IAOMT
International Academy of Oral Medicine and Toxicology

Policy position on ingested fluoride and fluoridation

Documentation and discussion

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Discussions of fluoride and fluoridation over the last 50-plus years by the general public or casual observer have often been complicated by the lack of discernment concerning the differences between effects

caused by systemic exposures and topical applications. Scientific discussions have been further complicated by providing undue weight to claims of effectiveness that have resulted in the abandonment of margins of safety that are essential to any toxicological profile and establishment of public policy.

In IAOMT's ongoing examination of the toxicological data on fluoride, the Academy has made several preliminary determinations over the last 18 years, each concluding that fluoride added to the public water supply, or prescribed as controlled-dose supplements, delivers no discernible health benefit, and causes a higher incidence of adverse health effects.

This current policy position by IAOMT confirms those earlier assessments and asserts that there is no discernible health benefit derived from ingested fluoride and that the preponderance of evidence shows that ingested fluoride in dosages now prevalent in public exposures aggravates existing illnesses, and causes a greater incidence of adverse health effects.
Ingested fluoride is hereby recognized as unsafe, and ineffective for the purposes of reducing tooth decay.

Effectiveness

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This analysis was achieved after exhaustive review of the peer-reviewed literature available. The Academy's previous conclusions of ineffectiveness differed with long-held conclusions by the American Dental Association and other trade associations based on tenets of scientific integrity and reliability in study design; however, as is noted below, the journals for the ADA and other trade associations have now revised their stance.

Upon examination of studies espoused by promoters of fluoridation as proof of effectiveness, the Academy was able to ascertain that there are no epidemiological studies indicating effectiveness of ingested fluoride that conform to scientific standards for broad-based or random selection, blinded examinations, and appropriate controls.

While this may appear to be a bold statement, the cover story of the July 2000 Journal of the American Dental Association (JADA) has confirmed for the rest of the dental community that the mechanism by which fluoride may have a meaningful impact on the reduction of dental caries is by topical application, not ingestion; thus supporting

the contention that the claimed study-results of large scale reduction in tooth decay are results obtained by study-design bias.

To further clarify, examination of the physiological method by which fluoride was assumed to affect the incidence of tooth decay reveals that the theory that fluoride incorporated into dental enamel during the formative process would cause the tooth to be more resistant to acid dissolution has been finally recognized as false.

In addition, the entire body of epidemiological studies used to support the contention that ingested fluoride reduces tooth decay neglected to control for essential factors. To name only a few: 1) the fact that greater than 85% of tooth decay occurs on pits and fissures of the tooth where fluoride has always been recognized to be ineffective (this is widely disseminated as support for the need for protective sealants); 2) the amount of water that the subjects consumed, or even whether the subjects drank fluoridated water; and 3) the variability in total exposure to fluoride from all other sources, meaning that at no point was the actual dosage of fluoride ever determined.

After fifty years of controversy, the test that eluded the spotlight on this subject is simple: a healthy bicuspid, extracted during preparation for orthodontics, is measured for fluoride concentration in the enamel; is immersed in a substance that will rapidly de-mineralize the tooth (Coca Cola will do fine); then is measured for its resistance to acid dissolution relative to the concentration of fluoride in the enamel.

The result? As described by Featherstone in JADA, July 2000, "The fluoride incorporated into the tooth developmentally -- meaning systemically, in the normal tooth mineral -- is insufficient to have a measurable effect on acid dissolution."

"Importantly, this means that fluoride incorporated during tooth mineral development at normal levels of 20 to 100 ppm (even in areas that have fluoridated drinking water or with the use of fluoride supplements) does not measurably alter the solubility of the mineral," writes Featherstone. "Even when the outer enamel has higher fluoride levels, such as 1000 ppm, it does not measurably withstand acid-induced dissolution any better than enamel with lower levels of fluoride."{Author’s parentheses}

More recently, the Center for Disease Control, which strongly supports water fluoridation, acknowledge in their long awaited report of August 17, 2001, **“The prevalence of dental caries in a population is not**

inversely related to the concentration of fluoride in enamel (37), and a higher concentration of enamel fluoride is not necessarily more efficacious in preventing dental caries (38).” {CDC references}

Concerning whether fluoride present in saliva due to ingestion will have any beneficial impact, CDC further states, “The concentration of fluoride in ductal saliva, as it is secreted from salivary glands, is low _ approximately 0.016 parts per million (ppm) in areas where drinking water is fluoridated and 0.006 in nonfluoridated area. **This concentration of fluoride is not likely to affect cariogenic activity.”**[1]

These results concur with the findings of the November, 1997 Canadian Dental Association Consensus Conference on prescription fluoride drops and tablets which found, “no reliable scientific evidence of significant dental benefit from ingested fluoride.”

In addition, carefully controlled studies have found increased tooth decay in vulnerable subsets of the population when exposed to fluoride in drinking water.[2] [3] [4]

Safety and Adverse Health Effects

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In our quest for more information on ingested fluoride the Academy sought the input from both sides of the fluoridation issue and ultimately heard from more than 13 different experts, both in favor and opposed to fluoridation, and in 1998 conducted an extensive scientific risk assessment on the health effects of ingested fluoride.

This conference resulted in the publication of a Public Health Goal (PHG) in the journal *Fluoride* that applied standard US EPA protocols to current studies. The risk assessment used four studies where daily dose could be calculated and applied the standard EPA Global 86 program to establish the minimum risk level of 0.0001 mg/L for ingested fluoride. This level is well below our current exposure levels and should be of concern to everyone.[5]

Furthermore, otherwise unaware members of IAOMT were shocked to learn that the chemical widely used in the artificial fluoridation schemes is untreated hydrofluosilicic acid waste from the phosphate fertilizer mining industry that has not been tested, much less been proven safe or effective.[6] This product, along with its salt form used in

91% of the fluoridation schemes, contains numerous contaminants, including arsenic and lead, which have never been factored in to any risk assessment.

Since no benefit can be determined from ingested fluoride and numerous risks are apparent, the appropriate PHG has been established by the IAOMT as zero. This risk assessment raises serious concerns about the pervasive over-exposure to fluoridated drinking water and fluorine-containing foods, beverages, pharmaceuticals, oral care products, and time-release dental fillings.

It is the position of this Academy that from a toxicological point of view fluoride proposes unacceptable health risks. IAOMT has determined that fluoride is not an approved dental material and should not be taken internally.

IAOMT has adopted criteria for establishing a Public Health Goal from the California Office of Environmental Health Hazards Assessment, and has applied these criteria toward establishment of a Patient Health Goal for purposes of dissemination to IAOMT membership and other interested parties.

A discussion of the criteria used in establishing a Public Health Goal, and an IAOMT Patient Health Goal, which are herein used interchangeably, and criticism of the California OEHHA establishment of a PHG for Fluoride, in which they defy their own criteria, are presented below for purposes of understanding the science and policy questions inherent in the fluoride discussion.

This report concludes with a comprehensive bibliography of the peer reviewed scientific literature, and other sources concerning fluoride that were consulted while establishing this Patient Health Goal.

Public Health Goal (PHG) for Ingested Fluoride

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The International Academy of Oral Medicine and Toxicology (IAOMT) has received input for this public health goal (PHG) from more than a dozen sources and co-hosted a scientific symposium on the health effects of ingested fluoride as a final step in developing this PHG. Adverse health effects demonstrated were: fluorosis; cancers; genetic damage; bone pathology; trans placental and brain transport; histological brain, artery, and kidney damage; and neurological impairment.

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International Academy of Oral Medicine and Toxicology
Standards of Care

Review of Health Effects of Ingested Fluoride and Applications in Dentistry

Preface

Patient Health Goal (PHG) and the suitability of Fluoride for use in dentistry with respect to adverse health effects and biocompatibility:

This IAOMT Technical Support Document (TSD) provides a review of the health effects and the currently available scientific literature. It also describes our methodology of analysis. This TSD was developed utilizing the best available toxicological data in the scientific literature. These documents and the analyses contained in them provide estimates of the levels of exposure that pose minimal risk levels (MRL) through chronic exposure over a lifetime.

We have adopted an MRL for the purpose of implementation in our standards of care in dentistry as a guide to our members in selecting suitable dental treatment and materials for their patients.

We have incorporated the following guidelines.

1. The PHG for acutely toxic substances shall be set at levels at which scientific evidence indicates that no known or anticipated adverse effects on health will occur, plus an adequate margin-of-safety.
2. PHG's for carcinogens or other substances which can cause chronic disease shall be based solely on health effects without regard to cost impacts and shall be set at levels which the IAOMT has determined do not pose any significant risk to health.
3. To the extent the information is available the IAOMT shall consider possible synergistic effects resulting from exposure to two or more compounds.
4. IAOMT shall consider the existence of sub groups in the population that are more susceptible to adverse effects of the compound than a normal healthy adult.

5. IAOMT shall consider the compound exposure and body burden levels that alter physiological function or structure in a manner that may significantly increase the risk of illness.
6. In cases where scientific ambiguity exists, the IAOMT shall use the criteria most protective of public health and shall incorporate uncertainty factors of non-carcinogenic substances for which scientific research indicates a safe dose-response threshold.
7. In cases where scientific evidence demonstrates that a safe dose-response threshold for a substance exists, then the PHG should be set at that threshold.
8. The PHG may be set at zero if necessary to satisfy the requirements listed above.
9. IAOMT shall consider exposure to compounds in media other than dental products, including drinking water, food, and air and the resulting body burden.
10. PHGs adopted by IAOMT shall be reviewed periodically and revised as necessary based on the availability of new scientific data.

Chemical Profile

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In the free state, fluorine is a pale yellow diatomic gas. Fluorine is never found in this form in nature, because it is very chemically reactive and combines with every other element except the inert gases. It is the 13th most abundant element, commonly occurring in the minerals fluorspar (CaF_2), cryolite (Na_3AlF_6) and fluorapatite ($3Ca_3(PO_4)_2\ Ca(F,Cl)_2$).[7] [8]

Fluorine is the ninth element on the periodic table. It has an atomic weight of 18.9984. It is the most reactive of all of the elements and forms strong electro negative bonds. It is particularly attracted to the divalent cations of Calcium and magnesium. It is the lightest and most reactive member of the halogen family. Fluorine reacts with other elements to produce such ionic compounds as hydrogen fluoride (HF), sodium fluoride (NaF) and many others. When these ionic compounds are dissolved in water, the ions dissociate and fluorine is present as the negatively charged ion fluoride. Fluoride, usually as the sodium salt, is added to drinking water. The most common form of fluoride added to drinking water are sodium fluoride (9% of water systems) and Hydrofluosilicic acid and silicofluoride (91% of water systems). Fluoride salts are also naturally occurring in geological formations, and therefore are found as contaminants in some sources of drinking water.

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Uses

Fluorine is used in aluminum, steel, glass, enamel, brick, tile, pottery and cement manufacturing; fluorinated chemical and phosphate fertilizer production; and metal casting, welding and brazing.[9] [10] Sodium fluoride (NaF) is used in various pesticide formulations, including insecticides and wood preservatives.[11] Sodium aluminum fluoride cryolite (Na_3AlF_6) is widely used as a pesticide and is found in substantial quantities as residue on most non-organically grown fruits and vegetables. Fluoride-containing compounds, primarily silicofluorides, are employed in the artificial fluoridation of drinking water allegedly for the prevention of dental caries.[12] Fluoride-containing dental products are now widely available, including toothpaste, supplements, mouth rinses and professionally applied gels and varnishes.[13] Fluoride (primarily as NaF) has also been used unsuccessfully in the treatment of osteoporosis.[14] Treatment of people with osteoporosis with fluoride resulted in increased radiographic bone density and a dramatic increase in hip fracture.[15] No fluoride containing substance for the purpose of treating or preventing either osteoporosis or tooth decay intended for ingestion has been approved by the US Food and Drug Administration.[16]

Both natural and anthropogenic sources can contribute fluoride to soil, air, water and food. About 23 500 tons of inorganic fluorides are released from anthropogenic sources in Canada each year, 4 whereas global volcanic sources are estimated to release 60-6000 kt annually.[17] Fluoride can occur naturally in surface waters as a result of the deposition of particulates from the atmosphere and the weathering of fluoride-containing rocks and soils. Groundwater can also contain high concentrations of fluoride owing to leaching from rocks. Chemical manufacturing plants and waste ponds can contribute fluoride to raw water sources directly through effluents or indirectly through volatilization. [3,18] Free fluoride ions predominate in aqueous solutions, but both ionic (i.e., inorganic) and nonionic forms of fluoride can be present in plant and animal tissues. [8,19]

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Exposure

Elevated levels of naturally occurring fluoride in drinking water are found in every state except Alaska, District of Columbia, Tennessee, Rhode Island, and Vermont. Some states (Colorado, Kansas, Oklahoma, Arkansas and Texas) have areas with high endemic fluoride contamination.[20] Elevated levels of endemic fluoride contamination in drinking water are relatively infrequent in Canada, although communities in Quebec, Saskatchewan and Alberta have recorded concentrations as high as 2.5 to 4.3 ppm. [21] 20 or more years ago the typical fluoride concentrations in fresh and cooked foods from Canada and the United States include 0.01 to 0.80 *u*g/g for dairy products; 0.01 to 0.58 *u*g/g for fruit; 0.04 to 4.57 *u*g/g for meats, fish and eggs; 0.05 to 0.13 *u*g/g for fats; and 0.02 to 0.86 *u*g/g for sugar-based foods.[22] [23], Since that time the standards for pesticide residue on foods and the maximum contaminant levels of fluoride in drinking water have been greatly relaxed. A mean fluoride concentration of 0.54 *u*g/g (543 *u*g/L) (<0.05 to 5.85 *u*g/g or 0.5 ppm) was recorded in a 1990 survey of 172 bottled waters obtained across Canada.[24]

The fluoride concentration of water used to reconstitute or prepare beverages and dry concentrates can greatly influence their fluoride content. [25] [26] [27] In the United States, fluoride concentrations in infant formulas were found to range from 0.127 mg/L for ready-to-use milk-based formulas to 0.854 mg/L for soy-based powdered formula prepared using water containing a fluoride concentration of 1.0 mg/L.[28] A Canadian survey found that women consuming non-fluoridated drinking water (<0.16 ppm (mg/L) fluoride) produced milk with a mean fluoride concentration of 4.4 ng/g (*u*g/L), whereas breast milk from women consuming fluoridated drinking water (1 ppm fluoride) contained .0098 ppm .[29]

No Canadian data are available on fluoride concentrations in indoor air. Average monthly ambient air concentrations (gaseous and particulate) reported for a residential area of Toronto (Ontario) in 1981 ranged from 0.01 to 0.05 *u*g/m^3, with a mean of 0.03 *u*g/m^3.[30]

Canadian estimates of mean soil fluoride concentrations range from 6 ppm (*u*g/g) for a forest in Newfoundland (depth and range not specified) to 309 ppm (63 to 1000 ppm at depths of 0 to 130 cm) for 23 Canadian Soil Survey Committee (CSSC) reference samples.[31] [32]

The most commonly used fluoride-containing dental product is toothpaste. At least 95% of the toothpastes sold in North America

contain fluoride as NaF and/or disodium monofluorophosphate (MFP, or Na_2PO_4F), with an effective fluoride concentration of approximately 1000 ppm (*u*g/g). [33] [34] [35]- [36] [37]The mean amount of toothpaste ingested per brushing by children 1 to 4 years of age ranges from 0.13 to 0.39 g. In contrast, adults 20 to 35 years of age ingest an average of 0.04 g toothpaste per brushing.[38]

Other fluoride-containing dental products include fluoride supplements (NaF tablets or drops) for infants and children, fluoride mouth rinses and topical fluoride gels (12,000 to 15,000 ppm) applied by dentists and dental hygienists.[39] [40] Some countries in the European Common Market have quietly removed systemic fluoride tablets from the market and others have placed an outright ban on the sale of such products based upon their concern for the health and safety of the citizens. [41]

The estimated daily intake of fluoride from drinking water, air, soil, food and toothpaste for two age groups (7 months to 4 years and 20+ years) in the general Canadian population is shown in Table 1.

Daily fluoride intakes from supplements, mouth rinses and gels were not estimated, as the available data on the proportion of the general population using these products or the amount of fluoride ingested from them were considered inadequate. However, regular supplement use in accordance with either Canadian Pediatric Society or Canadian Dental Association recommendations could add as much as 19 to 76 ug/kg bw/day to the daily fluoride intakes of preschool children. Although supplements are not recommended for children who are already ingesting fluoride from toothpaste and or drinking water many physicians continue to dispense supplements in areas where they are clearly never indicated.

For children less than 6 months of age with a body weight (bw) of 7 kg and a daily consumption of 0.75 L of breast milk, daily fluoride intake can be estimated to be less than from 0.47 to 1.05 *u*g/kg bw per day. For the child using 1 ppm tap water based formula the daily dose would range from 250 to 91.5 *u*g/kg bw or approximately 250 to 500 times more fluoride than the breast fed infant.

The US EPA has established 60 *u*g/kg bw as the minimum risk level for excess fluoride exposure in children. It is clear from the current exposure levels that some children who brush their teeth and live in a non-fluoridated area already exceed this level.

