in bone, and 99% of the fluoride in a human body is contained in the skeleton (cited in Bassin et al., 2006), a number of studies have examined the effects of fluoride on bone. Ingestion of fluoride at very high concentrations results in thickened bone and can lead to bone deformities (skeletal fluorosis). Debilitating skeletal fluorosis is rare in the U.S. (NRC, 2006), and there is no evidence that ingestion of fluoride at levels used to treat drinking water leads to significant skeletal fluorosis. However, exposure to fluoride at relatively high concentrations has been linked to an increased risk of bone fractures because fluoride incorporation, while increasing bone density, also leads to a decrease in bone strength. The Committee on Fluoride in Drinking Water (NRC, 2006) found that people consuming drinking water containing 4 ppm or greater fluoride over their lifetime had an increased risk of bone fractures. However, they could not reach a conclusion about the relationship between consumption of water containing lower concentrations of fluoride and risk of bone fractures.

There are a number of studies on the relationship between fluoride consumption and bone fractures. Interestingly, since fluoride is known to increase bone density, treating patients at risk of osteoporosis with fluoride was once a clinically accepted strategy. However, studies suggesting, at best, no protection against fractures and a high level of side effects have led to a decline in fluoride treatment (Vestergaard et al., 2008). Studies are confounded by factors that include the possibility that fluoride may affect different bones differently (NRC, 2006). Two comprehensive reviews of the literature have concluded that there is no clear association between hip fractures (either positive or negative) or osteoporosis and water fluoridation (McDonagh et al., 2000; Yeung, 2008). Overall, the data suggesting an increased risk of bone fractures in populations drinking fluoridated water in the concentration range recommended for drinking water are not conclusive.

Cancer

The potential link between fluoride and cancer, most specifically osteosarcoma, is an area of recent controversy. Since fluoride incorporates readily into developing bone and increases the proliferation of osteoblasts, it has been hypothesized that there could be a link between fluoride and osteosarcoma. Published studies have drawn different conclusions about whether or not there is a relationship, in part complicated by the relative rarity of this type of cancer. But several studies have indicated a potential link, including a 1990 study conducted by the U.S. National Toxicology Program (Bucher et al., 1991). In this study, where rats were exposed to high levels of fluoride, there appeared to be a relationship between osteosarcoma frequency in male rats and the level of exposure to fluoride.

A more recent paper by Bassin et al. (2006) on humans used a case-control approach to assess the patient history of 103 patients with osteosarcoma matched with 215 controls. The authors concluded "our exploratory analysis found an association between fluoride exposure in drinking water during

childhood and the incidence of osteosarcoma among males but not consistently among females.” Interestingly, Dr. Bassin’s PhD supervisor, Chester Douglass, challenged the data in a rebuttal published in the same issue of the journal that the Bassin et al. paper appeared (Douglass and Joshipura, 2006). In that rebuttal he suggested that a paper was forthcoming with more extensive data that would show no link. To date, no such paper has been published. Our task force committee chair contacted Dr. Douglass by e-mail to try to get more information. Dr. Douglass was not forthcoming with information, only stating that: “A paper has been submitted to a scientific journal for publication. Thank you for your interest.” A literature search in late November 2010 did not find a publication on this topic by Dr. Douglass.

While the Bassin paper is intriguing, the authors admit that the results are in contrast to several other case control studies (see Bassin et al., 2006) that found no link between fluoride consumption and osteosarcoma. They were careful to outline limitations to their preliminary study, including lack of data on actual consumption of fluoride by their subjects, lack of data on other potential unidentified factors, and selection bias. The authors cautiously referred to their study as “exploratory” and urged that “further research is required to confirm or refute this observation.” Unfortunately, as of 2010 it appears that no more comprehensive studies have been published that might shed light on a possible link between fluoride consumption and osteosarcoma. We find that although there may be such a link, the data published to date suggesting a link are limited and published studies are conflicting in their conclusions. This conclusion is supported by comprehensive reviews of the literature (Yeung, 2008; McDonagh et al., 2000), which both concluded that there is no clear association between water fluoridation and overall cancer incidence and mortality.

Other Effects

Endocrine Effects: Fluoride exposure has been shown to affect some endocrine glands and may function as an endocrine disruptor. Although fluoride is generally not thought to accumulate in soft tissues, there is evidence that it may accumulate in the thyroid where exposure can lead to decreased thyroid function. According to the NRC’s *Fluoride in Drinking Water* report (2006), many effects of low-dose fluoride exposure may be “subclinical effects, meaning there are no adverse health effects.” However, they also point out that “borderline hormonal imbalances” might lead to an increased risk of adverse health effects. Their report concluded that studies to date on the effects of fluoride on endocrine function have limitations and that further research is needed to explore the possible connections between fluoride, particularly at low doses, and endocrine function. Additional research is important since there is some indication that concentrations of fluoride in drinking water of 4 ppm or less may affect endocrine function in “young children” or in “individuals with high water intake.”

