

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.
Harlee S. Strauss, Ph. D.

**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

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.

Table of Contents

COVER LETTER TO THE SELECTMEN	2
FINDINGS, CONCLUSION, AND RECOMMENDATION OF THE NATICK FLUORIDATION STUDY COMMITTEE	3
Introduction	3
Findings	3
Conclusion	4
Recommendations	4
BACKGROUND AND HISTORY	9
History of the Fluoridation Issue in Natick	9
Appointment of the Natick Fluoridation Study Committee	9
Recommendation and Charter of the NFSC	10
Mechanism of Approach to be Taken	10
Charter of the Fluoridation Study Committee	11
Format of the Report	11
General Background of the Fluoridation Issue	12
References	14
QUESTION 1 AN ANALYSIS OF THE SIDE EFFECTS OF FLUORIDATION	15
The Beneficial Effects of Fluoridation	15
History of the Fluoridation Program in the United States	15
Features of the Fluoridation Program	16
Supporters of Community Water Fluoridation	16
Other Positive Effects of Fluoridation	16
References	17
Refutation -- Water Fluoridation today is at best only minimally effective	18
Table I - Fluoride Concentrations (ppm) in Food	19
Table II - Estimated Daily Fluoride Intake of Children (1)	19
Figure I - Decline in DMFS Index in 12-14 Year Olds (12)	20
Summary	22
References	23

Toxic Effects of Fluoridation and Inorganic Fluoride	24
The Acute Toxicity of Fluorine-Containing Materials	24
Summary and Conclusions on Acute Toxicity	24
References	25
Dental Fluorosis - An Undesirable Effect of Fluoridation	26
Table I - Dean's Survey(s) of Dental Fluorosis 1939-1940	26
When, why and how does Dental Fluorosis occur?	27
Why is the Prevalence of Dental Fluorosis Increasing?	27
Table II - Percentages of Children with Dental Fluorosis	28
The Anticipated Occurrence of Dental Fluorosis and Needed Corrective Measures	29
Summary and Conclusions	30
References:	31
Skeletal Fluorosis, Osteosclerosis & Related Disorders	32
Osteoporosis	32
Skeletal (Osteo)Fluorosis	32
Hip and other Fractures	33
Summary	34
References	35
Carcinogenicity	36
Animal Studies	36
Human epidemiology studies	38
Summary of cancer data	39
References	40
Metabolic & Enzymatic Effects	41
Background Material	41
Characteristics of Fluorine and Fluoride Ion	41
Structural Susceptibility of Biomolecules	42
Fluoride and Calcium Metabolism.	42
Summary and Conclusion	43
References	43
Hypersensitivity and "Allergic" Reactions	45
Summary	45
References	45
Central Nervous System: behavioral and IQ effects	47

Animal Toxicology Studies	47
Behavioral Changes in Humans Associated with Exposure to Fluoride as Adults	49
Decreases in IQ in humans exposed to fluoride in utero	49
Table I - IQ Ranges for Different Exposures to Fluoride (8)	50
Table II - Mean IQ scores for children in areas with different prevalence of fluorosis (9)	50
Table III - Distribution of child IQ scores from areas of differing fluorosis prevalence (9)	51
Biochemical studies of the brain	51
Summary and Conclusions Regarding CNS Effects	52
References	53
Reproductive and Developmental Toxicity (other than nervous system)	54
Reproductive Toxicity	54
Developmental Toxicity (other than neurotoxicity)	55
Summary	56
References	57
Lead Contamination	58
Lead Contamination	58
Table I - Fluoridation and Venous Blood Lead Levels in MA Children Aged 0-4 Yrs.	60
Summary	60
References	60
Non - Health Issues	61
Forced Medication/Freedom of Choice	61
Economic Issues of Fluoridation	61
Direct Process Costs	61
Cost-effectiveness of Fluoridation	62
Indirect Costs	62
Liability Ramifications	63
Environmental Impact	63
Political Ramifications of Referenda and Plebiscites	63
Summary of Non-Health Issues	64
References	65
QUESTION 2 The Establishment of an appropriate dosage	66
Discussion	66
Conclusion and Committee Response to Question 2	66