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Executive Summary Response to CA OEHHA setting of PHG of 1 ppm

J. William Hirzy, Ph.D., Senior Vice President of the union that consists of and represents all of the scientists and other professionals at the U.S. Environmental Protection Agency, Washington, D.C. submitted references for neurological impairment and behavioral change, carcinogenicity, updated science concerning fluoride's topical effects versus ingestion effects, hip fracture studies, correction of errors in computing total daily intake necessary to cause crippling skeletal fluorosis and other phases of skeletal fluorosis, and high incidence of abnormal dental occlusion; as well as a statement from the union outlining their scientific reasons for concluding that the health and welfare of the public is not served by addition of fluoride to the public water supply.[42]

In addition, Dr. Hirzy requested that congress review the raw data of the NTP carcinogenicity study rather rely than the disputed United States public Health Service's review that downgraded classifications two standards from "probable" to "equivocal" without adequate justification. On June 29, 2000 before the Subcommittee on Wildlife, Fisheries and Drinking Water of the United States Senate Doctor Hirzy called for an immediate moratorium on all water fluoridation schemes in the United States.[43]

California Occupational Environmental Health Hazard Assessment presents a table of Estimated Total Daily Intake in the Draft and acknowledges the necessity of taking all factors into account, yet ignores all other sources in arriving at a PHG that guarantees over-exposure.

OEHHA establishes a PHG even higher than a still-disputed-as-excessive "Estimated Safe and Adequate Daily Dietary Intakes (ESADDI)" for fluoride in the 1989 Recommended Dietary Allowance publication of the National Research Council, which recommends 0. 1 to 0.5 mg Total Daily Intake for younger infants (0-0.5 yr.)

After recommending a PHG that supports a higher level of fluoride in the public water supply than an infant should receive from their entire diet, OEHHA has the gall to warn that "Excessive exposure to fluoride should also be avoided by pregnant women, especially in the latter weeks of pregnancy when the teeth of the fetus are beginning to form" (Pg. 18), yet fails to mention that in California and the US there are no labeling requirements for foods, beverages, or bottled waters to disclose fluoride content.

Fluoride is so ubiquitous that no diet can be constructed for man that is deficient or lacking in fluoride. Never once mentioned in the OEHHA Draft is the fact that fluoride cannot be removed by carbon or other screening filtration, nor boiled away. Distillation, which does remove fluoride, is not commercially viable for all products, or accessible to the majority of the population, much less the highly susceptible or indigent.

In 1979 the FDA required the deletion of all government references previously classifying fluoride as "essential or probably essential" (Federal Register, March 16, 1979, pg. 16006).

25 countries, representing 98% of Europe's population, with bodies of health professionals, scientists and public health agencies of their own, reject fluoridation, some with outright bans. Like our European counterparts, 83% of Californians remain non-fluoridated, despite attempts by promoters to force the ingestion of increased levels of fluoride upon us for more than 50 years. A major difference between fluoridation status in Europe and California that must be noted is that California citizens have had to act on their own to protect the public safety when public agencies abandon their scientific integrity and social ethics in order to promote a political agenda, as has happened in the recent OEHHA report.

While promoters tout thousands of studies espousing the effectiveness of fluoridation, they have yet to reveal the existence of even one study that conforms to normal standards of scientific credibility. (Sutton)

Fluorides are used in general anesthetics and many psychotropic drugs such as Prozac (fluoxetine). Millions of Americans are exposed to these drugs that are intended to inhibit seratonin, a chemical in the brain.

The two diet drugs just removed from the market by the FDA for damage to the heart and lungs, with mood altering effects, Phen-Fen (fenfluramine) and Redux (dexfenfluramine), are both fluoride products that are obviously not prescribed to improve dental health.

OEHHA as do all of the promoters of ingested fluoride makes no attempt to address that fluoride is employed to impact other areas of the body other than teeth, much less identify what role fluoride plays.

OEHHA blatantly and negligently omits all reference to fluoride's neurological effects. Within the prescribed time period for inclusion in the November Draft of the PHG, William Hirzy, Ph.D., Senior Vice President of National Federation of Federal Employees, local 2050, which represents

all of the scientists, attorneys, statisticians, and engineers at U.S. EPA headquarters in Washington, D.C., presented OEHHA with a rat study by Mullenix, et al. which shows fluoride causes neurological impairment and behavioral changes. This study is supported by two epidemiological studies from China that show a correlation between fluoride and lower IQ in children. All three studies are enclosed and referenced later in this critique.

Within the prescribed time period for inclusion in the November Draft, Maureen Jones appeared before the OEHHA and presented an oral description and supportive documents outlining the mathematical error that has universally been utilized by promoters of fluoridation to justify their claim that it would take 20 to 80 mg/day for 10 to 20 years to cause skeletal fluorosis.

The mathematical error was corrected in *Health Effects of Ingested Fluoride, NAS/NRC, 1993* to 10 to 20 mg/day for 10 to 20 years.

That same accumulation over 10 to 20 years requires only 2.5 to 5 mg/day over a 40 to 80 year period, which is a level of Total Fluoride Intake already surpassed by both children and adults.

However, this calculation is an estimate of the exposure to fluoride which would cause Phase III crippling skeletal fluorosis. Phase I and Phase II occur at much earlier stages of exposure, which causes suffering first from sporadic pain and stiffness of joints, and then arthritic symptoms, slight calcification of ligaments, with or without osteoporosis.

Even residents of non fluoridated communities will have to reduce their fluoride intake from other sources than water to avoid the devastating long term effects.

The most obvious manipulation of fact by the OEHHA is the establishment of a NOAEL of 1 mg/L. The NOAEL x BW in the formula is intended to represent the no-observed-adverse-effect-level of Total Daily Intake. The draft leaps to the 1 mg/L with the explanation that other source contribution is considered in all of the studies at 1 mg/L when in fact the original establishment of 1 mg/L (Dean, 1942), which was disputed at the time and is still disputed today as too high, assumed only 1 liter of consumption and no other significant source of contribution. Thus the disputed original no-observed-adverse-effect-level Total Daily Intake was established at 1 mg/day.

It should be apparent to any reader of this Draft that fluoride toothpaste, fluoride mouth rinse, fluoride floss, and fluoride supplements were not available at that time. Mechanically de-boned chicken with high-fluoride content was not a food staple for the general population in the 1940's. Fluoride based disinfectants currently used on chickens and other poultry were not prevalent in the 1940's. Nor was any other part of the general food supply exposed to increased levels of fluoride from the public water systems as they are today.

Exposure to high levels of fluorine-based pesticides in the food supply was not as prevalent in 1942. Baby foods and packaged juices of today frequently use white grape juice (high in fluoride from pesticide residue) as sweetener, which was not the practice in 1942.

Other sources of fluoride in fruit juices made from concentrate, and other beverages, raisins, grains, cereals, general anesthetics, psychotropic drugs, children's vitamins, dental materials, and dental topical applications were also not prevalent in 1942.

OEHHA presents a graph (Fig. 1) showing an increase in dental fluorosis relative to ppm fluoride in the water, when in fact this is another distortion. The effect includes all sources of fluoride intake. Applying Table 1 of Estimated Total Daily Intake to this graph indicates how 8 to 51% of children in fluoridated communities suffer from dental fluorosis, and how 3 to 26% of children in non fluoridated communities suffer from dental fluorosis.

If the OEHHA chart is to be believed, it is clear that those children consuming as much as 4 mg/day Total Daily Intake are at ever-increasing risk of severe dental fluorosis, and that children in non fluoridated communities at the upper range of the OEHHA Estimate of Total Daily Intake are not immune to severe dental fluorosis either.

OEHHA selectively chooses to report a mean prevalence in four fluoridated cities of a 22% incidence of dental fluorosis, in order to minimize the incidence of fluoridation.

It should be noted that the examination process to determine the presence of dental fluorosis entails identifying dental fluorosis only when at least two teeth are damaged, and the severity is classified as the least effected; so in reality the severity is always understated. A classification of mild dental fluorosis indicates that up to fifty percent of the least effected tooth is damaged, while moderate fluorosis indicates that more than 50% of the least of two effected teeth is damaged.

At the Canadian Conference on Fluoride Supplements on November 29, 1997, reports estimated dental fluorosis in Canada effects 30% to 65% of Canada's children.

With dental fluorosis admittedly on the rise, the OEHHA refusal to recognize overdose, even in non fluoridated communities, is scientifically bewildering.

Although the OEHHA Draft gives lip service to the need to consider all sources of intake, even supplying a Fluoride Intake table, OEHHA evades a mathematical computation considering total intake, in favor of an end result amenable to the pro-fluoridation projection of safety. Using the still-disputed-as-excessive 1 mg/day as a NOAEL and a Relative Source Contribution of 21.6% from 1 mg's representation of Table 1's estimated 4.6 mg Total Daily Intake for children (pg. 4), leaving all other factors constant, would result in a PHG of .216 mg/L ___ but of course this does not support the pro-fluoridation agenda.

OEHHA admits that the PHG provides little or no margin-of-safety, but never attempts to address any of the subsets of the population that are identified as unusually susceptible (ATSDR, 1993). OEHHA sloughs off the requirement to consider the most sensitive individuals (Pg. 17), stating that they, indicating only ("i.e. children"), were included in the study population.

The populations identified as unusually susceptible include the elderly (age 50+, Hanhijarvi, 1974), people with deficiencies of calcium, magnesium, and/or vitamin C, and people with cardiovascular and kidney problems. Impaired renal clearance of fluoride has also been found in people with diabetes mellitus.

These individuals are not included in the study base, nor considered in any formulation. Nor are outdoor laborers, athletes, people with excessive thirst or diabetes insipidus, and individuals who drink more than the average amount of water for purposes of detoxification.

Adults with diabetes insipidus routinely drink 8 to 12 liters of water per day. Children similarly afflicted are assumed to drink approximately half that amount -- 4 to 6 liters/day. Using the still-disputed-as-excessive NOAEL of 1 mg/day (no NOAEL has ever been established by any scientific standard for more than 1 mg/day) and a Relative Source Contribution of 0.62, representing 6 mg of fluoride from the 6 liters of water of the 9.6 mg Total Daily Intake for children (Table 1, Pg. 4, 4.6

mg+ 5 additional mgs), dividing by 6 liters, without adjusting for Uncertainty Factor, would result in an OEHHA fluoride PHG of .103 mg/L.

Using Mullenix's Rat study showing neurological impairment and behavioral changes as an end-point, Mullenix's study produced a no-observed-adverse-effect-level in a 6 week sub-chronic test that used 75 mg to produce a 0.12 F serum level that is producible in humans with 4 to 8 mg F. OEHHA could have calculated the PHG with neurological impairment as the endpoint as follows; 4 mg x 100% RSC divided by Uncertainty Factor of 100 for animal extrapolation and severity, equaling a PHG of 0.04 mg/L.

Crippling skeletal fluorosis can be produced at 2.5 to 5 mg/ day for 40 to 80 years. A quick check of Table 1, Estimated Total Intake, reveals that both children and adults in fluoridated communities are already being overdosed, with some children in non fluoridated communities at risk. Phase I and Phase II appear to be inevitable to some degree for almost everyone unless a reduction in exposure prevails. The severity dictates a PHG of 0.00.

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Scientific Critique

Summary of Criticisms:

The recommendation for ingested fluoride is extremely flawed and decidedly biased. In order to be accurate a review of the literature must be comprehensive, yet advocates for drinking water fluoridation repeatedly ignore much of the available scientific information and utilizes out-of-date flawed research studies that are not valid by today's standards.

In addition, they rely almost entirely on reviews of fluoride rather than upon original scientific experiments. The reviews themselves have been frequently attacked in the scientific literature. In some cases scientific fraud was alleged for preparing favorable pseudoscientific reviews. The review papers often have changed the results to fit their pro-fluoridation perspective and thus deceive the readers into believing that valid science actually exists.

The subject under review is the safety of ingested fluoride; therefore, it is not reasonable to include biased remarks regarding the alleged beneficial effects of water fluoridation upon the dental health of children. The pro-fluoride rhetoric and illogical bias displays the mindset of the reviewers

and partially explains why they have opted to defy the established scientific guidelines for the scientific review.

While topical fluoride may reduce tooth decay in children, ingested fluoride does not. All of the broad based, blinded studies of animals or humans that have either found an increase in decay of permanent teeth at 1 ppm or no difference. There are several studies which have found a delay in tooth eruption for children ingesting fluoride from the drinking water (Sutton[44], Limeback[45], NIDR 1987). The delay in eruption fully accounts for the transient reduction in tooth decay seen in the 5 to 8 year old children (Yiamouyiannis [46]). The delay in eruption is not a health benefit, but is indicative of a generalized slow-down in the growth of the child that has enormous implications for the future health of that child.

Mirth et al demonstrated by an animal experiment that animals with oral F releasing implants had caries inhibition and those with continuous slow release F pump implanted under the skin did not.[47] [48] [49] [50]

Fluoride has produced considerable delay in the eruption of children's teeth.[51] Drs. L. Krook and G. A. Maylin describe a mechanism that produces marked delay in the eruption of teeth (1.5 to 3.0 years) in cattle crippled with fluorosis (fluoride damage to bone), due to atmospheric fluoride pollution.

Krook et. al. found that exposure to fluoride had produced a great decrease in the number of certain cells in bone (resorbing osteocytes) which play a major role in the resporption of the roots of the deciduous (first) teeth and of bone; both of which processes are necessary before permanent teeth can erupt normally. They stated: "The *delay in eruption in the permanent teeth has also been reported in children in fluoridated communities." "The cause of the delay in eruption was shown in the present material. Fluoride arrests resorption of deciduous tooth roots and of the supporting bone. By inducing one disease (fluorosis), fluoride delays the manifestations of another (dental caries)*[52]."

The formula for establishing a safe daily intake of fluoride is blatantly manipulated by proponents of fluoridation. None of the reviews established a scientifically valid NOAEL. OEHHA admits that severe dental fluorosis occurs even at 1 ppm (pg. 15). Purposely substituting a known observed level for a no-observed-level can only lead to observable incidence and no margin-of-safety. Therefore the formula must include an uncertainty factor above 1. The OEHHA review cites positive correlations to severe adverse health effects, then erects inconsistent requirement for proof. Rather than utilizing scientific methodology to compute uncertainty

factors, OEHHA claims uncertainty factors are a reason for abandoning consideration.

The CDC ATSDR on page 112 described the at-risk populations for fluoride ingestion.

U.S. DEPARTMENT OF HEALTH & HUMAN SERVICES
TP-91/17
HEALTH EFFECTS OF FLUORIDE

2.7 POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE

> "Existing data indicate that subsets of the population may be unusually susceptible to the toxic effects of fluoride and its compounds. These populations include the elderly, people with deficiencies of calcium, magnesium, and/or vitamin C, and people with cardiovascular and kidney problems.
>
> Because fluoride is excreted through the kidney, people with renal insufficiency would have impaired renal clearance of fluoride (Juncos and Donadio 1972). . . .
>
> People on kidney dialysis are particularly susceptible to the use of fluoridated water in the dialysis machine (Anderson et al. 1980). . . .
>
> Impaired renal clearance of fluoride has also been found in people with diabetes mellitus and cardiac insufficiency (Hanhijarvi 1974). People over the age of 50 often have decreased renal fluoride clearance (Hanhijarvi 1974). This may be because of the decreased rate of accumulation of fluoride in bones or decreased renal function. This decreased clearance of fluoride may indicate that elderly people are more susceptible to fluoride toxicity.
>
> "Recent studies indicate that fluoride may increase the rate of hip fractures in elderly men and women."

The proposed PHG of one PPM protects none of the above populations. Instead, OEHHA chooses to use dental fluorosis as the sole risk factor considered in the PHG. In the case of skeletal fluorosis, OEHHA does not protect against the latent development of stage III severe skeletal fluorosis and virtually assures the development of stages I and II in the majority of the population. Stiff back syndrome is already prevalent in our over-fluoridated society.

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Scientific Summary

In summary, The IAOMT following our previously listed risk assessment guidelines, presents a comprehensive review of the available scientific literature. We find that the present US EPA maximum contaminant level for water (4 ppm) and the recommendation for drinking water fluoridation (1 ppm) will produce a measurable increased risk of cancer, hip fracture, dental fluorosis, and neurological impairment and virtually assures the development of stages I and II skeletal fluorosis in many individuals exposed to these levels of fluoride in their drinking water. The IAOMT PHG for fluoride is appropriately zero. As will all cumulative toxic substances, avoidance of exposure, wherever possible, is the most appropriate public health goal and the only way to completely prevent adverse health effects.

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Referencing known science to criteria for Patient Health Goal

The stated goal of the PHG is to protect the most vulnerable segment of society from fluoride related injury and adverse health effects even over a lifetime of exposure utilizing the best available toxicological data. Thus their recommended PHG should offer no significant risk to individuals.

Skeletal fluorosis and dental fluorosis develops in vulnerable populations at very low levels, therefore, the PHG must be supportive of the goal of preventing adverse health effects including the earlier signs and symptoms of fluoride overdose. Early signs of fluoride overdose start with cartilage and with *"vague pains , noted most frequently in the small joints of the spine. These cases are frequent in the endemic (local) areas and may be misdiagnosed as rheumatoid- or osteo-arthritis.*

In later stages, there is an obvious stiffness of the spine with limitation of movements, and still later, the development of kyphosis (hunch back).[53].