Neurotoxicity and Neurobehavioral Effects: A number of studies have reported changes to the nervous system following fluoride exposure that could lead to functional effects. Of the neurobehavioral studies, epidemiological studies suggesting a link between fluoride exposure and cognitive abilities are of particular interest. For example, several Chinese studies have consistently reported lower IQs in children drinking water containing 2.5 to 4 ppm fluoride (e.g., see NRC, 2006). The mechanism of the action of fluoride on IQ is not clear (Tang et al., 2008) but could be related to changes in membrane lipids in brain cells or to effects of fluoride on thyroid activity. It is unclear how the Chinese studies relate to U.S. populations, since U.S. populations are generally

exposed to drinking water with less than 2.5 ppm and there may be other confounding factors affecting the Chinese communities studied. Although the NRC's Fluoride in Drinking Water committee (2006) did not include neurological effects on their list of adverse effects not protected by the current EPA maximum allowable concentration for fluoride in drinking water, they did strongly advise that because of the "consistency of the results" in studies, such as those conducted on Chinese populations, additional research on the effects of fluoride on intelligence and on other neurological processes is warranted. A literature search conducted in December 2010 did not find published results that provide new information. It appears that there is reasonably good evidence that fluoride in drinking water at concentrations above 4 ppm may have neurological effects, including an effect on cognitive abilities. But the effects, if any, at lower concentrations of fluoride are not clear.

Effects on Other Organ Systems: Other systems that may be affected by fluoride exposure include the gastrointestinal system, kidneys, liver, and immune system. The NRC committee (2006) found a lack of well-documented studies on humans exposed to drinking water at 4 ppm or less for all of these systems. They concluded that the risk of adverse effects was likely to be low for most individuals drinking water with fluoride at 4 ppm but that there is a possibility of adverse effects in particular subpopulations such as those with renal impairment. In an apparent response to the possibility of an increased risk of adverse health effects for renal-impaired patients, the National Kidney Foundation recently changed its position on fluoridated water from "safe" to "takes no position" and "further research is needed" (www.kidney.org/atoz/pdf/Fluoride_Intake_in_CKD.pdf).

Findings

1. The problematic relationship between fluoride concentration in drinking water and "fluoride dose" (due to varying amounts of water consumed by individuals and to other sources of ingested fluoride) severely complicates attempts to determine both health risks and benefits associated with 1 ppm fluoride in drinking water. In particular, at this time commonly available foods and beverages range from high (greater than 2 ppm) to negligible fluoride content, and fluoridated toothpaste is variably swallowed. We believe that these factors grossly complicate interpretation of drinking water studies and explain why the numerous studies conducted have come to a variety of different conclusions.
2. The only commonly agreed-upon adverse effect related to drinking water with 1 ppm fluoride is mild dental fluorosis. Although debate continues concerning the quality of the studies, there are a large number that report deleterious effects from elevated fluoride in drinking water. On the other hand, numerous communities around the world use drinking water with natural fluoride concentrations of 1 ppm with no obvious ill effects, aside from mild dental fluorosis.
3. A fluoride concentration in water of 4 ppm is not protective for several adverse effects, including bone effects. That means that at best there is only a safety factor of about six for persons drinking Fairbanks water fluoridated to 0.7 ppm.
4. Although there may be a link between fluoride and osteosarcoma, the data published to date suggesting a link are limited and published studies are conflicting in their conclusions.
5. Fluoridated water is not recommended for all consumers. Recently several organizations have expressed concern about using fluoridated water to reconstitute infant formula. Consequently, the American Dental Association has recommended that parents of infants who primarily consume

reconstituted formula consult with their health care providers about the potential risks of using fluoridated water to make up infant formula. Despite those recommendations and cautions, pediatricians in the Fairbanks area (polled by committee member Dr. Medford) were not aware of these recommendations. The National Kidney Foundation has also changed its position on fluoridated water from “safe” to “takes no position” and “further research is needed.”

6. Research on possible adverse effects of drinking fluoridated water (at concentrations less than 2 ppm) on the endocrine glands, nervous system, or other organ systems has showed mixed results, with many studies showing no effects. However, studies involving extensive review of the literature (e.g., McDonagh et al., 2000; NRC, 2006) recommend that more high-quality research is warranted.

Chapter 8

Socioeconomic Issues

One of the public policy arguments put forward for fluoridation of public water supplies has been that it reduces disparities in dental health among populations. The argument goes that, if fluoridated water reduces the incidence of caries, it seems reasonable that the availability of fluoridated water for an entire community should provide particular benefit to those with the greatest risk of developing caries. This argument has been strongly put forward by professional organizations and government officials, including former U.S. Surgeon General David Satcher who “noted that water fluoridation is a powerful strategy in efforts to eliminate health disparities among populations” (ADA, 2005, p. 46).

For decades it has been noted that members of lower socioeconomic categories have significantly higher rates of caries than those who are more fortunate (Kozol, 1992; CDC, 2010a), so fluoridation should provide particularly valuable benefits to these groups. The refereed literature contains numerous reports that support (for example, Riley et al., 1999; Jones and Worthington, 2000) and refute this proposal (for example, Bradnock et al., 1984; Carmichael et al., 1989). McDonough et al. (2000) could reach no clear consensus on whether this public policy argument is valid, and shortly thereafter Cohen and Locker (2001) concluded that there is “little evidence that water fluoridation has reduced social inequalities in dental health” (p. 579). However, the most recent reviews of the matter tend to be guardedly positive (Cheng et al., 2007; Pizzo et al., 2007; Parnell et al., 2009; Newbrun, 2010). Newbrun’s review provides a good example of the dilemma. It cites evidence in support of the proposition but concludes by stating, “whether fluoridation reduces disparities in caries is a continuing research question.”