QUESTION 3 The advisability of a dental survey	67
Discussion	67
Conclusion and/or Committee Response to Question 3	67
QUESTION 4 Source of the survey and probable costs	68
Discussion	68
Conclusion and Committee Response to Question 4	68
APPENDIX A Source Materials	69
Materials Provided by the Board of Health	69
Materials Provided by Shirley Brown	70
Materials Provided by Myron Coplan	71
Materials Provided by NFSC Members	71
APPENDIX B The Difficulty of Research on Fluoridation	72
Reference	74
APPENDIX C Recommended Dosage of Fluoride	75
Table I Recommended Fluoride Supplementation (mg/day) for Children	75
Table II Estimated Tap Water and Fluoride Intake for Children	75
Reference	76
APPENDIX D The Measurement of Fluoridation Parameters	77
The Dental Caries Reduction Measurement	77
The Quantification of Dental Fluorosis	79
Table I Degrees of Dental Fluorosis	79
References	80
Members of the Committee	81
Curricula Vitae of the Committee Members	82
Curriculum of Benedict J. Gallo	82
Curriculum of Jason Kupperschmidt	84
Curriculum of Alfred J. Murray	86
Curriculum of Harlee S. Strauss	88
Curriculum of Norman R. Mancuso	89
ERRATA	91

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 Megoako 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 antifluoridationists 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)	

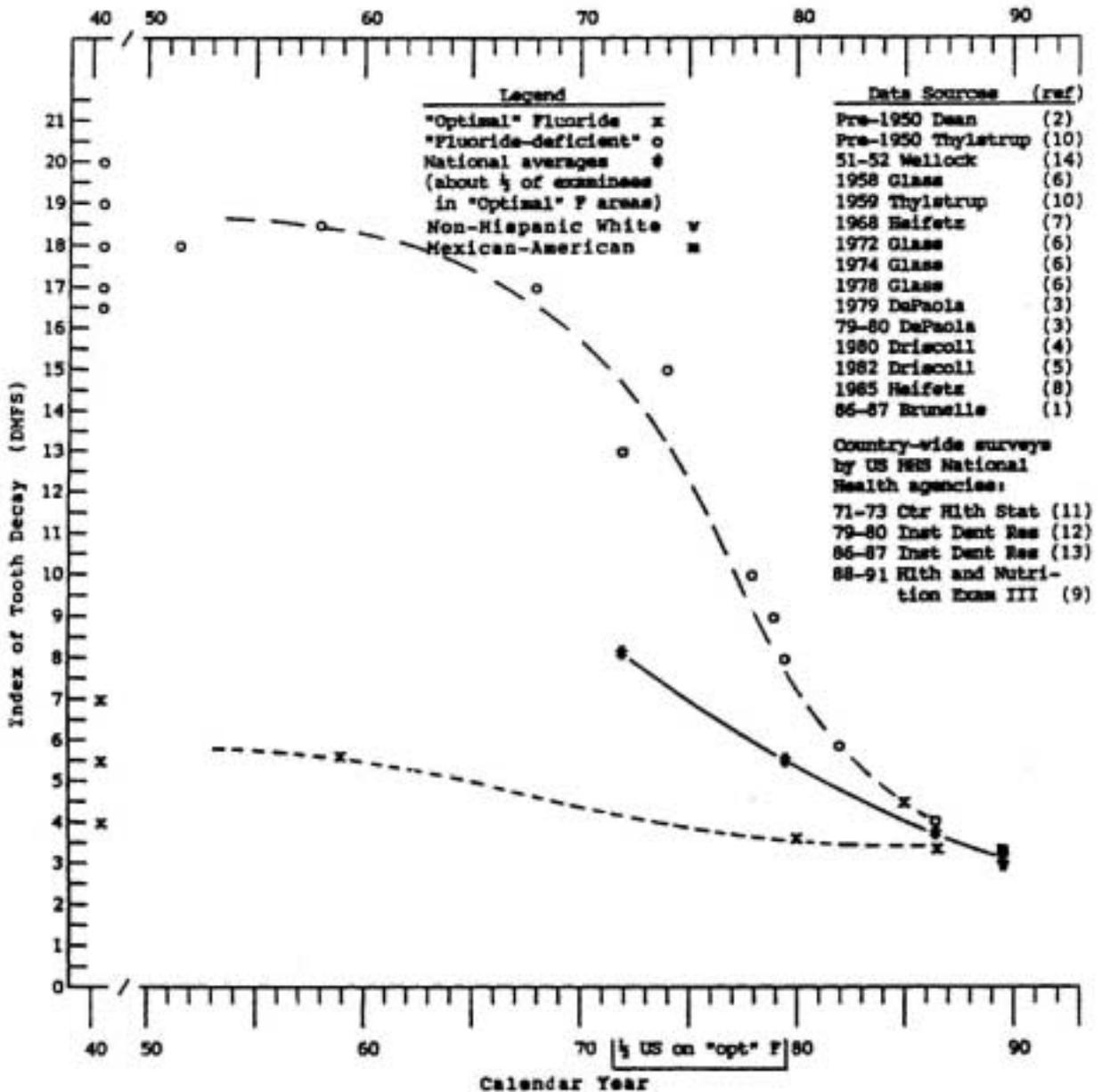
(a) Assumed that dentrifice used twice daily