Page 57 of the 1993 ATSDR TP 91/17 under Health Effects states, "If this effect is confirmed, it would mean that hip fracture in the elderly replaces dental fluorosis in children as the most sensitive endpoint of fluoride exposure".

It is important to recall that since 1993 when the ATSDR was prepared, additional confirmatory research linking fluoride to hip fracture has been

published.[54] Animal and human research linking dental fluorosis to neurological impairment has also been published.[55] [56] [57] Neither of these developments is referenced in the pro-fluoridation CDC review papers.

Fluoride is a violent protoplasmic poison that accumulates, over a lifetime, in calcium-rich tissues. A presumably toxic dose (PTD) is approximately 5 mg/kg body weight for humans. However, death in susceptible individuals has been reported at 0.3 mg/kg estimated dose. (Hoopers Bay).

Some people with cardiovascular problems may be at increased risk of fluoride toxicity. Fluoride inhibits glycolysis by inhibiting enolase.[58] [59] It also inhibits energy metabolism through the tricarboxylic acid cycle by blocking the entry of pyruvate and fatty acids and by inhibiting succinic dehydrogenase.[60]

One of the most susceptible populations would be infants fed entirely tap-water based formula or home-prepared vegetables, rice and other water-absorbing foods. Infants fed baby foods such as mechanically de-boned chicken who have impaired renal function, or diabetes insipidus with poor fluid retention, are at great risk. It is a well established fact that dental fluorosis is linked to a combination of fluoride in the water and the absence of breast milk. Human breast milk usually contains less than 0.01 ppm fluoride. Fluoridated tap water therefore contains 100 times more fluoride than breast milk. When a baby is fed infant formula mixed with tap water it receives a daily dose 100 times greater than the infant on breast milk.

The latest Academy of Pediatrics guideline for infant-feeding recommends breast feeding for as long as mutually agreeable, and for at least one year. They note that an infant's failure to nurse is linked to numerous adverse health effects, including cancers and sudden-infant-death syndrome. Sudden-infant-death syndrome has been linked to water fluoridation in at least one study[61].

The vulnerable, sick infant segments of the population are not mentioned in the PHG. Some infants do not have completely formed kidneys. Approximately 50% of ingested fluoride is excreted through the kidneys. Since some infants are born with impaired kidney function and little is known about how a normal newborn's kidney handles fluoride, the uncertainty factor must be increased in the formulation of a PHG.

The dose of fluoride necessary to cause dental fluorosis is 0.04 mg/kg. An infant that weighs 8.8 pounds or 4 kg who drinks one liter per day of

water-based formula would receive a daily dose of 0.25 mg/kg or roughly 6 times the lowest dose necessary to cause dental fluorosis. Dental fluorosis has increased steadily since the introduction of fluoride into the community drinking water of this country, and now affects 8 to 51% of the children in fluoridated communities (NRC, 1993). Some underprivileged fluoridated communities such as Augusta, GA are reporting dental fluorosis rates as high as 80%[62]. California has traditionally experienced less dental fluorosis since water fluoridation has been rejected by the majority of communities. Dental fluorosis is known to occur in non fluoridated communities (3%-26%, NRC, 1993). Therefore, even if the PHG were set at 0.0 ppm it would not fully protect our children from dental fluorosis.

This is particularly important since the full nature and extent of other health effects of dental fluorosis is not fully known. Some authors have identified adverse psychological impact in children who suffer from the unsightly defects of dental fluorosis. OEHHA attempts to minimize the social impact by characterizing dental fluorosis as a cosmetic defect; however, in 1986 the California Department of Health rejected the US EPA contention that dental fluorosis is only cosmetic and ruled that dental fluorosis is an adverse health effect.

Dental fluorosis is caused by fluoride damage to the cells (ameloblasts) making tooth enamel during tooth formation. At the same time enamel is forming, many other tissues in young children are also growing. Brain damage and bone damage have now both been linked to dental fluorosis[63] [64]. Additional research is badly needed to determine the full extent of the harm caused by fluoride; however, it is clear that the daily dose for many children in non fluoridated areas, from sources other than water, already exceeds the tolerable safe intake of fluoride.

The PHG's for acutely toxic substances should be set at levels which scientific evidence indicates has no known or anticipated adverse effects on health, plus an adequate margin of safety.[65] It is within the scope of OEHHA to establish a PHG of 0.0 mg/L for fluoride and this is supportable by the science available.

Item 2 of the Preface: The PHG for **carcinogens** is to be based solely on health effects without regard to cost impact and shall not pose any significant risk to health.

One of the first positive findings of carcinogenicity of fluorides in humans was the Burke-Yiamouyiannis 1975 study that linked drinking water fluoridation to increased cancers in the general population. Congressman

Fountain explained to the public after extensive hearings that he could not assure the public that fluoride was not a carcinogen. Congress ordered the National Cancer Institute to immediately begin cancer studies and report back to congress no later than 1980. They furnished no report until almost a decade later. The NCI paid Battelle Laboratories to test rats and mice for carcinogenicity. In the words of the Battelle Laboratories Pathologists, the high-dose animals were "awash with disease". They had cancers of the oral cavity, liver, and bone. Their kidneys were damaged and they looked like death was nearly upon them.

This study was turned over the United States Public Health Service for "peer review." In the process of preparing the draft report for peer review, every cancer was downgraded, not one level but two. The rare cancers of the liver (hepatocholangiocarcinomas) were downgraded to hepatomas, a common benign tumor frequently found in animals. The review committee used studies from other NCI experiments as controls in order to lower the significance of the osteosarcomas. These outside studies had no control of fluoride in their diet, and analysis of their bone fluoride levels more accurately places them at the mid-range dose animals.

It is unprecedented in research to give a mid-range dose of the suspected carcinogen to a control group and then claim that these animals' cancer rate can be used to lower the significance of the cancers found in the study subjects. This is exactly what occurred in the NTP fluoride/cancer peer review process. The low-dose animals had no osteosarcomas. The historical control group (mid-range dose) experienced a relatively high bone cancer rate of 0.6%. The fact that there are over 6,000 animals in the historical controls makes these findings very significant.

This OEHHA Draft relies heavily upon the US PHS version of the NTP data for it's claim that fluoride is not a carcinogen. OEHHA also includes as supporting evidence of lack of mutagenicity the Ames Salmonella assays in-vitro study[66]. Dr. Ames himself has clearly stated that his bioassay is not suitable for a material like fluoride. It is an inappropriate test and yields no significant information. Why, then, was it included except to give the PHG the appearance of scientific validity. The NTP also investigated fluoride mutagenicity in-vitro. In every test except the Ames test, fluoride produced mutations[67]. Both the NTP and OEHHA are suppose to take into consideration disturbing results such as these. Instead the authors chose to rely upon the biased reviews, rather than upon the research itself.

Following the revelation of male rat osteosarcomas in a dose-dependent fashion from the NTP study, Dr. Cohn of the New Jersey Department of Health surveyed the prevalence of osteosarcoma in young males of the state. He found a dramatic increase in osteosarcomas in fluoridated areas of New Jersey. The National Cancer Institute surveyed the entire United States population and concluded there was no increase in osteosarcomas, yet the data published in their study indicated that there is a 68% greater chance of developing osteosarcomas in fluoridated communities than non fluoridated communities. This is not the first time the NCI has been caught in producing misleading information with regard to the carcinogenicity of fluoride, including the use of erroneous data and giving false testimony under oath.[68].

And of course the osteosarcoma did not go unnoticed in the TP 91/17 ATSDR 1993:

> *"A large study of fluoride conducted by the National Toxicology Program with both rats and mice found that a small number of male rats developed bone cancer (osteosarcoma) after drinking water with high levels of fluoride in it throughout their lives. . . . The bone cancer seen in the rat study is rare in humans,* ***although its frequency has recently increased among males in countries with fluoridated water.****"*
>
> *"****The osteosarcoma rate in males living in fluoridated areas has increased markedly in recent years*** *. . ." (Page 123)*

The NTP study is far too extensive to go into in its entirety here, however, the rare form of liver cancer found in both the rats and mice is significant by itself. Dr. Mel Ruber, the pathologist who originally described this cancer has confirmed that the rats did suffer from hepatocholangiocarcinomas despite the claims of the US PHS to the contrary.

FLUORIDE LINKED TO INCREASE IN HEPATOCHOLANGIOCARCINOMAS

Fluoride in drinking water	Percent of animals wit hepatocholangiocarcinc and total number o	as animals examined
	Males	Females

0 ppm	0% (0/79)	0% (0/79)
11 ppm	2% (1/50)	2% (1/52)
45 ppm	2% (1/51)	0% (0/50)
79 ppm	4% (3/80)	4% (3/80)

Dr. William Marcus blew the whistle on the Public Health Service for alterations to the data of the NTP study. He had consulted with numerous cancer experts and is a specialist in osteosarcoma himself. He states that the changes to the hepatocholangiocarcinomas and oral cancers were not appropriate. He called for an independent review of the NTP changes. He was subsequently fired and then won his whistle-blower lawsuit with punitive damages. The US PHS service has arrogantly refused to answer a single criticism of their obvious scientific fraud.

Studies of cancerous animals indicate that fluoride is a cancer promoter. The tumors grow faster and better in animals exposed to fluoride. No consideration was given to those segments of our population who are already battling cancer, who now may lose that battle for life, due to this failed and thoroughly discredited public policy of drinking water fluoridation[69].

In Kennedy versus Lungren, Sacramento Superior Court, 1997, The California Legislative Analysts Office acknowledged that high doses of fluoride do cause cancer.

Item #3 To the extent the information is available, we shall consider possible synergistic effects resulting from exposure to two or more contaminants.

The synergistic actions of fluoride would fill most libraries, and much is still to be learned about this very aggressive poison. Fluoride is the most reactive element in the periodic chart, therefore it interacts with everything. It will etch asbestos, glass, concrete, and any other substance.

In a soft water system such as is found throughout Northern California, fluoride will etch the pipes and deteriorate the city plumbing. In the process it will release asbestos from the concrete water lines and leach lead out of solder joints. In 1992 Tacoma, Washington had to shut down the fluoridation equipment due to the fact that fluoride had eaten the pipes. The municipal water had approximately 32 parts per billion (ppb) lead at the time of the breakdown. After the breakdown, the lead level dropped to 17 ppb. When the equipment was fixed, the lead level shot

right back up to 32 ppb. The city fathers decided to discontinue the use of fluoride, and the lead level again dropped. Over the next several years the lead level continued to drop, and today it is about 5 ppb.

Thurmont, Maryland had an identical experience with fluoride raising lead levels in their municipal water system. The EPA Maximum Contaminant Level for lead is 15 ppb. The Federal MCLG for lead is 0. Adding fluoride to the water supply in soft water areas will unquestionably increase the users exposure to lead.

Literally tons of other neutralizing chemicals, such as lime, must be added to counteract the addition of fluoride.

Calcium, Magnesium, Boron, Selenium, and Vitamin C have been found to mitigate fluoride poisoning. Undernourished and underprivileged children, and adults with deficiencies of these mitigating factors, will suffer increased rates and more severe damage from ingested fluoride. No assessment of the impact of fluoride on individuals of different nutritional status, or the possibility of co-carcinogenicity is addressed.

Fluoride readily replaces the other elements of the halogen group: chlorine, bromine, and iodine. OEHHA considers no association with these elements, or any deficiencies in other chemicals due to over-exposure to fluoride.

Item #4 The IAOMT shall consider the existence of groups in the population that are more susceptible to adverse effects of the contaminants than a normal healthy adult.

The 1993 ATSDR to find the following scientifically established facts (page 112),

> "Existing data indicate that subsets of the population may be unusually susceptible to the toxic effects of fluoride and its compounds. These populations include the elderly, people with deficiencies of calcium, magnesium, and/or vitamin C, and people with cardiovascular and kidney problems.
>
> Because fluoride is excreted through the kidney, people with renal insufficiency would have impaired renal clearance of fluoride (Juncos and Donadio 1972). . . .

Item #5 The IAOMT shall consider the contaminant exposure and body burden levels that alter physiological function or structure in a manner that may significantly increase risk of illness.

Storage of fluoride in bone is a progressive process.[70] Small ingested amounts of fluoride, such as from fluoridated water, beverages, food sources or swallowing fluoride toothpaste, accumulate in the bone.

Approximately 50% of each fluoride dose accumulates in the hard tissues of the body, primarily the bones. The toxic effects of fluoride in bone have been established for over 60 years. In classical empirical experiments, Kaj Roholm measured the bone burden of cryolite workers who developed skeletal fluorosis. From these experiments it was determined that the body bone burden of fluoride sufficient to cause crippling stage III skeletal fluorosis in adult males was 36,525 to 146,100 mg or 10 to 20 mg per day for 10 to 20 years.

Fluoridation of the public water supplies forces whole communities and whole generations in to a lifetime of exposure. The OEHHA PHG Draft Table 1, page 4, clearly shows that an adult high average daily consumption of fluoride from all sources is 7 mg/day. 7 mg/day X 365 X 75 years of life in fluoridated communities divided by 2 for 50% retention yields a body burden estimate of 95,812 mg/body burden. Thus, even simple arithmetic, not even considering excessive thirst, pre-existing diseases, or renal pathology, demonstrates body burden levels three times higher than Kaj Roholm found in cases of crippling stage III skeletal fluorosis. Stages I and II would occur at a much earlier point in the progressive poisoning from drinking water with 1 ppm fluoride.

A recent study by Sowers (1997) found that women 35 to 50 already have the same amount of aches and pains as their parents. The great fluoridation experiment has apparently induced more rapid aging of the bones, ligaments and back. This is exactly what was predicted before the experiment began in the 1940's.

Dr. Marcus expressed the concern for bone burden of fluoride from the NTP study very well in his May Day Memo (Marcus 1990);

> *This is an important consideration because as the document reports on page 9, the levels of fluoride in bone are linearly dependent upon dose and length of exposure ("depend upon total intake") in people. The level of fluoride in ashed samples of bone of 20-30 year old people is 200 - 800 mg/kg compared to 70 to 80 year old people of 1,000 - 2,500 mg/kg. In the document, the authors cited*

Zipkin [71]who reported on bone fluoride concentrations in four groups of individuals with average ages of 56 to 76 who lived in areas with fluoride concentrations in water of 0. 1, 1, Z 6, or 4 ppm The relationship to bone fluoride concentrations and water fluoride content was linear; bone fluoride ranged from about 800 to 7, 000 ppm ash with increasing water fluoride."

In the animal studies the levels of fluoride (Appendix 1) found in the bones of the animals were the same as or lower than those found in people. The highest dosed level of rats had lower levels of fluoride in their bones (5,470 ppm) compared to people (7,000 ppm) at the MCL of 4 ppm. This can be interpreted as people who ingest drinking water at the MCL have 1.3 times more fluoride in their bones than male rats who get osteosarcoma This is the first time in my memory that animals have lower concentrations of the carcinogen at the sight of adverse effect than do humans. An important toxicologic consideration is that a toxic substance stores at the same place it exerts it toxic activity. This is true of benzene and now for fluoride. Fluoride, however, is at twice the concentration in human bones compared to benzene which is 10 to 100 greater in animal marrow. This portends a very serious problem. One would expect to be able to discern a carcinogenic effect in the exposed population when compared to the unexposed population especially if data exist on the populations before fluoridation.

Investigators found that water fluoridation increased the bone burden substantially after only 15 years and that people who had impaired kidney function had double the level of fluoride in their bones as compared to those with good function[72]. Normal bone ash has only 500 to 1,000 ,mg F/kg[73 74 75]. In some cases people with impaired kidneys have over 3,800 mg F/kg after only 15 years. Based upon the works of Kaj Roholm stage I skeletal fluorosis could begin in an 80 pound susceptible individual after only 6 years of consuming 5 mg/day.

The PHG does not protect the public from a body burden of fluoride which is known to cause adverse health effects. It utterly fails to address susceptible subsets of the population to life-long exposure to this cumulative poison.

Item # 6 In cases of scientific ambiguity, IAOMT shall use criteria most protective of public health and shall incorporate uncertainty factors of non carcinogenic substances for which scientific research indicates a safe dose-response threshold.

Criteria has been established for the use of the uncertainty factors for drinking water by the National Academy of Sciences. Documentation is submitted with this position paper.

> *"A number that reflects the degree or amount of uncertainty that must be considered when experimental data in animals are extrapolated to man. When quality and quantity of data are high the uncertainty factor is low and when data are inadequate or equivocal, the uncertainty factor must be larger.*
> *The following general guidelines have been adopted in establishing the uncertainty factors.*
>
> *1. Valid experimental results from studies on prolonged ingestion by man, with no indication of carcinogenicity.*
>
> *Uncertainty factor = 10*
>
> *2. Experimental results of studies of human ingestion not available or scanty (e.g., acute exposure only). Valid results of long-term feeding studies on experimental animals or in the absence of human studies, valid animal studies on one or more species. No indication of carcinogenicity.*
>
> *Uncertainty Factor = 100*
>
> *3. No long-term or acute human data. Scanty results on experimental animals. No indication of carcinogenicity.*
>
> *Uncertainty Factor = 1,000.*
>
> *These uncertainty factors are used in every case as a divisor of the highest reported long-term dose which is observed not to produce any adverse effect.*

Since the US PHS altered the data on the NTP rat/mouse study without good reason it cannot be relied upon for determination of fluoride's potential as a carcinogen. However, since the peer review refused to say there was no evidence of carcinogenicity and chose instead to list fluoride as an **EQUIVOCAL** carcinogen. Clearly a UF above 1,000 is indicated.