Arguments that members of lower socioeconomic groups disproportionately benefit from fluoridation of public water supplies raise questions about the existence of evidence that these groups also bear elevated risk of adverse effects from consuming fluoridated water. While the task force could find no good evidence on this topic, it does note that there is documentation that breast-feeding rates among mothers from lower socioeconomic groups are lower than those of their more affluent counterparts (Scanlon et al., 2010). Thus the task force’s concerns about the exposure of formula-fed infants to fluoride (see Chapter 5) are particularly directed toward those from lower socioeconomic groups.

Finding

Although claims are made both that the detriments and the benefits of fluoridated water are greater for those in lower socioeconomic status, documentation of this is not conclusive.

Chapter 9

Cost

The proponents of water fluoridation continue to tout its cost effectiveness. For example, both the Centers for Disease Control and Prevention (CDC, 2010a) and the American Dental Association (ADA, 2005) claim that the fluoridation of public water supplies in the United States costs between approximately \$0.50 and \$3.00 per person per year and provides something on the order of \$40 per person in annual benefits (decreased costs of dental care) for every dollar invested. However, both costs and benefits are very difficult to identify and quantify in any generally agreed upon and reliable way, so there is widespread disagreement about the legitimacy of any of these estimates.

In Fairbanks, the only clearly quantifiable cost of the water fluoridation program is the annual GHU expenditure for sodium fluorosilicate, which is \$10,000 to \$12,000 per year. The additional indirect costs to GHU for handling the material, adding it to the water, and monitoring the concentration of fluoride in the distributed water are difficult to estimate but are probably negligible in that these duties are incorporated into the work schedules of employees who dedicate the majority of their time and effort to other responsibilities. Similarly, while there are real costs associated with the purchase, operation, and maintenance of equipment used in the fluoridation process, those costs have never been documented but are probably modest.

If GHU discontinues its fluoridation process, it will have to adjust its protocol for conditioning the distributed water. While the task force did not investigate the projected costs of the required changes (mostly focused on maintenance of an appropriate pH), it seems likely that they will not be significant.

No attempts have been made to quantify indirect medical and dental costs or benefits resulting from the fluoridation of Fairbanks water.

Finding

There is little in the way of reliable data that can be used to estimate the cost of fluoridating Fairbanks' water or the net savings or costs associated with discontinuing the existing fluoridation process.

References

- Alaska Department of Environmental Conservation (2010). "07-01-2010 Fluoride Information Request (Raw Water)." Online at www.ci.fairbanks.ak.us/boardscommissions/fluoridetaskforce/fluoridetaskforceexposure.php.
- American Dental Association (ADA) (2005). *Fluoridation Facts*. Chicago: ADA.
- Armfield, J. M. (2007). When Public Action Undermines Public Health: A Critical Examination of Anifluoridationist Literature. *Australia and New Zealand Health Policy* 4:25ff.
- Agency for Toxic Substances and Disease Registry (ATSDR). Online at <http://www.atsdr.cdc.gov/mrls/index.html>. Accessed March 14, 2011.
- Bassin, E. B., Wypij, D., Davis, R. B., and Mittleman, M. A. (2006). Age-specific Fluoride Exposure in Drinking Water and Osteosarcoma (United States). *Cancer Causes Control* 17:421–428. DOI 10.1007/s10552-005-0500-6.
- Berg, J., C. Gerweck, P. P. Hujoel, R. King, D. M. Krol, J. Kumar, S. Levy, H. Pollick, G. M. Whitford, S. Strock, K. Aravamudhan, J. Frantsve-Hawley, and D. M. Meyer (2011). Evidence-based clinical recommendations regarding fluoride intake from reconstituted infant formula and enamel fluorosis. *J. Am. Dent. Assoc.* 142:79–87.
- Bethke, C. M. (1996). *Geochemical Reaction Modeling*. New York: Oxford Press.
- Block, L. E. (1986). Antifluoridationists Persist: The Constitutional Basis for Fluoridation. *J. Public Health Dent.* 46(4):188–198.
- Bradnock, G., Marchment, M., and Anderson, R. (1984). Social Background, Fluoridation and Caries Experience in 5 Year Old Population. *Br. Dent. J.* 156:127–131.
- British Fluoridation Society (2010). *One in a Million—The Facts About Water Fluoridation* (2nd ed.). Manchester, UK: British Fluoridation Society. Online at www.bfsweb.org/onemillion/onemillion.htm.
- Bryson, C. (2004). *The Fluoride Deception*. New York: Severn Stories Press.
- Bucher, J. R., Hejtmancik, M. R., Toft, J. D. II, Persing, R. L., Eustis, S. L., and Haseman, J. K. (1991). Results and Conclusions of the National Toxicology Program's Rodent Carcinogenicity Studies with Sodium Fluoride. *Int. J. Cancer* 48:733–737.
- Carmichael, C., Rugg-Gunn, A., and Ferrell, R. (1989). The Relationship Between Fluoridation, Social Class and Caries Experience in 5 Year Old Children in Newcastle and Northumberland in 1987. *Br. Dent. J.* 167:57–61.
- Centers for Disease Control and Prevention (CDC) (2010a). Community Water Fluoridation: Cost Savings of Community Water Fluoridation. Online at www.cdc.gov/fluoridation/fact_sheets/cost.htm.
- Centers for Disease Control and Prevention (CDC) (2010b). Prevalence and Severity of Dental Fluorosis in the United States, 1999–2004. NCHS Data Brief No. 53. Online at www.cdc.gov/nchs/data/databriefs/db53.pdf.
- Centers for Disease Control and Prevention (CDC) (2001). Recommendations for Using Fluoride to Prevent and Control Dental Caries in the United States. *Morbidity and Mortality Weekly Report* 50(RR14):1–42.
- Centers for Disease Control and Prevention (CDC) (1999a). Fluoridation of Drinking Water to Prevent Dental Caries. *Morbidity and Mortality Weekly Report* 48(41):933–940.
- Centers for Disease Control and Prevention (CDC) (1999b). Water Fluoridation and Costs of Medicaid Treatment for Dental Decay—Louisiana, 1995–6. *Morbidity and Mortality Weekly Report* 48(34):753–757.
- Cheng, K. K., Chalmers, K., and Sheldon, T. A. (2007). Adding Fluoride to Water Supplies. *BMJ* 335:699–702.
- Cohen, H., and Locker, D. (2001). The Science and Ethics of Water Fluoridation. *J. Can. Dent. Assoc.* 67(10):578–580.
- Colquhoun, J. (1998). Why I Changed My Mind about Water Fluoridation. *Fluoride* 31(2):103–118.