(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 Benefit 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. Braunfels, TX	7-18	0.3	9	(14)
Cadillac, MI	6-12	0.0	12	(13)
New York State	12-17	0-0.3	4	(15)

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

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

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

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

The Anticipated Occurrence of Dental Fluorosis and Needed Corrective Measures

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

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

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

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

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

Summary and Conclusions

Excessive fluoride intake by children causes a toxic dental condition known as dental fluorosis which is marked by visible mottling/discoloring of tooth enamel, pitting of the enamel and disturbed tooth shape. Dental fluorosis occurs during early childhood while the baby and permanent teeth and tooth enamel are still being mineralized and before they erupt in the mouth. The severity of the dental fluorosis is directly proportional to fluoride ingested in excess of 0.03 mg to 0.07 mg fluoride/kg of body weight/day. The ultimate result is the increased porosity of the teeth and, in extreme cases, loss of afflicted teeth. The prevalence of dental fluorosis is increasing in communities that have water supplies that are "optimally fluoridated" and in those with fluoride deficient 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 Gylcolytic Enzymes by Fluoride Ions.*, Dtsc. 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-1.*, 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)

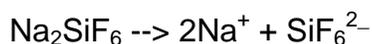
The health effects of lead can be as severe as coma and possible death at high blood levels (exceeding 80 ug/dL). Severe effects are unlikely to occur from drinking water. Low level lead exposure (as determined by blood lead levels greater than 10 ug/dL) is more likely to occur from drinking water. Low 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):

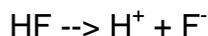
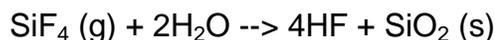
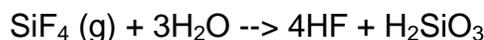
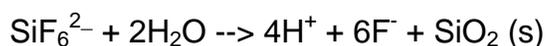
".....Under certain water-quality conditions, a small increase in the corrosivity of potable water that is already corrosive may be observed after treatment with alum, chlorine, fluorosilicic acid, or sodium fluorosilicate. This increase in corrosivity is caused by a depression of pH resulting from these 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.

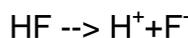
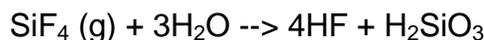
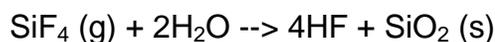
An important aspect of this corrosivity effect requires a consideration of the dissociation of sodium fluorosilicate, which takes place as follows: (1, 5)



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	H₂SiF₆

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 M CJ-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		<0.3	0.3-0.6	0.6
From	To	Units	mg/day	mg/day	mg/day
0	6	months	0	0	0
6	36	months	0.25	0	0
3	6	years	0.5	0.25	0
6	1	years	1.0	0.5	0

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

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

Table II Estimated Tap Water and Fluoride Intake for Children

Daily F & H 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. Szelag has evaluated the "dental age" of a number of children and has reported that not only is there evidence of delayed dentition but that the greater the dosage of fluoride taken up during development, the greater was the retardation of dentition. (3) Opponents of fluoridation have pointed out that when due consideration is given to this fact, the reported DMFT and DMFS scores fail to reflect a significant difference between fluoridated and unfluoridated areas. For example, Coplan states:

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

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

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

<u>Name</u>	<u>Home or Office Address</u>
*Benedict J. Gallo, Ph. D.	72 Washington Street, Natick, MA
Jason Kupperschmidt, B. S.	17 Greenwood Road, Natick, MA
Alfred J. Murray, M.S.T.	51 Crest Road, Framingham, MA
Harlee S. Strauss, Ph. D.	21 Bay State Road, Natick, MA
Norman R. Mancuso, Ph. D. Chairman of the Committee	24 Myrtle Street, Framingham, MA

*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.**

Norman R. Mancuso Associates
24 Myrtle Street Suite B Framingham, MA 01702
508-876-5696 (Voice) - 508-875-6003 (Voice & FAX)
Email: 72070.416@compuserve.com
nrmancuso@msn.com

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

Educational Background

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

Academic and Industrial Honors

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

Academic and Industrial Positions

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

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

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

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

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

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

Curriculum Vitae of Alfred J. Murray

Alfred J. Murray
Framingham, MA 01702
Email murray@meol.mass.edu

Education:

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

Massachusetts Teaching Certification:

- Secondary school principal, mathematics, chemistry and general science

Work Experience:

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

Publication:

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

Awards:

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

Curriculum Vitae of Harlee S. Strauss

Harlee S. Strauss, Ph.D.
H. Strauss Associates, Inc.
Natick, Massachusetts 01760
Tel: 508-651-8784
Fax: 508-655-5116
Email: HStrauss@aol.com

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"