OEHHA remarks on page 18, "*Individual variability might lead to a wide range of exposures not accounted for in the development of the PHG*" This statement indicates the necessity of a higher Uncertainty Factor.

OEHHA and the pro-fluoridation dentists often dismiss many of the relevant scientific studies by alleging that the adverse health effects are not yet proven or that the study did not adjust properly for some unspecified variable. There is no requirement of a PHG to have absolute proof positive of an adverse health effect.

Freni (1994) reported that reproductivity of humans declined with increasing fluoride, and his study is supported by animal studies.[76] OEHHA dismissed this study as a preliminary study, which it was not. Again scientific methods of establishing Uncertainty Factors should be employed.

"Another source of uncertainty is the added exposure to fluoride from other sources (estimates in the range of 20 to 80%) including diet, toothpaste, mouthwash, and dental supplements."

Item #7 In cases where scientific evidence demonstrates a safe dose-response threshold for a contaminant exists, then the PHG should be set at that threshold.

Proponents have expressed the belief in a threshold for fluorosis since the early days of water fluoridation. Anyone familiar with threshold would recognize that the 1942 graph of dental fluorosis clearly indicates there is no threshold for fluorosis but that fluorosis is a cumulative dose-dependent disease.

Item #8 The PHG may be set at zero if necessary to satisfy the requirements listed above.

The PHG for lead is zero. Lead is less toxic than fluoride and like fluoride accumulates in bone. The PHG for fluoride should also be zero. There are already too many sources of exposure to fluoride that cannot be controlled. The effect of fluoride on tooth decay germs is topical. The adverse health effects from ingested fluoride are systemic. Therefore, water should not contain fluoride since almost all, if not all, of the fluoride contained in water will be ingested, and produce nothing but adverse health effects.

Item #9. IAOMT shall consider exposure to contaminants in media other than drinking water, including food and air and the resulting body burden.

In 1996, and again in 1997, the California Legislative Analysts Office acknowledged that dental fluorosis will increase with water fluoridation.

However, any reasonable PHG must take into consideration that fluoridating public drinking water contaminates other food sources as well. Total Daily Intake from all sources must be considered to establish full body burden and to arrive at a protective PHG with an adequate margin-of-safety.

Looking at the 1942 table of dental fluorosis, it is clear that 0.8 ppm, even in 1942 when fluoride was not found in beverages, tooth pastes, mouth rinses, vitamins, and many pesticides, was not a low enough concentration to protect the public.

Item #10 PHG's adopted by IAOMT shall be reviewed periodically and revised as necessary based upon new scientific data. We are actively pursuing new research in this field including experiments with a goal of determining how to improve human health through defluoridation of the public drinking water.

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Fluoride Risks

RISK #1 Fluoridation is cancer-causing, cancer-promoting, and is linked to increased cancer rates in rats, mice, and humans.[77]

RISK #2 Hip fracture rates are substantially higher in people residing in fluoridated communities.[78]

RISK #3 Dental fluorosis, the first visible sign of fluoride poisoning, affects from 8% to 51% of the children drinking fluoridated water and has substantially increased over the last 40 years.[79]

RISK #4 All of the recent large-scale studies on fluoridation and tooth decay show that fluoridation does not reduce tooth decay.[80]

RISK #5 Fluoride drops and tablets are not approved by the U.S. Food and Drug Administration as safe or effective.[81]

RISK #6 Fluoride exposure and dental fluorosis are linked to lower IQ and neurological impairment.[82]

RISK #7 The citizens of America are already overdosed with fluoride.[83]

Children during growth accumulate fluoride more rapidly in their bones than adults. Systemic exposure to fluoride during tooth

formation will cause fluoride to accumulate in the teeth also. The primary route of excretion is through the kidneys and secondarily the liver.

Systemic exposure to water fluoride during tooth development causes dental fluorosis and does not affect the tooth decay rate of the permanent teeth. It does affect the bone metabolism and calcification. It does increase cancers at the epiphysis. Life-long exposure to low levels of fluoride will increase hip fracture.

Cancer:

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In 1977 an epidemiological study found a slight increase in all cancers was linked to fluoridated community water supplies[84]. Subsequent to that original paper several other studies have published research linking fluoride to cancer and genetic damage. Subsequent studies have failed to produce black and white answers and as a result the controversy continued.[85]

Pursuant to congressional order the National Cancer Institute through the National Toxicological Program (NTP) researched the fluoride cancer question in rats and mice. The two year study was conducted by the Battelle Columbus Laboratory.[86] Preliminary results, published in 1989, found a dramatic increase in bone cancers in only the male rats exposed to fluoride, and no bone cancers in the female rats, male and female mice. They also found an increase in oral cancers and dysplasias. The high dose rats drank 79 ppm fluoride and developed lip, cheek, throat cancers and dysplasias. Thus, the cancer and dysplasias appear to be due to the topical effect of fluoride. Since toothpaste is 1000 to 1500 parts per million, anyone brushing with fluoridated toothpaste would be exposed to considerably higher concentrations than the high dose rats which, in the words of the Battelle report were, "awash with disease . . . "

Dr. Cohn, at the New Jersey Department of Health, reported a significant association of bone cancer (osteosarcoma) rates of young men living in fluoridated cities compared to young men in unfluoridated communities[87] and was confirmed by Yiamouyiannis in a larger study[88]. Dr. Yiamouyiannis also linked fluoride to oral cancers as did the NTP study. It should be remembered that residents of unfluoridated communities will also have a great deal of fluoride exposure from other sources, such as beverages high in fluoride, soft drinks, tea, and reconstituted juices made with fluoridated water. In addition, very high levels of fluoride found in most commercial tooth paste, available in the United States, are ingested by small children who may swallow as much as 100% of the substance. It

is estimated that residents of unfluoridated communities have about 50% of the exposure to fluoride as residents of fluoridated communities.

Dr. Cohn's study can be criticized for all the same reasons as almost every single dental decay study. It is an epidemiological study with confounding variables. Decay studies that have not adjusted for confounding variables are useless in answering the question of effectiveness. Never-the-less, Peebles', 1974, badly flawed study of fluoride supplementation effects on tooth decay can be relied upon to discover the prevalence of dental fluorosis. The fluorosis was mostly mild to very mild from "optimal" controlled doses.[89]

Mahoney (1991) found that in the United States bone cancers in males had increased significantly since 1955. They concluded that water was not the source of the increase, but since their study had no unexposed controls, this conclusion does not seem justified. The largest study of osteosarcoma in young males to date was conducted by the Center for Disease Control. They concluded that nationwide osteosarcoma is not increased by water fluoridation. By correcting the CDC data for age, the results indicated a 68% greater incidence of osteosarcoma in young men in fluoridated communities than unfluoridated ones.[90]

In the case of bone and oral cancers the research appears to bear out the thesis that chronic exposure to fluoride causes sex related cancer in young men and oral cancers in both men and women.

The Battelle Columbus Laboratory NTP report on fluoride found that the high dose rats and mice both developed hepatocholangiocarcinomas. Dr. Mel Ruber, the pathologist credited with originally describing hepatocholangiocarcinomas, reviewed the Battelle pathological slides and confirmed the correct diagnosis of liver cancer. According to sworn testimony, Dr. William Marcus Senior Science Advisor at the Environmental Protection Agency, Office of Drinking Water, this rare form of liver cancer alone is significant, and "This changes the equivocal findings of the board (US PHS) to at least some evidence or clear evidence of carcinogenicity."

The liver cancer diagnosis was downgraded by the U. S. Public Health Department "Peer Review" of the Battelle study. According to Dr. Marcus the downgrade was not justified.[91] In addition, three of the four in-vitro studies were positive for carcinogenesis. The laboratory studies, combined with in-vitro studies indicating carcinogenesis and epidemiological studies, indicate that fluoride, in all probability, is a cancer producing substance.

The alteration of the board certified pathologists interpretation of the slides and the refusal of the U.S. PHS to allow "peer review committee" to view the slides in order to make their determination, is why it is best to review the data personally and arrive at reasonable decisions.

Hip Fracture

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In 1990, Bailey et al. concluded, as have three other U. S. studies, that fluoride "therapy" (40 to 60 mg/daily) may be implicated in the pathogenesis of hip fractures which may occur in treated patients despite a rapid, marked increase in bone mass.[92] [93] [94] [95] Eight other studies have found a positive correlation to hip fracture and water fluoridation (Ref. #2).

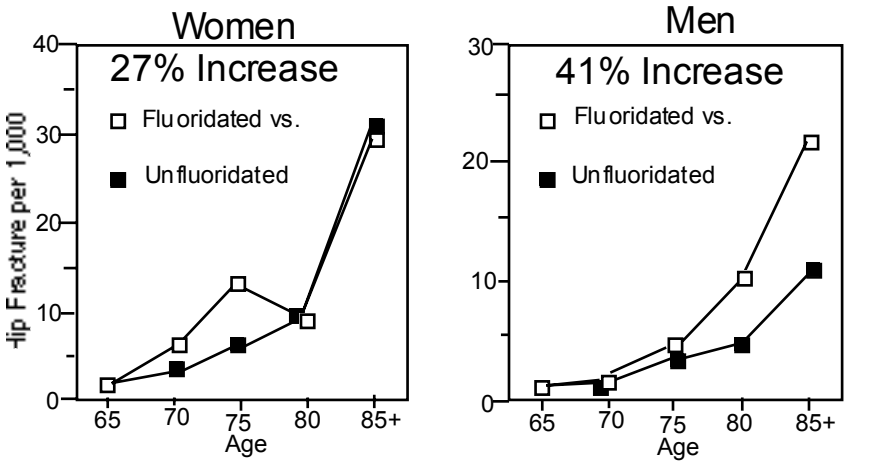
The progression of research published in the Journal of the American Medical Association from 1990 to 1995 is remarkable. The first study to appear was a statistical analysis of the entire U.S. by county, which found a significant positive correlation to water fluoride levels and hip fracture[96]. Cooper initially in 1990 did not find a correlation to water fluoride levels and hip fracture rates.[97] Later, when weighted for population size, he did find an increase.[98]

The third study is a carefully designed study that uses age, sex and religion to minimize confounding variables.[99] 70% of the experimental subjects in both the test city and control cities were of the Mormon religion which forbids smoking and drinking. The study, although small in actual numbers of subjects, produces a very clear picture of gradual increase in hip fracture in both male and females over time. Older women appeared unaffected. (Figure 1)

What is even more interesting about the study is the dramatic increase in hip fracture in women after 26 years of exposure who were pre menopausal at the time of fluoridation. This study, although by itself would mean little, when combined with the other studies of hip fracture, appears to accurately present a picture which should be of concern to everyone. Advocates of fluoridation point to only three hip fracture study as proof of fluoride safety for the elderly.[100] [101] [102] All three of these are small studies of elderly women with limited exposure time (6 years).

For example, Cauley's study looked at 1,878 white women aged 65-93 years (mean age =70.9), only 73% of whom had exposure to public drinking water, with a mean exposure time of only 6.0 years. Since bone turnover (remodeling) rate is relatively rapid before menopause and slow

after menopause, fluoride's major effect on bone is most likely to occur during the years before menopause (i.e., before age 45-50), as was clearly shown in Danielson's study. Therefore, these studies may, in fact, be accurate but only for elderly women with very limited exposure time. (See following graph from Danielson et. al JAMA 1992)



All the subjects of the Danielson study were born before fluoridation was introduced and therefore, drank unfluoridated water, breathed unfluoridated air and brushed with unfluoridated toothpaste for the first 40 years of life. Due to the wide spread use of fluoride in the United States, all children raised in this country today will be exposed to much more fluoride than these experimental subjects. All sources of fluoride exposure are cumulative.

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Dental Fluorosis

In the 1930's and 1940's H. Trendley Dean surveyed 65 cities for the prevalence of dental fluorosis. He reported on 21 of the survey cities and concluded that dental fluorosis would not occur in cities fluoridated at 1 part per million (ppm). No modern research has been able to confirm this optimistic view. To test the effectiveness of fluoride tablets, children were given 1 milligram tablets. This dose was selected because it provides the same dose of fluoride found in a glass of water. The research found that 67% of the children developed dental fluorosis.

Research clearly indicates that minority children and the undernourished will suffer dental fluorosis at, and below, the 1 ppm fluoride level.[103] [104] The National Research Council studied the prevalence of dental fluorosis and found that it has increased dramatically over the last 50 years that this country has experimented with fluoridation.[105] Presently, the incidence of fluorosis in fluoridated communities varies between 8% and 51%, and has risen in poverty areas to as much as 80%. In unfluoridated communities, between 3 and 26% of the children will display the first outwardly visible signs of fluoride poisoning. This is clear evidence that

fluoride exposure has increased all over this country, both in fluoridated and unfluoridated communities, and has in many children exceeded the toxic effect level.

Dental fluorosis is symptomatic of an over-exposure to fluoride throughout the body. Its visible characteristics are the discoloration or pitting of the teeth. White flecks in the teeth may also occur. Fluorosis can lead to tooth decay. FDA's claim that fluorosis is only a "cosmetic" effect is unsubstantiated. It effects all age groups with both long and short-term harmful health consequences.

Most fluoride proponents are preoccupied with fluoride as a "cosmetic effect" of no consequence to health. They are oblivious to the fact that fluorosis connotes fluoride toxicity far more important than mere dental disfigurement. According to Dr. J. Colquhoun, former Chief Dental Officer of Auckland, N.Z.: the claim that only tooth-forming cells are damaged by fluoride is extremely implausible, contrary to common sense, and can be disputed on scientific grounds. There is evidence of more general harm."

The hip fracture is most likely due to fluorosis of the bone. We don't as yet know how high the hip fracture rate will be for children who suffer dental fluorosis, and who will be exposed to a lifetime of highly variable amounts of fluoride. Presumably it will be much higher than their unfluoridated predecessors.

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Tooth Decay: (Not a Determining Factor for Safety of PHG)

All of the recent large-scale studies on the relationship between drinking water fluoridation and tooth decay show that **fluoridation does not affect tooth decay.**[106]

A careful review of the available literature failed to find even one random blinded tooth decay study of humans or animals where water fluoridated at 1 ppm significantly reduced caries incidence.

On the contrary, there are several large studies of humans that have reported no significant difference in decay rates of adult teeth. "When the socioeconomic variable is allowed for, child dental health appears to be better in the unfluoridated areas.[107] "Survey results in British Columbia with only 11% of the population using fluoridated water show lower DMFT rates than provinces with 40-70% of the population drinking fluoridated water."[108] and "school districts recently reporting the highest caries-free rates in the province were totally unfluoridated."[109]

The largest study of tooth decay in North America was done in 1986-1987 by the worlds greatest proponents of drinking water fluoridation, the National Institute of Dental Research, who has lobbied continuously for the last fifty years for total drinking water fluoridation in the United States. 39,000 children between the ages of 5 to 17 from 84 cities were surveyed. Three types of communities were selected for study; fluoridated, partially fluoridated, and unfluoridated. No statistically significant difference was found in decayed, missing and filled permanent teeth (DMFT).[110] (Figure 2)

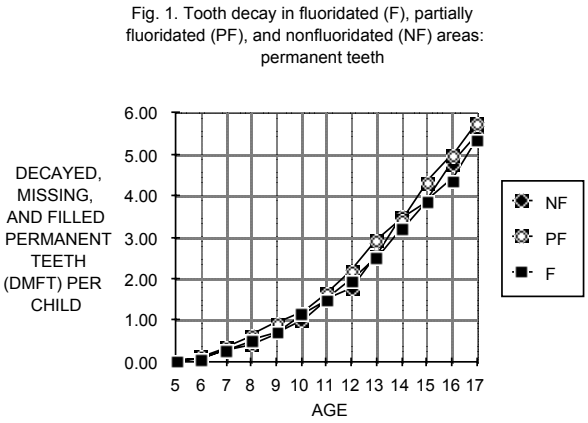

Figure 2

The data from the six cities of California that were studied in the previous survey, when analyzed separately, shows that after 44 years of water fluoridation there is no statistically significant difference in the DMFT rate for the two largest California cities. (Figure 3) The highest decay rate is seen in low income areas such as Cutler/Orsi. San Francisco, fluoridated since 1952, fared no better than non fluoridated Lodi. non fluoridated Los Angeles is not statistically different from affluent San Francisco.

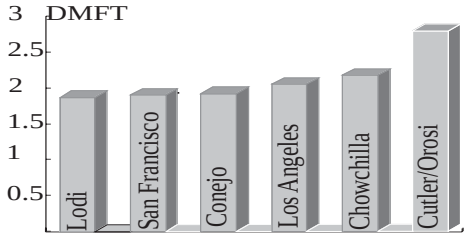

Figure 3

In the largest study of tooth decay and water fluoridation, Dr. Colquhoun, former New Zealand dental officer and past President of the Fluoridation Society, compared the decay rate of 30,000 children in New Zealand. Official statistics showed no difference in the dental status of children in fluoridate and unfluoridated communities.[111]

Tooth decay is known to be an infection of the tooth caused by the bacteria strep mutans. Tooth decay has declined throughout the United States since the 1940's both in fluoridated and non fluoridated areas. It

varies with nutrition, parental education, family income, oral bacteria, oral hygiene and several other factors. Consequently, the DMFT rate will vary in the United States from one community to another. Accurate comparison of decay rates must therefore be adjusted for these confounding factors.