- Committee to Coordinate Environmental Health and Related Programs, United States Public Health Service (1991). Review of Fluoride: Benefits and Risks: Report of the Ad Hoc Subcommittee on Fluoride of the Committee to Coordinate Environmental Health and Related Programs, U.S. Public Health Service, Department of Health and Human Services. Online at <http://health.gov/environment/ReviewofFluoride/>.
- Connett, P., Beck, J., and Micklem, H. S. (2010). *The Case Against Fluoride*. White River Junction, VT: Chelsea Green Publishing.
- Cox, G. J., Matuschak, M. C., Dixon, S. F., Dodds, M. L., and Walker, W. E. (1939). Experimental Dental Caries IV. Fluorine and Its Relation to Dental Caries. *J. Dent. Res.* 57:481–490.
- Cross, D. W., and Carton, R. J. (2003). Fluoridation: A Violation of Medical Ethics and Human Rights. *Int. J. Occup. & Environ. Health* 9(1):24–29.
- Dean, H. T., Arnold, F. A., and Elvove, E. (1942). Domestic Water and Dental Caries. *Public Health Report* 57:1155–1179.
- Dean, H. T., Arnold Jr., F. A., and Elvove, E. (1941). Domestic Water and Dental Caries. *Public Health Report* 56:761–792.
- Diesendorf, Mark (1986). The Mystery of Declining Tooth Decay. *Nature*, July 10, pp. 125–29.
- Ditmyer, Marcia, Dounis, Georgia, Mobley, Connie, and Schwarz, Eli (2010). A Case-control Study of Determinants for High and Low Dental Caries Prevalence in Nevada Youth. *BMC Oral Health* 10:24. Online at www.biomedcentral.com/1472-6831/10/24.
- Douglass, C. W., and Joshipura, K. (2006). Caution Needed in Fluoride and Osteosarcoma Study. *Cancer Causes Control* 17:481–482.
- Environmental Protection Agency (EPA) (2000). Estimated Per Capita Water Ingestion in the United States (EPA 822-R-00-008). Online at water.epa.gov/action/.../2005_05_06_criteria_drinking_percapita_Text.pdf.
- Environmental Protection Agency (EPA) (2004). Estimated Per Capita Water Ingestion and Body Weight in the United States—An Update. (EPA-822-R-00-001). Online at www.epa.gov/waterscience/criteria/drinking/percapita/2004.pdf.
- Environmental Protection Agency (EPA) (2010). Fluorine (Soluble Fluoride) (CASRN 7782-41-4). Online at www.epa.gov/iris/subst/0053.htm.
- Environmental Protection Agency (EPA) (2011). EPA proposes to withdraw sulfuryl fluoride tolerances (<http://www.epa.gov/pesticides/sulfuryl-fluoride/evaluations.html>)
- Fagin, D. (2008). Second Thoughts About Fluoride. *Sci. Am.* 298(1):74–81.
- Fawell, J., Bailey, K., Chilton, J., Dahi, E., Fewtrell, L., and Magara, Y. (2006). *Fluoride in Drinking-water*. World Health Organization. London: IWA Publishing.
- Featherstone, J. D. B. (2000). The Science and Practice of Caries Prevention. *J. Am. Dent. Assoc.* 131(7):887–899.
- Federal-Provincial-Territorial Committee on Drinking Water (2009). Fluoride in Drinking Water—Draft For Public Consultation. Online at www.hc-sc.gc.ca/ewh-semt/consult/_2009/fluoride-fluorure/draft-ebauche-eng.php.
- Fejerskov, O., Thylstrup, A., and Larsen, M. J. (1981). Rational Use of Fluoride in Caries Prevention: A Concept Based on Possible Cariostatic Mechanisms. *Acta Odontol Scand.* 39:241–249.
- Fluoride Recommendations Work Group (2001). Recommendations for Using Fluoride to Prevent and Control Dental Caries in the United States. *Morbidity and Mortality Weekly Report* 50 (RR14):1–42. Online at <http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5014a1.htm>.
- Fluoride Technical Study Group (2003). Report of the Fort Collins Fluoride Technical Study Group. Online at <http://www.healthdistrict.org/fluoridereport/FTSG.htm>.
- Freni, S. C. (1994). Exposure to High Fluoride Concentrations in Drinking Water is Associated with Decreased Birth Rates. *J. Toxicol. Environ. Health* 42:109–121.

- Graham, R. J., and Morin, P-J. (1999). Highlights in North American Litigation During the Twentieth Century on Artificial Fluoridation of Public Water Supplies. *J. Land Use and Environmental Law* 14(2):195-247.
- Griffin, S. O., Gooch, B. F., Lockwood, S. A., and Tomar, S. L. (2001). Quantifying the Diffused Benefit from Water Fluoridation in the United States; *Community Dent. Oral Epidemiol.* 29:120-129.
- Griffin, S. O., Regnier, E., Griffin, P. M., and Huntley, V. (2007). Effectiveness of Fluoride in Preventing Caries in Adults. *J. Dent. Res.* 86:410-415. DOI 10.1177/154405910708600504.
- Hayes, A. W., ed. (2008). *Principles and Methods of Toxicology* (5th ed.). New York: Informa Healthcare USA.
- Health Canada (2010). Fluoride and Human Health. Online at www.hc-sc.gc.ca/hl-vs/iyh-vsv/environ/fluor-eng.php#prov.
- Heller, K. E., Eklund, S. A., and Burt, B. A. (1997). Dental Caries and Dental Fluorosis at Varying Water Fluoride Concentrations. *J. Public Health Dentistry* 57:136-143.
- Hellwing, E., and Lennon, A. M. (2004). Systemic Versus Topical Fluoride. *Caries Res.* 38:258-262.
- Hirzy, J. W. (2000). Statement Before the Subcommittee on Wildlife, Fisheries and Drinking Water, United States Senate, June 29, 2000. Online at www.fluoridealert.org/HirzyTestimony.pdf.
- Hodge, H. C., and Smith, F. A. (1965). Biological Properties of Inorganic Fluorides. In *Fluorine Chemistry*, Simons, H. H., ed., pp. 1-42. New York: Academic Press.
- Iida, H., and Kumar, J. V. (2009). The Association Between Enamel Fluorosis and Dental Caries in U.S. Schoolchildren. *J. American Dental Association* 140:855-862.
- Institute of Medicine (1997). Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride. Report of the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Washington, DC: National Academies Press.
- Institute of Medicine (2000). *Dietary Reference Intakes*. Washington, DC: National Academies Press.
- Jones, C., and Worthington, H. (2000). Water Fluoridation, Poverty, and Tooth Decay in 12-year-old Children. *J. Dent.* 28:389-393.
- Jones, S., Burt, B. A., Petersen, P. E., and Lennon, M. A. (2005). The Effective Use of Fluorides in Public Health. *Bull. World Health Organization* 83(9):670-676.
- Juneau Fluoride Study Commission (2006). Report to Assembly of the City and Borough of Juneau, July 11, 2006. Online at www.ci.fairbanks.ak.us/boardscommissions/fluoridetaskforce/fluoridetaskforceotherreferencesJuneaudocs.php.
- Kalsbeek, H., Kwant, J. W., Groeneveld, J., Backer Dirks, J., van Eck, A.M. J., and Theuns, H. M. (1993). Caries Experience of 15-Year Old Children in the Netherlands After Discontinuation of Water Fluoridation. *Caries Res.* 27:201-205.
- Komárek, Arnošt, Lesaffre, Emmanuel, Härkänen, Tommi, Declerck, Dominique, and Virtanen, Jorma I. (2005). A Bayesian Analysis of Multivariate Doubly-Interval-Censored Dental Data. *Biostat* 6(1):145-155. DOI 10.1093/biostatistics/kxh023.
- Kozol, J. (1992). *Savage Inequalities: Children in America's Schools*. New York: Crown Publishers.
- Künzel, W., Fischer, T., Lorenz, R., and Brühmann, S. (2000). Decline of Caries Prevalence After the Cessation of Water Fluoridation in the Former East Germany. *Community Dent. Oral Epidemiol.* 28:382-9.
- Kurtio, P., Gustavsson, N., Vartaninen, T., and Pekkanen, J. (1999). Exposure to Natural Fluoride in Well Water and Hip Fracture. A Cohort Analysis in Finland. *Am. J. Epidemiol.* 150:817-824.
- Lalumandier, J. A., and Ayers, L. W. (2000). Fluoride and Bacterial Content of Bottled Water Versus Tap Water up to 1 ppm. *Arch. Fam. Med.* 9:246-250.
- Levy, S. M., and Zarei-M, Z. (1991). Evaluation of Fluoride Exposures in Children. *J. Dent. Child.* 58(6):467-473.
- Levy, S. M., Broffitt, B., Marshall, T. A., Eichenberger-Gilmore, J. M., and Warren, J. J. (2010). Associations Between Fluorosis of Permanent Incisors and Fluoride Intake from Infant Formula, Other Dietary Sources and Dentifrice During Early Childhood. *J. American Dental Association* 141:1190-1201.