In order to determine if there is an economic benefits of water fluoridation to the government of California, we analyzed California dental cost data for welfare recipients. The study represents two equal socioeconomic groups since participation in the program is dependent upon family income and monitored by the welfare eligibility rules. Welfare dental fees are also the same in all areas of the state. The 1994 weighted average annual cost of dental care in the fluoridated communities of California (90% or more drinking fluoridated water) was $120.01 per eligible recipient and $108.48 in the non fluoridated (0% fluoridated drinking water). The 1995 weighted average annual cost of dental care in the fluoridated communities of California (90% or more drinking fluoridated water) was $125.27 per eligible recipient and $110.06 in the non fluoridated (0% fluoridated drinking water).[112]

Proponents of water fluoridation argue that the reason no benefit was found is because fluoride is available from many other sources such as beverages bottled in fluoridated communities and tooth paste. If this explanation were true, it is also a reason to not fluoridate drinking water.

Dr. Yiamouyiannis reported that the NIDR data showed a 42% lower decayed, missing, and filled rate for baby teeth (dmft) of children 5 years old but, the difference soon disappeared as the children grew older. By age 8 there was no difference in DMFT score. Further examination of the results indicates that drinking water fluoridation may have produced a statistically significant effect by delaying the eruption of the permanent teeth.[113]

Teachers have reported that children with early eruption of their permanent dentition are the most advanced in their grade level. Brain development and tooth development appear to be parallel. This fact appears to fit disturbingly well with the research reported in 1994 at the International Society for Fluoride Research (ISFR) XX Conference in Beijing which linked dental fluorosis to lower IQ;

Mullinex, Co-founder of the toxicology department at the Harvard Forsyth Dental Research Institute, published a study in neurotoxicology that found fluoride more potent than lead in damage to behavior of experimental animals.[114] The research is further corroborated by the well-

established psychopharmacology of fluoride.[115] Delaying the eruption of permanent teeth may provide transient protection from decay bacteria but the damage to the growth and development of the child does not justify the use of water fluoridation.

Conclusion of Report

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The IAOMT performed the task of reviewing fluoride in a comprehensive, scientific and unbiased manner in accordance with criteria established to assure the protection of the public safety. The International Academy of Oral Medicine and Toxicology review of fluoride and resulting PHG of zero is the only acceptable systemic exposure level to this common xenobiotic.

Submitted by,

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References

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1 Center For Disease Control Morbidity Mortality Weekly Report Recommendations for using Fluoride to Prevent and Control Dental Caries in the United States Aug. 17, 2001 / Vol. 50 / No. RR-14

2 Steelink, C. Letter. Chemical and Engineering News 27 July 1992:2-3

3 Steelink, C. and Jones, T. Tooth decay in Tucson, AZ a 50 year perspective with known risk factors considered. Proceedings of the International Academy of Oral Medicine and Toxicology Annual Scientific Symposium Tucson, AZ 1992.

4 Teotia M, Teotia SP, Singh KP, Endemic chronic fluoride toxicity and dietary calcium deficiency interaction syndromes of metabolic bone disease and deformities in India: year 2000. Indian J Pediatr 1998 May-Jun;65 (3):371-81

5 Kennedy D C and Hirzy J W. Public Health Goal for Ingested Fluoride. Fluoride: 31 (3) 1998.

6 Masters RD, Coplan MJ, Hone BT, and Dykes JE Association of Silicofluoride Treated Water with Elevated Blood Lead Neurotoxicology Vol. 21 #6 pp 1091-1100, 2000

7 Cotton, F. A. and Wilkinson, G. Advanced Inorganic Chemistry. John Wiley & Sons, New York, NY. pp. 546 (1988).

8 Mackay, K. M. and Mackay, R. A. Introduction to Modern Inorganic Chemistry. 4th edition. Prentice Hall, Englewood Cliffs, NJ. pp. 339 (1989)

9 Canadian Public Health Association (CPHA). Canadian public health association. Criteria document in support of a drinking water standard for fluoride. Final report. Chapter 3. Fluoride in the Environment. Canadian Public Health Association, Ottawa, Ont (1979).

4. Canada. Environment Canada and Health Canada. Inorganic Fluorides, Priority Substances List Assessment Report. Ottawa: Supply and Services Canada. Also published in French entitled: Fluorides Inorganiques. DSS Catalogue No. En 40-215/32E. ISBN 0-662-21070-9 (1993).

5. Budavari, S. (Editor). The Merck Index An Encyclopedia of Chemicals, Drugs, and Biologicals Eleventh Edition. Merck & Co., Inc., Rahway, NJ. p 8565 (1989).

6. Reeves, T. G. Water Fluoridation. In: Chapter 15 of Water Quality and Treatment. 4th edition. F. W. Pontius (ed.). McGraw-Hill, Inc., New York, NY (1990).

7. Ripa, L. W. Fluorides. Health and Welfare Canada. Chapter 6 of Preventive Dental Services. 2nd edition. Department of National Health and Welfare, Ottawa, Ont (1988).

8. Review of fluoride benefits and risks. Report of the Ad Hoc Subcommittee on Fluoride of the Committee to Coordinate Environmental Health and Related Programs. U.S. Department of Health and Human Services U.S. Department of Health and Human Services. Public Health Service, Research Triangle Park, NC, (February 1991).

9. Symonds, R.B., Rose, W.I., and Reed, M.H. Contribution of Cl- and F-bearing gases to the atmosphere by volcanoes. Nature 334: 415-418 (1988) (cited in reference no. 4).

10. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for fluorides, hydrogen fluoride, and fluorine (F). ATSDR/TP-91/17, U.S. Public Health Service, [Atlanta, GA] (April 1993).

11. World Health Organization (WHO) Fluorine and Fluorides. Environmental Health Criteria 36. IPCS International Programme on Chemical Safety. World Health Organization. Geneva (1984).

12. Health and Welfare Canada, Yukon Territory. Chemical water analysis relative to surface and drinking waters in the Yukon Territory from the year 1986 to 1989. Provided by Mr. Gordon Wm. Allen, Environmental Health Officer, Environmental Health Division, Medical Services Branch, Health and Welfare Canada, Yukon Territory (1989) (cited in reference no. 39).

13. Environment Canada. Atlantic region. Federal-provincial toxic chemical survey of municipal drinking water sources. Data summary report. Province of Prince Edward Island 1986-1988. Environment Canada, Inland Waters Directorate, Water Quality Branch, Moncton, NB (report IWD-AR-WQB-89-156) (1989) (cited in reference no. 39).

14. Greater Vancouver Regional Water District. 1989 Summary of Chemical and Physical Analysis for the Seymour, Capilano and Coquitlam Water Supplies. Provided by Mr. G.T. Marsh, Greater Vancouver Regional Water District, Burnaby, B.C. (1990) (cited in reference no. 39).

15. Droste, R. L. Fluoridation in Canada as of December 31, 1986. Environmental Health Directorate, Health Protection Branch, Health and Welfare Canada, Ottawa, ON (June 1987).

16. Quebec Ministry of the Environment. Personal communication with S. Théberge concerning Quebec municipalities that fluoridate drinking water and the levels of fluoride found in January, 1989 (1990) (cited in reference no. 39).

17. Ontario Ministry of the Environment. 1989 DWSP Results of Fluoride in Raw and Treated Waters. Printout provided by Mrs. Patricia Lachmaniuk, Water Resources Branch, Ontario Ministry of the Environment. (1990) (cited in reference no. 39).

18. Alberta Environment. Fluoride Summary 1989 - Composite Community Data. Printout provided by G.P. Halina, Senior Technologist, Municipal Branch, Standards and Approvals Division, Environmental Protection Services. (1990) (cited in reference no. 39).

19. Dabeka, R. W. and McKenzie, A.D. Personal Communication (1993) (cited in reference no. 39).

20. Taves, D. R. Dietary intake of fluoride ashed (total fluoride) v. unashed (inorganic fluoride) analysis of individual foods. Br. J. Nutr. 49: 295-301 (1983) (cited in reference no. 39).

21. Dabeka, R. W., Conacher, B. S., Salminen, J., Nixon, G. R., Riedel, G., Crocker, R. and Dubé, G. Survey of bottled drinking water sold in Canada. Part I. Lead, cadmium, arsenic, aluminum, and fluoride. J. AOAC Int. 75: 949-953 (1992).

22. Marier, J.R. Intakes of magnesium and fluoride, and some systemic effects. Proc. Finn. Dent. Soc. 87: 581-594 (1991) (cited in reference no. 39).

23. Schamschula, R.G., Un, P.S., Sugar, E., and Duppenthaler, J.L. The fluoride content of selected foods in relation to the fluoride concentration of water. Acta Physiol. Hung. 72: 217-227 (1988) (cited in reference no. 39).

24. Kumpulainen, J. and Koivistoinen, P. Fluroine in foods. Residue Rev. 68: 37-57 (1977) (cited in reference no. 39).

25. McKnight-Hanes, M.C. Leverett, D.H., Adair, S.M., and Shields, C.P. Fluoride content of infant formulas: soy-based formulas as potential factor in dental fluorosis. Pediatr. Dent. 10: 189-194 (1988) (cited in reference no. 39).

26. Dabeka, R.W., Karpinski, K.F., McKenzie, A.D., and Bajdik, C.D. Survey of lead, cadmium and fluoride in human milk and correlation of levels with environmental and food factors. Food Chem. Toxic. 24: 913-921 (1986) (cited in reference no. 39).

27. McGrath, T.M. Assessment of Fluoride Exposure in Populations Residing Close to Fluoride Emitting Brick Plants. Ontario Ministries of Environment, Health and Labour. Toronto: Ministry of Labour, Special Studies and Services Branch (1983) (cited in reference no. 39).

28. Sidhu, S.S. Fluoride deposition through precipitation and leaf litter in a boreal forest in the vicinity of a phosphorus plant. Sci. Total Environ. 23: 205-214 (1982) (cited in reference no. 39).

29. Schuppli, P.A. Total fluorine in CSSC reference soil samples. Can. J. Soil Sci. 65: 605-607 (1985) (cited in reference no. 39).

30. Newbrun, E. Current regulations and recommendations concerning water fluoridation, fluoride supplements, and topical fluoride agents. J. Dent. Res. 71: 1255-1265 (1992).

31. Stookey, G.K. Review of benefits vs. fluorosis risk of self-applied topical fluorides (dentrifices, mouth rinses, gels). presented at the workshop, "Evaluation of Current Recommendations Concerning Fluorides". Toronto, Ontario. April 9-11, 1992 (1992).

32. Stookey, G.K. Review of fluorosis risk of self-applied topical fluorides - dentrifice, mouth rinses, and gels. Community Dentistry and Oral Epidemiology. 22(3): 181-186 (1994).

33. Burgess, R.C. Fluoride ingestion from dental products. In: Investigation of Inorganic Fluoride and its effects on the occurrence of Dental Caries and Dental Fluorosis in Canada. Report prepared for Health and Welfare Canada under research contract no. 3726 (1993).

34. Beltran, E.D., Szpunar, S.M. Fluoride in toothpaste for children: suggestions for change. Pediatric Dent. 3: 185-188 (1988) (cited in reference no. 33).

35. Levy, S.M. A review of fluoride intake from fluoride dentrifice. J. Dent. Child. 60: 115-124 (1993).

36. Clark, D.C. Appropriate uses of fluorides for children: guidelines from the Canadian Workshop on the Evaluation of Current Recommendations Concerning Fluorides. Can. Med. Assoc. J. 149(12): 1787-1793 (1993).

37. Canadian Paediatric Society (CPS). Nutrition Committee. Fluoride Supplementation. Canadian Paediatric Society Statement: N 86-01. Ottawa, Ont. (1986).

38. McFarlane, D.J. (former Senior Dental Consultant, Ontario Ministry of Health). Personal Communication (1993) (cited in reference no. 33).

39. Health Canada. Inorganic Fluorides. Supporting Documentation, Health Related Sections for Priority Substance Assessment Report. (unpublished, 1993).

40. Health Canada. Canadian Environmental Protection Act. Human Health Risk Assessment for Priority Substances. Ottawa: Supply and Services Canada. Also published in French entitled: L'évaluation du risque à la santé humaine des substances d'intérêt prioritaire. DSS Catalogue No. En 40-215/41E. ISBN 0-662-22126-5 (1994).

41. Whitford, G.M. Allman, D.W., and Shahed, A.R. Topical fluorides: effects on physiologic and biochemical process. J. Dent. Res. 66: 1072-1078 (1987) (cited in reference no. 33).

42. Greenberg, A.E., Clesceri, L.S., and Eaton, A.D. (editors) Standard Methods for the Examination of Water and Wastewater. 18th edition. American Public Health Association. American Water Works Association. Water Environment Federation. Washington, D.C. (1992).

43. Tillman, G.M. Water Fluoridation. Operator's Guide Series. Lewis Publishers. Boca Raton, Fl. (1993).

44. Leung, D.C.W. and Hrudey, S.E. Removal of Fluorides from Water Supplies. Report prepared for Standards and Approvals Division, Municipal Engineering Branch, Alberta Environment (July, 1985).

45. Environmental Health Directorate. Guidelines for Canadian Drinking Water Quality. Water Treatment Principles and Applications. A Manual for the Production of Drinking Water. Health Protection Branch, Department of National Health and Welfare. Canadian Water and Wastewater Association. Ottawa, Ont. (1993).

46. Ontario Ministry of the Environment. De-fluoridation of potable water. Water Resources Branch. Toronto, Ont. (unpublished - 1992).

47. Health and Welfare Canada. Recommended Nutrient Intakes for Canadians. Health Protection Branch, Department of National Health and Welfare. Ottawa (1983).

48. Health and Welfare Canada. Nutrition Recommendations. The Report of the Scientific Review Committee. Supply and Services Canada. Ottawa. pp. 160 (1990).

49. United States National Research Council, Food and Nutrition Board. Recommended dietary allowances. 10th edition. National Academy Press. Washington, D.C. (1989) (cited in reference no. 181).

50. Ekstrand, J. Pharmacokinetic aspects of topical fluorides. J. Dent. Res. 66: 1061-1065 (1987) (cited in reference no. 39).

51. Ekstrand, J. and Ehrnebo, M. Influence of milk products on fluoride bioavailability in man. Eur. J. Clin. Pharmacol. 16: 211-215 (1979) (cited in reference no. 39).

52. Sato, T., Yoshitake, K., and Hitomi, G. Mechanisms of fluoride absorption from the gastrointestinal tract in rats. Fluoride Research 1985. Studies in Environmental Science. 27: 325-332 (1986) (cited in reference no. 39).

53. Messer, H.H., Nopakum, J., and Rudney, J.D. Influence of pH on intestinal fluoride transport in vitro. J. Dent. Assoc. Thai. 39: 226-232 (1989) (cited in reference no. 39).

54. United States Environmental Protection Agency (US EPA). Drinking Water Criteria Document on Fluoride. U.S. EPA Office on Drinking Water, Cincinnati, OH (contract 68-03-3279) (1985).

55. Spencer, H., Kramer, L., Norris, C., and Wiatrowski, E. Effect of aluminum hydroxide on fluoride metabolism. Clin. Pharmacol. Ther. 28: 529-535 (1980) (cited in reference no. 39).

56. Carlson, C., Armstrong, W., and Singer, L. Distribution and excretion of radiofluoride in the human. Proc. Soc. Exp. Biol. Med. 104: 235-239 (1960) (cited in reference no. 39).

57. Ekstrand, J., Alvan, G., Boreus, L.O., and Norlin, A. Pharmacokinetics of fluoride in man after single and multiple oral doses. Europ. J. Clin. Pharmacol. 12: 311-317 (1977) (cited in reference no. 54).

58. Ekstrand, J. Odont, L., and Ehrnebo, M. The relationship between plasma fluoride, urinary excretion rate and urine fluoride concentration in man. J. Occup. Med. 25: 745-748 (1983) (cited in reference no. 54).

59. McIvor, M.E. Acute fluoride toxicity: pathophysiology and management. Drug Safety 5: 79-85 (1990) (cited in reference no. 39).

60. Nedeljkovic, M., Matovic, V., and Maksimovic, M. Toxicokinetic studies of fluoride in rabbits. In Nutrient Availability: Chemical and Biological Aspect. Southgate, D.A., Johnson, I.T., Fenwick, G.R. (editors). Royal Society of Chemistry Publication No. 72, pp. 290-292 (1989) (cited in reference no. 39).

61. Hodge, H.C. and Smith, F.A. Biological Properties of Inorganic Fluorides. In: Fluorine Chemistry. Simons, J.H. (editor) pp. 1-43. Academic Press. New York (1965) (cited in reference no. 39).

62. Hamilton, M. Water fluoridation: a risk assessment perspective. J. Environ. Health 54: 27-32 (1992) (cited in reference no. 39).