- Limeback, H. (2000). Why I Am Now Officially Opposed to Adding Fluoride to Drinking Water. Online at www.fluoridealert.org/limeback.htm.
- Macek, M. D., Matte, T. D., Sinks, T., and Malvitz, D. M. (2006). Blood Lead Concentrations in Children and Method of Fluoridation in the United States, 1988–1994. *Environ. Health Perspect.* 114(1):130–13.
- MacFayden, E. E., McNee, S. G., and Weeman, D. A. (1982). Fluoride Content of Some Bottled Spring Waters. *Br. Dent. J.* 53:423–424.
- Masters, R. D., and Coplan, M. J. (1999). Water Treatment with Silicofluorides and Lead Toxicity. *Int. J. Environ. Stud.* 56:435–449.
- Masters, R. D., Coplan, M. J., Hone, B. T., and Dykes, J. E. (2000). Association of Silicofluoride Treated Water with Elevated Blood Lead. *NeuroToxicity* 21(6):1091–1100.
- McDonough, M., Whiting, P., Bradley, M., Cooper, J., Sutton, A., Chestnutt, I., Misso, K., Wilson, P., Treasure, E., and Kleinjen, J. (2000). *A Systematic Review of Public Water Fluoridation*. National Health Service Centers for Reviews and Dissemination. York, UK: University of York. DOI 10.1136/bmj.321.7265.855.
- McNally, M., and Downie, J. (2000). The Ethics of Water Fluoridation. *J. Can. Dent. Assoc.* 66(11):592–593.
- Mueller, S. H. (2002). A Geochemical Characterization of Groundwater Near Fairbanks, Alaska, with Emphasis on Arsenic Hydrogeochemistry. MSc. thesis, University of Colorado, Boulder.
- National Health and Medical Research Council (Australia) (2007). A Systematic Review of the Efficacy and Safety of Fluoridation. Online at www.nhmrc.gov.au/publications/synopses/eh41syn.htm
- National Research Council (NRC) (1993). *Health Effects of Ingested Fluoride*. Washington, DC: National Academies Press.
- National Research Council (NRC) (2006). *Fluoride in Drinking Water: A Scientific Review of EPA's Standards*. Washington, DC: National Academies Press. Online at www.nap.edu/catalog/11571.html.
- Newbrun, E. (2010). What We Know and Do Not Know About Fluoride. *J. Pub. Health And Dentistry* pp. 1–7. (Invited review presented at National Oral Health Conference, Portland, OR, April 21, 2009). DOI 10.1111/j.1752-7325.2010.00171.x.
- Ophaug, R. H., Singer, L., and Harland, B. F. (1985). Dietary Intake of 6-months and 2-year-old Children in Four Dietary Regions of the United States. *J. Clin. Nutr.* 42(4):701–707.
- Office of Pesticide Programs (OPP) (2011). Sulfuryl Fluoride—Revised Human Health Risk Assessment for Fluoride to Incorporate New Hazard and Exposure Information. Online at www.regulations.gov/#documentDetail;D=EPA-HQ-OPP-2005-0174-0113.
- Osmunson, W. (2010a). Letter #1 to Fairbanks Fluoride Task Force. “03-02-2010 Osmunson Letter to FTC—Fluoridation, Drugs, and Human Subject Research.” Online at www.ci.fairbanks.ak.us/boardscommissions/fluoridetaskforce/fluoridetaskforceotherreferences.php.
- Osmunson, W. (2010b). Letter #4 Effectiveness of Fluoridation, to Fairbanks Fluoridation Task Force. “03-13-2010 Dr. Osmunson Letter—Effectiveness of Fluoridation.” Online at <http://www.ci.fairbanks.ak.us/boardscommissions/fluoridetaskforce/fluoridetaskforceefficacy.php>.
- Parnell, C., Whelton, H., and O’Mullane, D. (2009). Water Fluoridation. *Eur. Arch. Paediatr. Dent.* Sep; 10(3):141–8.
- Peterson, P. E. (2003). *Fluoride in Drinking Water*. The World Oral Health Report 2003: Continuous Improvement of Oral Health in the 21st Century—The Approach of the WHO Global Oral Health Programme (WHO/NMH/NPH/ORH/03.2). Geneva: WHO Global Oral Health Program. Online at http://www.who.int/oral_health/publications/report03/en/.
- Pizzo, Giuseppe, Piscopo, Maria R., Pizzo, Ignazio, and Giuliana, Giovanna (2007). Community Water Fluoridation and Caries Prevention: A Critical Review. *Clin. Oral. Invest.* 11:189–193. DOI 10.1007/s00784-007-0111-6.
- Pratt, Jr., E., Rawson, R. D., and Rubin, M. (2002). Fluoridation at Fifty: What Have We Learned? *J. Law Med. Ethics* Fall 30(3) Suppl:117–121.