63. Kaminsky, L.S., Mahoney, M.C., Leach, J., Melius, J., and Miller, M.J. Fluoride: Benefits and risks of exposure. Crit. Rev. Oral Biol. Med. 1: 261-281 (1990) (cited in reference no. 39).

64. Grandjean, P. and Thomsen, G. Reversibility of skeletal fluorosis. Br. J. Ind. Med. 40: 456-461 (1983) (cited in reference no. 39).

65. Grynpas, M.D. Fluoride effects on bone crystals. J. Bone Min. Res. 5: S169-S175 (1990) (cited in reference no. 39).

66. Caraccio, T.P., Greensher, J., and Mofenson, H.C. The Toxicology of Fluoride. In: Clinical Management of Poisoning and Drug Overdose. Haddad, L. and Winchester, J. W.B. Saunders Co. Philadelphia, P.A. (1983) (cited in reference no. 39).

67. Whitford, G.M. The physiological and toxicological characteristics of fluoride. J. Dent. Res. 69: 539-549 (1990).

68. Hodge, H.C. and Smith, F.A. Minerals: Fluorine and dental caries. In: Dietary chemicals vs. dental caries. Advances in chemistry series. No. 94. American Chemical Society. Washington, DC. (1970) (cited in reference no. 39).

69. Weatherell, J.A., Deutsch, D., Robinson, C., and Hallsworth, A.S. Assimilation of fluoride by enamel throughout the life of the tooth. Cares Res. 11(Suppl. 1): 85-115 (1977) (cited in reference no. 11).

70. Schamschula, R.G., Sugar, E., Agus, H.M., Un, P.S.H., and Toth, K. The fluoride content of human tooth enamel in relation to environmental exposure to fluoride. Aust. Dent. J. 24(4): 243-247 (1982) (cited in reference no. 11).

71. Alhava, E.M., Olkkonen, H., Kauranen, P., and Kari, T. The effect of drinking water fluoridation on the fluoride content, strength and mineral density of human bone. Acta Orthop. Scand. 51: 413-420 (1980) (cited in reference no. 39).

72. Berndt, A.F. and Stearns, R.I. Dental Fluoride Chemistry. Charles C. Thomas. Springfield, IL. (1979) (cited in reference no. 39).

73. United States National Academy of Sciences (US NAS). Fluorides, Biologic Effects of Atmospheric Pollutants. National Academy of Sciences. Washington, D.C. (1971).

74. Van Rensburg, B.G. Metabolism of Fluorides. Tydskr. Tandheelkd. Ver. S. Afr. 34: 163-166 (1979) (cited in reference no. 8).

75. Ekstrand, J., Hardell, L.I., et al. Fluoride Balance Studies on Infants in a 1ppm-Water-Fluoride Area. Caries Res. 18(4): 87-92 (1984) (cited in reference no. 8).

76. Ekstrand, J., Spak, C.J., et al. Distribution of Fluoride to Human Breast Milk Following Intake of High Doses of Fluoride. Caries Res. 18(1): 93-95 (1984) (cited in reference no. 39).

77. Spencer, H., Osis, D., and Lender, M. Studies of fluoride metabolism in man: a review and report of original data. Sci. Total Environ. 17: 1-12 (1981) (cited in reference no. 39).

78. Spencer, H., Lewin, I., Wistrowski, E., and Samachson, J. Fluoride metabolism in man. Am. J. Med. 49: 807-813 (1970) (cited in reference no. 39).

79. Augenstein, W.L., Spoerke, D.G., Kulig, K.W., Hall, A.H., Hall, P.K., Riggs, B.S., Saadi, M.E., and Rumack, B.H. Fluoride ingestion in children: A review of 87 cases. Pediatrics 88: 907-912 (1991) (cited in reference no. 39).

80. Lim, J.K.J., Jensen, G.K. and King, O.H.Jr. Some toxicological aspects of stannous fluoride after ingestion as a clear, precipitate free solution compared to sodium fluoride. J. Dent. Res. 54: 615-625 (1975) (cited in reference no. 109).

81. Segreto, B.A., Yeary, R.A., Broks, R., and Harris, N.O. Toxicity study of stannous fluoride in Swiss strain mice. J. Dent. Res. 40: 623- (1960) (cited in reference no. 109).

82. Lim, J.K., Renaldo, G.J., and Chapman, P. LD50 of SnF2, NaF and Na2PO3F in the Mouse Compared to the Rat. Caries Res. 12: 177-179 (1978) (cited in reference no. 67).

83. Gruninger, S.E., Clayton, R., Chang, S.-B., and Siew, C. Acute Oral Toxicity of Dentrifice Fluorides in Rats and Mice. J. Dent. Res. 67: 334. Abst. No. 1769 (1988) (cited in reference no. 67).

84. DeLopez, O.H., Smith, F.A., and Hodge, H.C. Plasma fluoride concentrations in rats acutely poisoned with sodium fluoride. Toxicol. Appl. Pharmacol. 37: 75-83 (1976) (cited in reference no. 10).

85. Skare, J.A., Schrotel, K.R., Nixon, G.A. Lack of DNA-strand breaks in rat testicular cells after in vivo treatment with sodium fluoride. Mutat. Res. 170: 85-92 (1986) (cited in reference no. 10).

86. Whitford, G.M., Finidori, C., and Birdsong-Whitford, N.L. Acute LD50 Values of F Given as NaF and /or MFP in the Rat. Caries Res. 21: 166. Abst. No. 22 (1987) (cited in reference no. 67).

87. Shourie, K.L., Hein, J.W., and Hodge, H.C. Preliminary studies of the caries inhibiting potential and acute toxicity of sodium monofluorophosphate. J. Dent. Res. 29: 529-533 (1950) (cited in reference no. 67).

88. National Toxicology Program (NTP). Toxicology and Carcinogenesis Studies of Sodium Fluoride (CAS No. 7681-49-4) in F344/N rats and B6C3F1 Mice (Drinking Water Studies). United States Department of Health and Human Services, Public Health Service, National Institutes of Health. Research Triangle Park, NC. (NTP TR 393) (1990).

89. Maurer, J.K., Cheng, M.C., Boysen, B.G., and Anderson, R.L. Two-year carcinogenicity study of sodium fluoride in rats. J. Natl. Cancer Inst. 82: 1118-1126 (1990) (cited in reference no. 39).

90. Maurer, J.K., Cheng, M.C., Boysen, B.G., Squire, R.A., Stranberg, J.D., Weisbrode, J.L., and Anderson, R.L. Confounded carcinogenicity study of sodium fluoride in CD-1 mice. Reg. Toxicol. Pharmacol. 18: 154-168 (1993) (cited in reference no. 39).

91. Gruber, H.E. and Baylink, D.J. The effects of fluoride on bone. Clin. Ortho. 267: 264-277 (1991) (cited in reference no. 39).

92. Hodge, H.C. and Smith, F.A. Fluoride. In: Disorders of Mineral Metabolism. Bonner, F. and Colburn, J.W. (editors). Vol. 1. Academic Press, NY. pp 439-483 (1981) (cited in reference no. 8).

93. Sowers, M., Wallace, R.B., and Lemke, J.H. The relationship of bone mass and fracture history to fluoride and calcium intake: a study of three communities. Am. J. Clin. Nutr. 44: 889-898 (1986) (cited in reference no. 8).

94. Schlesinger, E.R., Overton, D.E., Chase, H.C., and Cantwell, K.T. Newburgh-Kingston caries-fluorine study XIII. Pediatric findings after ten years. Am. Dent. Assoc. 52: 296-306 (1956).

95. McCauley, H.B. and McClure, F.J. Effect of Fluoride in Drinking Water on the Osseous Development of the Hand and Wrist in Children. Pub. Health Rep. 69: 671-682 (1954) (cited in reference no. 8).

96. Leone, L.C., Stevenson, C.A., Hilbish, T.F., and Sosman, M.C. A roentgenologic study of a human population exposed to high-fluoride domestic water. Am. J. Roent. 74: 874-885 (1955) (cited in reference no. 39).

97. Stevenson, C.A. and Watson, A.R. Fluoride Osteosclerosis. Am. J. Roentgenol. 78:13-18 (1957) (cited in reference no. 8).

98. Krishnamachari, K.A.V.R. Fluorine. Trace Elements in Human and Animals Nutrition 1: 365-415 (1987) (cited in reference no. 39).

99. Royal College of Physicians of London. Fluoride, Teeth and Health. A Report and Summary on Fluoride and its Effects on Teeth and Health. The Royal College of Physicians of London. Pitman Medical. Great Britain. pp. 85 (1976) (cited in reference no. 39).

100. Singh, A. and Jolly, S.S. Chronic toxic effects on the skeletal system. In: Fluorides and Human Health. World Health Organization. Monograph Series. No. 59. pp. 238-249 (1970).

101. Felsenfeld, A.J. and Roberts, M.A. A report of fluorosis in the United States secondary to drinking well water. J.A.M.A. 265: 486-488 (1991).

102. Sauerbrunn, B.J.L., Ryan, C.M., and Shaw, J.F. Chronic Fluoride Intoxication with Fluorotic Radiculomyelopathy. Ann. Intern. Med. 63(6): 1074-1078 (1965).

103. Goldman, S.M., Sievers, M.L., and Templin, D.W. Radiculomyopathy in a Southwestern Indian Due to Skeletal Fluorosis. Ariz. Med. 28(9): 675-677 (1971).

104. Fisher, R.L., Medcalf, T.W., and Henderson, M.C. Endemic Fluorosis with Spinal Cord Compression: A Case Report and Review. Arch. Intern. Med. 149(3): 697-700 (1989).

105. Fisher, J.R., Sievers, M.L., Takeshita, R.T., and Caldwell, H.. Skeletal Fluorosis From Eating Soil. Ariz. Med. 38(11): 833-835 (1981).

106. Bruns, B.R. and Tytle, T. Skeletal Fluorosis. A Report of Two Cases. Orthopedics 11(7): 1083-1087 (1988).

107. Power, G.R.I. and Gay, J.D.L. Sodium fluoride in the treatment of osteoporosis. Clin. Inves. Med. 9: 41-43 (1986) (cited in reference no. 35).

108. Kleerekoper, M. and Balena, R. Fluorides and osteoporosis. Annu. Rev. Nutr. 11: 309-324 (1991) (cited in reference no. 39).

109. International Agency for Research on Cancer (IARC). some Aromatic Amines, Anthroquinones and Nitroso Compounds, and Inorganic Fluorides Used in Drinking-water and Dental Preparations. Vol. 27. IARC Monographs on the Evaluation of Carcinogenic Risks of Chemicals to Humans. Lyon, France (1982).

110. Keller, C. Fluorides in Drinking Water. Paper presented at the Workshop on Drinking Water Fluoride Influence on Hip Fracture and Bone Health. April 10, 1991. Bethesda, MD. (1991) (cited in reference no. 175).

111. May, D.S. and Wilson, M.G. Hip Fractures in Relation to Water Fluoridation: An Ecologic Analysis.Paper presented at the Workshop on Drinking Water Fluoride Influence on Hip Fracture and Bone Health. April 10, 1991. Bethesda, MD. (1991) (cited in reference no. 175).

112. Jacobsen, S.J., Goldberg, J., Cooper, C., Lockwood, S.A. 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) (cited in reference no. 39).

113. Jacobsen, S.J., O'Fallon, M., and Melton, L.J. Hip fracture incidence before and after fluoridation of the public water supply, Rochester, Minnesota. Am. J. Public Health. 83: 743-745 (1993) (cited in reference no. 39).

114. Danielson, C., Lyon, J.L., Egger, M., and Goodenough, G.K. Hip fracture and fluoridation in Utah's elderly population. J.A.M.A. 268: 746-748 (1992) (cited in reference no. 175).

115. Suarez-Almazor, M., Flowerdew, G., Saunders, D., Soskoline, C.L., Russel, A.S. The Fluoridation of drinking water and hip fracture hospitalization rates in two Canadian communities. Am. J. Public Health. 83: 689-693 (1993).

116. Sowers, M., Clark, M.K., Jannausch, M.L., and Wallace, R.B. A Prospective study of bone mineral content and fracture in communities with differential fluoride content. Am. J. Epidemiol. 133: 649-660 (1991) (cited in reference no. 39).

117. Cauley, J.A., Murphy, P.A., Riley, T., and Black, D. Public health bonus of water fluoridation: Does fluoridation prevent osteoporosis and its related fractures. Am. J. Epidemiol. 134: 768 (1991) (cited in reference no. 175).

118. Inkovaara, J.A. Is fluoride treatment justified today? Calcif. Tissue Int. 49 (suppl): S68-S69 (1991) (cited in reference no. 39).

119. Mamelle, N., Dusan, R., Martin, J.L., Prost, A., Meunier, P.J., Guillame, M., Guacher, A., and Zeigler, G. Risk-benefit ratio of sodium fluoride treatment in primary vertebral osteoporosis. Lancet. August 13: 361-365 (1988) (cited in reference no. 39).

120. Hedlund, L.R. and Gallagher, J.C. Increased incidence of hip fracture in osteoporotic women treated with sodium fluoride. J. Bone Min. Res. 4: 223-225 (1989) (cited in reference no. 39).

121. Riggs, B.L., Hodgson, S.F., O'Fallon, W.M., Chao, E.Y.S., Wahner, H.W., Muhs, J.M., Cedel, S.L., and Melton, L.J. Effect of fluoride treatment on the fracture rate in postmenopausal women with osteoporosis. New Engl. J. Med. 322: 802-809 (1990) (cited in reference no. 39).

122. Messer, H.H., Armstrong, W.D., and Singer, L. Influence of fluoride intake on reproduction in mice. J. Nutri. 103: 1319-1326 (1973) (cited in reference no. 39).

123. Tao, S. and Suttie, J.W. Evidence for lack of an effect of dietary fluoride level on reproduction in mice. J. Nutr. 106: 1115-1122 (1976) (cited in reference no. 39).

124. Pillai, K.S., Mathai, A.T., and Deshmuhk, P.B. Effect of fluoride on reproduction in mice. Fluoride 22: 165-168 (1989) (cited in reference no. 39).

125. Chinoy, N.J., Sequeira, E., and Narayanam, M.V. Effect of vitamin C and calcium on the reversibility of fluoride-induced alterations in spermatozoa of rabbits. Fluoride 24: 29-39 (1991) (cited in reference no. 39).

126. Chinoy, N.J. and Sequeira, E. Reversible fluoride induced fertility impairment in male mice. Fluoride 25(2): 71-76 (1992).

127. Aulerich, R.J., Napolitano, A.C. Bursian, S.J., Olson, B.A., and Hochstein, J.R. Chronic toxicity of dietary fluoride. J. Animal Sci. 65: 1759-1767 (1987) (cited in reference no. 39).

128. Susheela, S.K. and Kumar, A. 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. Fertil. 92: 353-360 (1991) (cited in reference no. 39).

129. Chinoy, N.J. and Sequeira, E. Effects of fluoride on the histoarchitecture of the reproductive organs of the male mouse. Reprod. Toxicol. 3: 261-267 (1989) (cited in reference no. 39).

130. Chinoy, N.J. and Sequeira, E. Fluoride induced biochemical changes in reproductive organs of male mice. Fluoride 22: 79-85 (1989) (cited in reference no. 39).

131. Kour, K. and Singh, J. Histological finding of mice testes following fluoride ingestion. Fluoride 13: 160-162 (1980) (cited in reference no. 39).

132. Aschengrau, A., Zierler, S., and Cohen, A. Quality of community drinking water and the occurrence of spontaneous abortion. Arch. Environ. Health 44: 283-290 (1989) (cited in reference no. 39).

133. Zierler, S., Theodore, M., Cohen, A., Rothman, K.J. Chemical quality of maternal drinking water and congenital heart disease. Int. J. Epidemiol. 17: 589-594 (1988) (cited in reference no. 39).

134. Aschengrau, A., Zierler, S., and Cohen, A. Quality of community drinking water and the occurrence of late adverse pregnancy outcomes. Arch. Environ. Health. 48: 105-113 (1993) (cited in reference no. 39).

135. Freni, S.C. Exposure to High fluoride concentrations in drinking water is associated with decreased birth rates. J. Toxicol. Environ. Health. 42: 109-121 (1994).

136. Glenn, F.B, Glenn, W.D., and Duncan, R.C. Fluoride table supplementation during pregnancy for caries immunity: A study of the offspring produced. Am. J. Obstet. Gynecol. 143: 560-564 (1982) (cited in reference no. 39).

137. Moriya, M., Ohta, T., Watanabe, K., Miyazawa, T., Kato, K., and Shirasu, Y. Further mutagenicity studies on pesticides in bacterial reversion assay systems. Mutat. Res. 116: 185-216 (1983) (cited in reference no. 39)

138. Tong, C.C., McQueen, C.A., Ved Brat, S., and Williams, G.M. The lack of genotoxicity of sodium fluoride in a battery of cellular tests. Cell Biol. Toxicol. 4: 173-186 (1988) (cited in reference no. 39).

139. Li, Y., Dunipace, A.J., and Stookey, G.K. Absence of mutagenic or antimutagenic activities of fluoride in Ames Salmonella assays. Mutat. Res. 190: 229-236 (1987) (cited in reference no. 39).

140. Gocke, E., King, M.-T., Echardt, K., and Wild, D. Mutagenicity of cosmetic ingredients licensed by the European Communities. Mutat. Res. 90: 91-109 (1981) (cited in reference no. 39).

141. Martin, G.R., Brown, K.S., Matheson, D.W., Lebowitz, H.L., Singer, L., and Ophaug, R. Lack of cytogenetic effects in mice or mutations in Salmonella receiving sodium fluoride. Mutat. Res. 66(2): 159-167 (1979) (cited in reference no. 39).

142. Litton Bionetics. Mutagenic Evaluation of Compound FDA 75-7, 007681-49-4, Sodium Fluoride. Report prepared for the U.S. Food and Drug Administration, Contract 223-74-2104. Kensington, Md.: Litton Bionetics, Inc. (1975) (cited in reference no. 8).

143. Tsutsui, T., Suzuki, N., and Ohmori, M. Sodium fluoride-induced morphological and neoplastic transformation, chromosome aberrations, sister chromatid exchanges and unscheduled DNA synthesis in cultured syrian hamster embryo cells. Cancer Res. 44: 938-941 (1984) (cited in reference no. 39).

144. Jones, C.A., Callahan, M.F. and Huberman, E. Sodium fluoride promotes morphological transformation of syrian hamster embryo cells. Carcinogenesis 9: 2279-2284 (1988) (cited in reference no. 39).

145. Jones, C.A., Huberman, E., Callahan, M.F., Tu, A., Halloween, W., Pallota, S., Sivak, A. Lubet, R.A., Avery, M.D., Kouri, R.E., Spalding, J., and Tennant, R.W. An inter-laboratory evaluation of the syrian hamster embryo cell transformation assay using fourteen coded chemicals. Toxicol. In Vitro. 2: 103-116 (1988) (cited in reference no. 39).

146. Lasne, C., Lu, Y-P., and Chouroulinkov, I. Transforming activities of sodium fluoride in cultured Syrian hamster embryo and BALB/3T3 cells. Cell Biol. and Toxicol. 4: 311-324 (1988) (cited in reference no. 39).

147. Cole, J., Muriel, W.J., and Bridges, B.A. The mutagenicity of sodium fluoride to L5178Y [wild-type and TK+/- (3.7.2C)] mouse lymphoma cells. Mutagenesis 1: 157-167 (1986) (cited in reference no. 39).

148. Caspary, W.J., Myhr, B., Bowers, L., McGregor, D., Riach, C., and Brown, A. Mutagenic activity of fluorides in mouse lymphoma cells. Mutat. Res. 187: 165-180 (1987) (cited in reference no. 39).

149. Caspary, W.J., Langenbach, R., Penman, B.W. Crespi, C., Myhr, B., Mitchell, A.D. The mutagenic activity of selected compounds at the TK locus: rodent vs. human cells. Mutat. Res. 196: 61-81 (1988) (cited in reference no. 39).

150. Crespi, C.L., Seixas, G.M., Turner, T., and Penman, B.W. Sodium fluoride is a less efficient human cell mutagen at low concentrations. Environ. Mol. Mutagenesis 15: 71-77 (1990) (cited in reference no. 39).

151. Moore, M.M., Clive, D., Hozier, J.C., Howard, B.E., Batsun, A.G., Turner, N.N., and Sawyer, J. Analysis of trifluorothymidine resistant (TFT') mutants of L5178Y/TK±mouse lymphoma cells. Mutat. Res. 151: 161-174 (1985) (cited in reference no. 39).

152. Tsutsui, T., Suzuki, N., Ohmori, M., and Maizumi, H. Cytotoxicity, chromosome aberrations and unscheduled DNA synthesis in cultured human diploid fibroblasts induced by sodium fluoride. Mutat. Res. 139: 193-198 (1984) (cited in reference no. 39).

153. Tsutsui, T., Koichi, I., and Maizumi, H. Induction of unscheduled DNA synthesis in cultured human oral keratinocytes by sodium fluoride. Mutat. Res. 140: 43-48 (1984) (cited in reference no. 39).

154. Skare, J.A., Wong, T., Evans, L.B., and Cody, D.B. DNA-repair studies with sodium fluoride: comparative evaluation using density gradient ultracentrifugation and autoradiography. Mutat. Res. 172: 77-87 (1986) (cited in reference no. 39).

155. Tong, C., McQueen, C.A., VedBrat, S., and Williams, G.M. The lack of genotoxicity of sodium fluoride in an in vitro test battery. Environ. Mutagenesis. 8[Suppl. 6]: 86 (abstract) (1986) (cited in reference no. 39).

156. Scott, D. Cytogenetic effects of sodium fluoride in cultured human fibroblasts. Mutagenesis 1: 69 (abstract) (1986) (cited in reference no. 39).

157. Scott, D. and Roberts, S.A. Extrapolation from in vitro tests to human risk: experience with sodium fluoride clastogenicity. Mutat. Res. 189: 47-58 (1987) (cited in reference no. 39).

158. Aardema, M.J., Gibson, D.P., and LeBoeuf, R.A. Sodium fluoride induced chromosome aberrations in different stages of the cell cycle: a proposed mechanism. Mutat. Res. 223: 191-203 (1989) (cited in reference no. 39).

159. Kishi, K. and Tonomura, A. Cytogenic effects of sodium fluoride. Mutat. Res. 130: 367 (abstract) (1984) (cited in reference no. 39).

160. Thomson, E.J., Kilanowski, F.M., and Perry, P.E. The effect of fluoride on chromosome aberration and sister chromatid exchange frequencies in cultured human lymphocytes. Mutat. Res. 144: 89-92 (1985) (cited in reference no. 39).

161. Li, Y., Heerema, N.A., Dunipace, A.J., and Stokey, G.K. Genotoxic effects of fluoride evaluated by sister-chromatid exchange. Mutat. Res. 192: 191-201 (1987) (cited in reference no. 39).

162. Li, J., Suzuki, Y., Hayashi, K., and Shimizu, H. The genotoxic effect of sodium fluoride. Mutat. Res. 252: 95 (abstract) (1991) (cited in reference no. 39).

163. Dominok, B. and Miller, G.W. Effects of fluoride on Drosophila melanogaster in relation to survival and mutagenicity. Fluoride 23: 83-91 (1990) (cited in reference no. 39).

164. Mitchell, B. and Gerdes, R.A. Mutagenic effects of sodium and stannous fluoride on Drosopila melanogaster. Fluoride 6: 113-117 (1973) (cited in reference no. 39).

165. Kram, D., Schneider, E.I., Singer, L., and Martin, G.R. The effects of high and low fluoride diets on the frequencies of sister chromatid exchanges. Mutat. Res. 57: 51-55 (1978) (cited in reference no. 39).

166. Li, Y., Zhang, W., Noblitt, T.W., Dunipace, A.J., and Stookey, G.K. Genotoxic evaluation of chronic fluoride exposure: sister-chromatid exchange study. Mutat. Res. 227: 159-165 (1989) (cited in reference no. 39).

167. Zeiger, E., Shelby, M.D., and Witt, K.L. Genetic toxicity of fluoride. Environ. Molec. Mutagen. 21: 309-318 (1993) (cited in reference no. 39).

168. Li, Y., Dunipace, A.J. and Stookey, G.K. Lack of genotoxic effects of fluoride in the mouse bone-marrow micronucleus test. J. Dent. Res. 66: 1687-1690 (1987) (cited in reference no. 39).

169. Dunipace, A.J., Zhang, W., Noblitt, T.W., Li, Y., and Stookey, G.K. Genotoxic evaluation of chronic fluoride exposure: micronucleus and sperm morphology studies. J. Dent. Res. 68: 1525-1528 (1989) (cited in reference no. 39).

170. Albanese, R. Sodium fluoride and chromosome damage (in vitro human lymphocytes and in vivo micronucleus assays). Mutagenesis 2: 497-499 (1987) (cited in reference no. 39).

171. Li, Y., Dunipace, A.J. and Stookey, G.K. Effects of fluoride on the mouse sperm morphology test. J. Dent. Res. 66: 1509-1511 (1987) (cited in reference no. 39).

172. Pati, P.C. and Bhunya, S.P. Genotoxic effect of an environmental pollutant, sodium fluoride in mammalian in vivo test system. Caryologia 40: 79-87 (1987) (cited in reference no. 39).

173. Ma, J., Cheng, L., Bai, W., and Wu, H. The effects of sodium fluoride on SCEs of mice and on micronucleus of the bone marrow of pregnant mice and fetal liver. Yichuan. 8: 39-41 (1986) (cited in reference no. 39).

174. Hayashi, M., Kishi, M., Ishidate, M. Micronucleus tests in mice on 39 food additives and eight miscellaneous chemicals. Food Chem. Toxicol. 26: 487-500 (1988) (cited in reference no. 39).

175. United States National Research Council (US NRC). Health Effects of Ingested Fluoride. Subcommittee on Health Effects of Ingested Fluoride. National Research Council. National Academy Press. Washington, D.C. U.S.A. (1993).

176. Freni, S.C. and Gaylor, D.W. International trends in the incidence of bone cancer are not related to drinking water fluoridation. Cancer 70: 611-618 (1992) (cited in reference no. 39).

177. Hoover, R.N., De Vesa, SS., Cantor, K.P., Lubin, J.H., Fraumeni, J.F.Jr. Fluoridation of drinking water and subsequent cancer incidence and mortality. (1991) (Appendix E in reference no. 8) (cited in reference no. 39).

178. Hoover, R.N. Devesa, S.S., Cantor, K.P., Faumeni, J.F.Jr. Time trends for bone and joint cancers and osteosarcomas in the Surveillance, Epidemiology and End Results (SEER) Program, National Cancer Institute. (1991) (Appendix F in reference no. 8) (cited in reference no. 39).

179. Knox, E.G. Fluoridation of Water and Cancer: A Review of the Epidemiological Evidence. Report of the British Working Party. Her Majesty's Stationary Office, London, United Kingdom (1985).

180. United States National Academy of Sciences (US NAS). National Research Council. Safe Drinking Water Committee: Drinking Water and Health. National Academy of Sciences Press. Washington, D.C. pp. 381-389 (1977) (cited in reference no. 8).

181. Burt, B.A. The Changing Patterns of Systemic Fluoride Intake. J. Dent. Res. 71 (Spec. Iss.): 1228-1237 (1992).

182. Fejerskov, O., Manji, F., and Baelum, V. The nature and mechanisms of dental fluorosis in man. J. Dent. Res. 69(special iss.): 692-700 (1990).

183. Cutress, T.W. and Suckling, G.W. Differential diagnosis of dental fluorosis. J. Dent. Res. 69(special iss.): 714-721 (1990).

184. Smith, G.E. Fluoride and fluoridation. Soc. Sci. Med. 26(4): 451-462 (1988).

185. Clark, D.C. Trends in Prevalence of Dental Fluorosis in North-America. Community Dentistry and Oral Epidemiology. 22(3): 148-152 (1994).

186. Limeback, H. Enamel formation and the effects of fluoride. Community Dentistry and Oral Epidemiology. 22(3): 144-147 (1994).

187. Evans, R.W. and Stamm, J.W. An epidemiologic estimate of the critical period during which maxillary central incisors are most susceptible to fluorosis. J. Public Health Dent. 51(4): 251-259 (1991).

188. Advisory Review Panel. Recommendations Regarding Fluoride Intake. Investigation of Inorganic Fluoride and Its Effects on the Occurrence of Dental Caries and Dental Fluorosis in Canada. Report prepared for Health and Welfare Canada under research contract no. 3726 (1993).

189. Dean, H.T., Arnold, F.A.Jr., and Elvove, E. Domestic water supplies and dental caries. V. Additional studies of the relation of fluoride domestic waters to caries experience of 4,425 white children, aged 12-14years, of 13 cities and 4 states. Pub. Health Rep. 57: 1155-1179 (1942).

190. Dean, H.T., Jay, P., Arnold, F.A.Jr., and Elvove, E. Domestic water and dental caries. II A study of 2,832 white children aged 12-14 years of 8 suburban Chicago communities. Including Lactobacillus Acdiophilus studies of 1,761 children. Pub. Health. Rep. 56: 761-792 (1941).

191. Clark, D.C. Working group report on the ingestion of inorganic fluoride and its effect on the occurrence of dental caries and dental fluorosis in Canada. In: Investigation of inorganic fluoride and its effects on the occurrence of dental caries and dental fluorosis in Canada. Report prepared for Health and Welfare Canada under research contract no. 3726 (1993).

192. Ismail, A.I. Dental caries, fluorosis, and fluorides. In: Investigation of inorganic fluoride and its effects on the occurrence of dental caries and dental fluorosis in Canada. Report prepared for Health and Welfare Canada under research contract no. 3726 (1993).

193. Eklund, S.A. and Striffler, D.F. Anitcaries effect of various concentrations of fluoride in drinking water: Evaluation of Empirical Evidence. Pub. Health Rep. 95: 486-490 (1980).

194. Thylstrup, A. Clinical evidence of the role of pre-eruptive fluoride in caries prevention. J. Dent. Res. 69 (Spec. Iss.): 742-750 (1990).

195. Beltran, E.D. and Burt, B.A. The Pre- and posteruptive effects of fluoride in the caries decline. J. Pub. Health. Dent. 48(4): 233-240 (1988).

196. Groeneveld, A., Van Eck, A.A.M.J., and Backer Dirks, O. Fluoride in caries prevention: Is the effect pre- or post-eruptive? J. Dent. Res. 69(Spec. Iss.): 751-755 (1990).

197. Grembowski, D., Fiset, L., Spadafora, A. How fluoridation affects adult dental caries. Systemic and topical effects are explored. J. A. D. A. 123: 49-54 (1992).

198. Hunt, R.J. Eldridge, J.B., and Beck, J.D. Effect of residence in a fluoridated community on the incidence of coronal and root caries in an older population. J. Pub. Health Dent. 49: 138-141 (1989).

199. Riordan, P.J. Perceptions of Dental Fluorosis. J. Dent. Res. 72(9): 1268-1274 (1993).

200. Riordan, P.J. Specialist clinicians' perceptions of dental fluorosis. J. Dent. Child. 60(4-5): 315-320 (1993).

201. Clark, D.C. Evaluation of aesthetics for the different classifications of the tooth surface index of fluorosis. Comm. Dent. Oral Epi. 23: 80-83 (1995).

202. Health Canada. Guidelines for Canadian Drinking Water Quality. Sixth Edition. Prepared by the Federal-Provincial Subcommittee on Drinking Water of the Federal-Provincial Committee on Environmental and Occupational Health. Canada Communication Group - Publishing. Ottawa (1996).

203. Health and Welfare Canada. Guidelines for Canadian Drinking Water Quality 1978. Supporting Documentation. Minister of Supply and Services Canada. Ottawa (1980).

10 Canada. Environment Canada and Health Canada. Inorganic Fluorides, Priority Substances List Assessment Report. Ottawa: Supply and Services Canada. Also published in French entitled: Fluorides Inorganiques. DSS Catalogue No. En 40-215/32E. ISBN 0-662-21070-9 (1993).

11 Budavari, S. (Editor). The Merck Index An Encyclopedia of Chemicals, Drugs, and Biologicals Eleventh Edition. Merck & Co., Inc., Rahway, NJ. p 8565 (1989).

12 Reeves, T. G. Water Fluoridation. In: Chapter 15 of Water Quality and Treatment. 4th edition. F. W. Pontius (ed.). McGraw-Hill, Inc., New York, NY (1990).

13 Ripa, L. W. Fluorides. Health and Welfare Canada. Chapter 6 of Preventive Dental Services. 2nd edition. Department of National Health and Welfare, Ottawa, Ont (1988).

14 Review of fluoride benefits and risks. Report of the Ad Hoc Subcommittee on Fluoride of the Committee to Coordinate Environmental Health and Related Programs. U.S. Department of

Health and Human Services U.S. Department of Health and Human Services. Public Health Service, Research Triangle Park, NC, (February 1991).

15 Riggs BL, Hodgson SF, O'Fallon WM, Chao EYS, et al. Effect of fluoride treatment on the fracture rate in postmenopausal women with osteoporosis. NEJM 1990; 322:802-9.
16 Letter dated Dec 21, 2000 to Honorable Congressman Ken Calvert Subcommittee on energy and environment Committee on Science from Food and Drug Administration Associate Commissioner Melinda K. Plaisier http://www.keepers-of-the-well.org
17 Symonds, R.B., Rose, W.I., and Reed, M.H. Contribution of Cl- and F-bearing gases to the atmosphere by volcanoes. Nature 334: 415-418 (1988) (cited in reference no. 4).

18 Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for fluorides, hydrogen fluoride, and fluorine (F). ATSDR/TP-91/17, U.S. Public Health Service, [Atlanta, GA] (April 1993).

19 World Health Organization (WHO) Fluorine and Fluorides. Environmental Health Criteria 36. IPCS International Programme on Chemical Safety. World Health Organization. Geneva (1984).

20 Fluoridation Census 1992 U.S. Department of Health and Human Services CDC
21 Droste, R. L. Fluoridation in Canada as of December 31, 1986. Environmental Health Directorate, Health Protection Branch, Health and Welfare Canada, Ottawa, ON (June 1987).
22 Dabeka, R. W. and McKenzie, A.D. Personal Communication (1993) (cited in reference no. 39).