- Riley, J. C., Lennon, M. A., and Ellwood, R. P. (1999). The Effect of Water Fluoridation and Social Inequalities on Dental Caries in 5-year-old Children. *Int. J. Epidemiol.* 28(2):300–305. DOI 10.1093/ije/28.2.300.
- Rojas-Sanchez, F., Kelly, S. A., Drake, K. M., Eckert, G. J., Stookey, G. K., and Dunipace, A. J. (1999). Fluoride Intake From Foods, Beverages and Dentifrice by Young Children in Communities With Negligibly and Optimally Fluoridated Water. A Pilot Study. *Community Dent. Oral Epidemiol.* 27(4):288–297.
- Scanlon, K. S., Grummer-Strawn, L., Chen, J., Molinari, N., and Perrine, C. G. (2010). Racial and Ethnic Differences in Breastfeeding Initiation and Duration by State—National Immunization Survey, United States, 2004–2008. *Morbidity and Mortality Weekly* 59(11):327–334.
- Scott, T. (1983). *Concise Encyclopedia of Biochemistry*. Berlin: Walter DeGruyter & Co.
- Seppa, L., Karkkainen, S., and Hausen, H. (2000). Caries Trends 1992–1998 in Two Low-Fluoride Finnish Towns Formerly With and Without Fluoridation. *Caries Res.* 34:462–468.
- Sigfried, T. (2010). Odds Are, It's Wrong. *Science News* 177(7):26ff.
- Sutton, P. (1960). *Fluoridation: Errors and Omissions in Experimental Trials* (2nd Ed.). Melbourne: Melbourne University Press.
- Tang, Qin-qing, Du, Jun, Ma, Heng-hui, Jiang, Shao-jun, and Zhou, Xiao-jun (2008). Fluoride and Children's Intelligence: A Meta-analysis. *Biol. Trace Elem. Res.* DOI 10.1007/s12011-008-8204-x. Humana Press Inc.
- Taubes, G. (2006). Epidemiology Faces its Limits. *Science* 269:164–169.
- Thiessen, K. M. (2006). Water Fluoridation: Suggested Issues for Consideration. Unpublished manuscript, 6 pp., “05-27-2010 Thiessen-EPA Comments.” Online at www.ci.fairbanks.ak.us/boardscommissions/fluoridetaskforce/fluoridetaskforcereviews.php.
- Thiessen, K. M. (2009a). Comments on Fluoride in Drinking Water. Unpublished manuscript, 15 pp., “11-27-2009 Thiessen-Water Fluoridation Suggested Issues for Consideration.” Online at www.ci.fairbanks.ak.us/boardscommissions/fluoridetaskforce/fluoridetaskforcetoxicityadverseeffects.php.
- Thiessen, K. M. (2009b). Comments in Response to Announcement of Chemicals Selected by OEHHHA for Consideration for Listing by the Carcinogen Identification Committee and Request for Relevant Information on the Carcinogenic Hazards of These Chemicals. Unpublished manuscript, 15 pp., “12-15-2009 Thiessen-Comments Responding to CA EPA Carcinogen Listings.” Online at www.ci.fairbanks.ak.us/boardscommissions/fluoridetaskforce/fluoridetaskforcereviews.php.
- Thiessen, K. M. (2010). Comments on the Need for Revision of the NPDWR for Fluoride. Unpublished manuscript, 22 pp., “15-27-2010 Thiessen-EPA comments.” Online at www.ci.fairbanks.ak.us/boardscommissions/fluoridetaskforce/fluoridetaskforcereviews.php.
- Truman, B., I, Gooch, B. F., Sulemana, I., Gift, H. C., Horowitz, A. M., Evans, C. A., Griffin, S. O., and Carande, K., V (2002). Reviews of Evidence on Interventions to Prevent Dental Caries, Oral and Pharyngeal Cancers, and Sports-Related Craniofacial Injuries (Structured abstract). *Amer. J. Preventive Medicine* 23:21–54.
- Turekian, K. K. (1969). The Oceans, Streams, and Atmosphere. In *Handbook of Geochemistry*, vol. 1, K. H. Wedepohl, ed., New York: Springer-Verlag.
- Urbansky, E. T. (2002). Fate of Fluorosilicate Drinking Water Additives. *Chem. Rev.* 102:2837–2854.
- U.S. Department of Agriculture (USDA) (2004). USDA National Fluoride Database of Selected Beverages and Foods—2004. Online at www.nal.usda.gov/fnic/foodcomp/Data/Fluoride/fluoride.pdf.
- U.S. Department of Health and Human Services (HHS) (2000). *Healthy People 2000. With Understanding and Improving Health and Objectives for Improving Health* (2nd ed.). Washington, DC: U.S. Government Printing Office.
- U.S. Geological Survey (USGS) (2001). Ground-Water Studies in Fairbanks, Alaska—A Better Understanding of Some of the United States' Highest Natural Arsenic Concentrations. (Fact Sheet FS-111-01). Online at <http://pubs.usgs.gov/fs/fs-0111-01/fs-0111-01.pdf>.

- U.S. Public Health Service (USPHS) (1991). Review of Fluoride: Benefits and Risks: Report of the Subcommittee on Fluoride of the EHPC. Washington, DC: Public Health Service, Department of Health and Human Services.
- Verplanck, P. I., Mueller, S. H., Youcha, E. K., Goldfarb, R. J., Sanzolone, R. F., McCleskey, R. B., Briggs, P. H., Roller, M., Adams, M., and Nordstrom, D. K. (2003). Chemical Analyses of Ground and Surface Waters, Ester Dome, Central, Alaska, 2000–2001. Open File Report 03-244. U.S. Geological Survey.
- Vestergaard, P., Jorgensen, N. R., Schwarz, P., and Mosekilde, L. (2008). Effects of Treatment with Fluoride on Bone Mineral Density and Fracture Risk: A Meta-analysis. *Osteoporosis Intern.* 19:257–268.
- Warren, J. J., Levy, S. M., Broffitt, B., Cavanaugh, J. E., Kanellis, M. J., and Weber-Gasparoni, K. (2009). Considerations on Optimal Fluoride Intake Using Dental Fluorosis and Dental Caries Outcomes—A Longitudinal Study. *J. Public Health Dent.* 69(2):111–115.
- Westendorf, J. (1975). The Kinetics of Acetylcholinesterase Inhibition and the Influence of Fluoride and Fluoride Complexes on the Permeability of Erythrocyte Membranes. Ph.D. dissertation, University of Hamburg, Hamburg, Germany.
- Whistler, B. J. (2007). *Alaska Oral Health Plan: 2008–2012*. Juneau, AK: Section of Women's, Children's and Family Health, Division of Public Health, Alaska Department of Health and Social Services.
- Whitford, G. M. (1996). *The Metabolism and Toxicity of Fluoride* (2nd Rev. ed.). Monographs in Oral Science, vol. 16. New York: Karger.
- Yeung, C. A. (2008). A Systematic Review of the Efficacy and Safety of Fluoridation. *Evid. Based Dent.* 9(2):39–43.
- Ziegelbecker R. (1998). Fluoridation in Europe. *Fluoride* 31(3):171–174.

Appendix A Resolution

Introduced By: Council Member Cleworth
Introduced: February 8, 2010

RESOLUTION NO. 4398

A RESOLUTION ESTABLISHING A TASK FORCE TO RESEARCH CURRENT POLICY REGARDING FLUORIDATION OF THE MUNICIPAL WATER SUPPLY.

WHEREAS, the health and security of Fairbanks citizens are a primary concern of the City Council; and

WHEREAS, the use of fluoride in the City's water supply was established in 1960 (FGC Sec. 82-1) as a way to enhance dental care; and

WHEREAS, this practice has raised questions regarding potential long-term effects caused by the use of fluoride; and

WHEREAS, it is advisable to periodically reanalyze this policy to make sure the potential benefits outweigh any potential side effects associated with fluoridation; and

WHEREAS, the amount of research available on this subject is voluminous and often extremely technical.

NOW, THEREFORE, BE IT RESOLVED, that a committee is formed consisting of the six individuals listed below to research documentation provided by both proponents and opponents of fluoridation through public hearings and to supplement this information with any other sources deemed appropriate. A final report along with analysis and recommendations will be presented to the City Council no later than early July. Legal notifications and assistance will be given by the City Clerk's office. The committee consists of individuals having extensive backgrounds in chemistry, biology, dentistry, and medicine, who have expressed a strong interest in objectively analyzing research regarding fluoridation.

Committee Chair: Dr. Paul Reichardt, former Provost, Dean, and Professor at UAF, with a Ph.D. in Organic Chemistry;

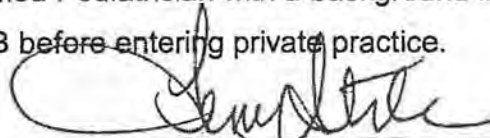
Dr. Dick Stolzberg: Professor Emeritus of Chemistry at UAF, with a Ph.D. in Chemistry, who has done extensive research in the field of analytical chemistry;

Dr. Rainer Newberry: Professor in Geochemistry, Mineralogy, and Economic Geology, with a Ph.D. in Economic Geology;

Dr. Bryce Taylor: Doctorate of Dental Surgery, formerly serving in public health with the TCC, now in private practice;

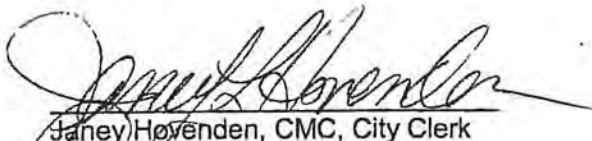
Dr. Joan Braddock: Most recently Dean of the College of Natural Science and Mathematics, with a Master's Degree in Microbiology and a Ph.D. in Oceanography;

Dr. Beth Medford: Board Certified Pediatrician with a background in biochemistry; formerly at Eielson AFB before entering private practice.


Terry Strle, City Mayor

AYES: Roberts, Eberhart, Gatewood, Bratcher, Cleworth, Stiver
NAYS: None
ABSTAIN:
ABSENT:
ADOPTED: February 08, 2010

ATTEST:


Janey Hovenden, CMC, City Clerk

APPROVED AS TO FORM:


Paul J. Ewers, City Attorney