23 Taves, D. R. Dietary intake of fluoride ashed (total fluoride) v. unashed (inorganic fluoride) analysis of individual foods. Br. J. Nutr. 49: 295-301 (1983)

24 Dabeka, R. W., Conacher, B. S., Salminen, J., Nixon, G. R., Riedel, G., Crocker, R. and Dubé, G. Survey of bottled drinking water sold in Canada. Part I. Lead, cadmium, arsenic, aluminum, and fluoride. J. AOAC Int. 75: 949-953 (1992).

25 Schamschula, R.G., Un, P.S., Sugar, E., and Duppenthaler, J.L. The fluoride content of selected foods in relation to the fluoride concentration of water. Acta Physiol. Hung. 72: 217-227 (1988)

26 Marier, J.R. Intakes of magnesium and fluoride, and some systemic effects. Proc. Finn. Dent. Soc. 87: 581-594 (1991)

27 Kumpulainen, J. and Koivistoinen, P. Fluroine in foods. Residue Rev. 68: 37-57 (1977)
28 McKnight-Hanes, M.C. Leverett, D.H., Adair, S.M., and Shields, C.P. Fluoride content of infant formulas: soy-based formulas as potential factor in dental fluorosis. Pediatr. Dent. 10: 189-194 (1988)

29 Dabeka, R.W., Karpinski, K.F., McKenzie, A.D., and Bajdik, C.D. Survey of lead, cadmium and fluoride in human milk and correlation of levels with environmental and food factors. Food Chem. Toxic. 24: 913-921 (1986).

30 McGrath, T.M. Assessment of Fluoride Exposure in Populations Residing Close to Fluoride Emitting Brick Plants. Ontario Ministries of Environment, Health and Labour. Toronto: Ministry of Labour, Special Studies and Services Branch (1983)

31 Sidhu, S.S. Fluoride deposition through precipitation and leaf litter in a boreal forest in the vicinity of a phosphorus plant. Sci. Total Environ. 23: 205-214 (1982)

32 Schuppli, P.A. Total fluorine in CSSC reference soil samples. Can. J. Soil Sci. 65: 605-607 (1985)

33 Newbrun, E. Current regulations and recommendations concerning water fluoridation, fluoride supplements, and topical fluoride agents. J. Dent. Res. 71: 1255-1265 (1992).

34 Stookey, G.K. Review of fluorosis risk of self-applied topical fluorides - dentrifice, mouth rinses, and gels. Community Dentistry and Oral Epidemiology. 22(3): 181-186 (1994).
35 Burgess, R.C. Fluoride ingestion from dental products. In: Investigation of Inorganic Fluoride and its effects on the occurrence of Dental Caries and Dental Fluorosis in Canada. Report prepared for Health and Welfare Canada under research contract no. 3726 (1993)
36 Beltran, E.D., Szpunar, S.M. Fluoride in toothpaste for children: suggestions for change. Pediatric Dent. 3: 185-188 (1988)
37 Stookey, G.K. Review of benefits vs. fluorosis risk of self-applied topical fluorides (dentrifices, mouth rinses, gels). presented at the workshop, "Evaluation of Current Recommendations Concerning Fluorides". Toronto, Ontario. April 9-11, (1992).
38 Levy, S.M. A review of fluoride intake from fluoride dentrifice. J. Dent. Child. 60: 115-124 (1993).

39 Clark, D.C. Appropriate uses of fluorides for children: guidelines from the Canadian Workshop on the Evaluation of Current Recommendations Concerning Fluorides. Can. Med. Assoc. J. 149(12): 1787-1793 (1993).

40 Canadian Paediatric Society (CPS). Nutrition Committee. Fluoride Supplementation. Canadian Paediatric Society Statement: N 86-01. Ottawa, Ont. (1986).

41 Magda Aelvoet the Minister of Public Health in Belgium bans fluoride supplements Reported in the BBC news Tuesday 30 July 2002
42 Updated 1999 as the NTEU White Paper available from Drs. Hirzy or Kennedy
43 http://www.keepers-of-the-well.org
44 Sutton, P., The Greatest Fraud Fluoridation Kurunda Pty. Ltd 1996

45 Limeback H., Delayed tooth eruption from water fluoridation J Dent Res 1996

46 Fluoride, the Aging Factor, Health Action Press, pp. 72-90 (1993)

47 Mirth et al, 1985 (Caries Research 19:466)

48 Margolis HC, Moreno EC. Physicochemical perspectives on the cariostatic mechanisms of systemic and topical fluorides. J Dental Res 1990:69 (special Issue): 606-13.

49 Hunt & Navia, 1975 (Archs Oral Biol 20:497)

50 Larson et al, 1977 (Archs Oral Biol 22:437)

51 Limebeck Journal of Dental Research

52 Fluoride Vol. 20 #4 Oct, 1987

53 S.S.Jolly, et al., "Endemic Fluorosis in Punjab", Fluoride, Vol. 6 , pp. 4-18 (1973)

54 Journal of the American Medical Association Vol. 273, pp. 775-776 (1995)

55 Li, XS; Zhi, JL; Gao, RO Effects of fluoride exposure on intelligence in children, Fluoride 28:4, pp. 189-192 (1995)

56 Zhao, LB; Liang, D; W Wu Lu-Liang Effects of a high fluoride water supply on children's intelligence. Fluoride vol. 29:4 pp. 190-192 1996

57 Mullenix, Phyllis et. al Neurotoxicity of Sodium Fluoride in Rats, Neurotoxicology and Teratology, Vol. 17, No. 2, pp. 169-177, 1995

58 Guminska and Sterkowicz 1975

59 Peters et al. 1964

60 Slater and Bonner 1952

61 Walker G.S.R. Australia's Fluoridated "Death Valley", The Australian Fluoridation News, 1990; 26:2

62 Health Effects of Ingested Fluoride, National Research Council, page 37 (1993)

63 Op cit Li et al, Zhao et al. and Mullenix et al.

64 Bone Pathology linked to water fluoridation

65 Preface PHG Item 1 Page ii

66 Li, Y., Dunipace, A., Stookey, G. Absence of mutagenic and antimutagenic activities of fluoride in Ames Salmonella assays Mutation Research 190:229-236 (1987)

67 Op Cit. Marcus' May Day 1990 Senior Science Advisor to the EPA Memo

68 L. H. Fountain's Congressional Subcommittee on Intergovernmental Relations (1977)
69References for Fact # 15

1) Carcinogenesis, Vol. 9, pp. 2279-2284 (1988)

2) Sodium Fluoride: individual animal tumor pathology table [rats], Battelle Memorial Institute, February 23, 1989

3) Sodium Fluoride: individual animal tumor pathology table [mice], Battelle Memorial Institute, April 11, 1989

4) Lancet 36, page 737 (1990)

5) Review of Fluoride: Benefits and Risks, U. S. Public Health Service, pp. F1-F7 (1991)

6) Fluoride Vol. 26, pp. 83-96 (1992)

7) A Brief Report on the Association of Drinking Water Fluoridation and the Incidence of Osteosarcoma among Young Males, New Jersey Department of Health, November 1992

8) Fluoride, the Aging Factor, Health Action Press, pp. 72-90 (1993)

70 Phillip R.N. Sutton Fluoride Vol. 20 #4 October, 1987

71 Zipkin, L., McClure, F.J., Leone, N.C., and Lee, W.A. 1958. Fluoride deposition in human bones after prolonged ingestion of fluoride in drinking water. Public Health Rep. 73, 732-740.

72 Alhava EM et al. The effect of drinking water fluoridation on the fluoride content, strength and mineral density of human bone. Acta Orthop Scand; 51:413-20, 1980

73 Table 23 Review of Fluoride Benefits and Risks US PHS (1991)

74 Arnala I. Bone fluoride, histomorphometry and incidence of hip fracture. Pub of the U. of Kuopio, Med Series Orig Rep, Kuopio, 1980.

75 Arnala I. et al. Effects of fluoride on bone in Finland: histomophometry of cadavar bone from low and high fluoride areas. Acta Ortho Scand; 56:161-6,1985

76 Stan C. Freni Exposure to high fluoride concentrations in drinking water is associated with decreased birth rates Journal of Toxicology and Environmental Health, 42:109-121, 1994

77References for Risk# 15

1) Carcinogenesis, Vol. 9, pp. 2279-2284 (1988)

2) Sodium Fluoride: individual animal tumor pathology table [rats], Battelle Memorial Institute, February 23, 1989

3) Sodium Fluoride: individual animal tumor pathology table [mice], Battelle Memorial Institute, April 11, 1989

4) Lancet 36, page 737 (1990)

5) Review of Fluoride: Benefits and Risks, U. S. Public Health Service, pp. F1-F7 (1991)

6) Fluoride Vol. 26, pp. 83-96 (1992)

7) A Brief Report on the Association of Drinking Water Fluoridation and the Incidence of Osteosarcoma among Young Males, New Jersey Department of Health, November 1992

8) Fluoride, the Aging Factor, Health Action Press, pp. 72-90 (1993)

78References for Risk #78

1) Jacobsen SJ, Goldberg J, Miles TP, Brody JA, et al. Regional variation in the incidence of hip fractures: U.S. white women aged 65 years and older. JAMA Vol. 264, pp. 500-502 (1990)

2) Cooper C, Wickham CAC, Barkcr DJR, and Jacobsen SJ. Water fluoridation and hip fracture [lettcr]. JAMA Vol. 266, pp. 513-514, 1991

3) Danielson C, Lyon IL, Egger M, and Goodenough GK. Hip fractures and fluoridation in Utah's elderly population. JAMA Vol. 268, pp. 746-748 (1992)

4) Journal of the American Medical Association Vol. 273, pp. 775-776 (1995)

5) Jacobsen SJ, Goldberg J, Cooper C, and Lockwood SA. The association between water fluoridation and hip fracture among white women and men aged 65 years and older: A national ecologic study. Ann Epidemiol 1992: 2:617-26

6) Sowers MFR, Clark MK, Jannausch ML and Wallce RB, A prospective study of bone mineral content and fracture in communities with differential fluoride exposure. Am J Epidemiol 1991; 133:649-60

7) Keller C. Fluorides in Drinking Water. Paper presented at the Workshop on Drinking Water Fluoride influence on Hip Fractures and Bone Health. April 10, 1991, Bethesda, Md.

8) May, DS and Wilson MG. Hip fractures in relation to water fluoridation: an ecologic analysis. Presented at the Workshop on Drinking Water Fluoride Influence on Hip Fractures and Bone Health. April 10, 1991, Bethesda, Md.

79References for Risk#79

1) Science Vol. 217, pp. 26-30 (1982)

2) Journal of the American Dental Association Vol. 108, pp. 56-59 (1984)

3) Journal of public Health Dentistry Vol. 46, pp. 184-187 (1986)

4) Health Effects of Ingested Fluoride, National Research Council, page 37 (1993)

80References for Risk#80

1) Community Health Studies Vol. 11, pp. 85-90 (1987)

2) Journal of the Canadian Dental Association Vol. 53, pp. 763-765 (1987)

3) Fluoride Vol. 23, pp. 55-67 (1990)

81References for Risk#81
1) Letter from Frank R. Fazzari, Chief, Prescription Drug Compliance, Food and Drug Administration to New Jersey Assemblyman John Kelly (June 8, 1993)

2) Preventing Tooth Decay: Results from a Four-Year National Study, Robert Wood-Johnson Foundation, Special Report number, pp. 18, (Feb. 1983)

3) Community Dentistry and Oral Epidemiology, Vol. 19, pp. 88-92 (1991)
4) 1992 Physicians' Desk Reference, page 2273.

Additional References:
82 Risk#6 Neurotoxicity and intelligence
A) Mullenix, Phyllis et. al Neurotoxicity of Sodium Fluoride in Rats, Neurotoxicology and Teratology, Vol. 17, No. 2, pp. 169-177, 1995

B) Li, XS; Zhi, JL; Gao, RO Effects of fluoride exposure on intelligence in children, Fluoride 28:4, pp. 189-192 (1995)

C) Zhao, LB; Liang, D; W Wu Lu-Liang Effects of a high fluoride water supply on children's intelligence. Fluoride vol. 29:4 pp. 190-192 1996
83
84 Yiamouyiannis, J.A. and Dean Burk, "Fluoridation and Cancer: Age Dependence of Cancer Mortality Related to Artificial Fluoridation," Fluoride, Vol. 10 #3 (102-123) 1977

85 Bayley, T. A., Harrison, J. E., Murray, T. M., Josse, R. G., Sturtridge, W., Pritzker, K. P., Strauss, A., Vieth, R., & Goodwin, S. Fluoride-induced fractures: relation to osteogenic effect. J Bone Miner Res., 5 Suppl 1, pp. S217-22, 1990.

86 NTP National Toxicology Program Fluoride Animal Study 4/26/90

87 Cohn, PD Association of Drinking Water Fluoridation and the Incidence of Osteosarcoma Among Young Males Environmental Health Services New Jersey Nov. 8, 1992

88 Yiamouyiannis, J Fluoridation and Cancer The Biology and Epidemiology of Bone and Oral Cancer Related to Fluoridation Fluoride Vol. 26 #2 1993

89 Peebles, T. C. Aasenden Effects of Fluoride Supplementation from Birth on Human Deciduous and Permanent Teeth. Arch. Oral Biol. Vol. 19, pp. 321-326, 1974

90 Yiamouyiannis op cit., 1993

91 Dr. William Marcus, Senior science advisor to the Office of Drinking Water, Environmental Protection Agency, May 1, 1990 Memo (Available from author)

92 Bayley, T. A., op cit.

93 Dambacher MA, Ittner J, and Ruegsegger P. Long-term fluoride therapy of postmenopausal osteoporosis. Bone Vol. 7 pp. 199-205, 1986

94 Riggs BL, Hodgson SF, O'Fallon WM, Chao EYS, et al. Effect of fluoride treatment on the fracture rate in postmenopausal women with osteoporosis. NEJM Vol. 322 pp. 802-9, 1990

95 Kleerekoper ME, Peterson E, Phillips E, Nelson D, et al. Continuous sodium fluoride therapy does not reduce vertebral fracture rate in postmenopausal osteoporosis [abstract] J Bone Miner Res. Res. 4 (Suppl. 1):S376, 1989

96 Jacobsen, S. J., Goldberg, J., Cooper, C., & Lockwood, S. A. (1992). 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(5), 617-26.

97 Cooper, C., Wickham, C., Lacey, R. F., & Barker, D. J. Water fluoride concentration and fracture of the proximal femur. J Epidemiol Community Health, Vol. 44(1), pp. 17-9, 1990

98 Cooper C, Wickham CAC, Barkcr DJR, and Jacobsen SJ. Water fluoridation and hip fracture [lettcr]. JAMA Vol. 266, pp. 513-514, 1991

99 Danielson, C., Lyon, J. L., Egger, M., & Goodenough, G. K. (1992). Hip fractures and fluoridation in Utah's elderly population [see comments]. Journal of the American Medical Association 1992, 268(6), 746-8.

100 Cauley JA, Murphy PA, Riley T, and Black D. Public health bonus of water fluoridation: Does fluoridation prevent osteoporosis and its related fractures [abstract] Am J Epidemiol 1991; 134:768.

101 Jacobsen SJ, O'Fallon WM, and Melton Ill LJ. Hip fracture incidence before and after fluoridation of the public water supply, Rochester, Minnesota. Am J Pub Health 1993; 83:743-5.

102 Goggin JE, Haddon W, Hambly GS, and Hoveland JR. Incidence of femoral fractures in postmenopausal women. Pub Health Rep 1965; 80:1005-12.

103 Massler, M et al Relation of endemic dental fluorosis to malnutrition JADA Vol. 44 Feb. 1952

104 U.S. Public Health Service U.S. DEPARTMENT OF HEALTH & HUMAN SERVICES FLUORIDES, HYDROGEN FLUORIDE, AND FLUORINE (F) Profile TP-91/17 April 1993 atsdr1.atsdr.cdc.gov:8080/atsdrhome.html

105 National, Research Council Effects of Ingested Fluoride National Academy Press 1993

106 Fluoride Vol. 23, pp. 55-67, 1990

107 Colquhoun, J. Influence of social class and fluoridation on child dental health Community Dent Oral Epidemiol Vol. 13, pp. 37-41, 1985

108 Journal of the Canadian Dental Association Vol. 53, pp. 763-765, 1987

109 Gray, AS Fluoridation Time for a New Base Line J Canadian Dent. Assoc. Vol. 10, 1987

110 Yiamouyiannis, J "National Institute Dental Research study shows no relationship between fluoridation and tooth decay rate" American Laboratory 5/1989

111 Colquhoun, J. Child Dental Health Differences in New Zealand, Community Health Studies Vol.. 11, #2, 1987

112 Unpublished research for Writ of Mandate Citizens Against Fluoridation vs. Attorney General Dan Lungren 7/1996

113 Yiamouyiannis, J "Fluoride the Aging Factor" Health Action Press 2nd ed. pp. 130-131, 1986

114 Mulinex et al. J Neurotoxicology 1995

115 Spittle, B. Psychopharmacology of fluoride: a review International Clinical Psychopharmacology Vol. 9, pp. 79-82, 1994