

IN THE HIGH COURT OF NEW ZEALAND
NEW PLYMOUTH REGISTRY

CIV 2013-443-107

UNDER the Judicature Amendment Act 1972 and the
Declaratory Judgments Act 1908

IN THE MATTER of an application for judicial review and an
application for a declaration

BETWEEN NEW HEALTH NEW ZEALAND INC
Plaintiff

AND SOUTH TARANAKI DISTRICT COUNCIL
Defendant

AFFIDAVIT OF KATHLEEN MOORE THIESSEN

Dated *October 29,* 2013

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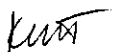
I, Kathleen Moore Thiessen, scientist of Tennessee, affirm:

Introduction

1. I am a Senior Scientist with Oak Ridge Center for Risk Analysis, Inc.
Oak Ridge Center for Risk Analysis investigates the environmental fate of radiological and chemical contaminants and evaluates human doses and health risks resulting from exposures to those contaminants.
2. My projects have involved a variety of assessments of contaminant transport, human exposures, toxicity, and health risks for both radiological and chemical contaminants.
3. I have read the statement of claim, statement of defence and the affidavits of: David Menkes, Martin Ferguson, David Sloan, Paul Connett, Stewart Jesamine, Gregory Simmons, John McMillan, Howard Wilkinson, Robyn Haisman-Welsh, Robin Wyman and Sandra Pryor.
4. I have also read, understood, and agree to comply with the Code of Conduct for expert witnesses.

Background and experience on fluoridation

5. I hold a Ph.D. degree in Biomedical Sciences (concentration, genetics) from the University of Tennessee-Oak Ridge Graduate School of Biomedical Sciences (1986) and a B.A. degree in Biology and Chemistry from Covenant College (1981).
6. While a member of the Chemical Hazard Evaluation Program of the Health and Safety Research Division of Oak Ridge National Laboratory, I authored a *Summary Review of Health Effects Associated with Hydrogen Fluoride*



and Related Compounds: Health Issue Assessment for the Environmental Protection Agency (1988), as well as health effects assessments for other chemicals. I have served on two National Research Council subcommittees, one dealing with fluoride exposure and toxicology (*Fluoride in Drinking Water: A Scientific Review of EPA's Standards*) (2006) and one dealing with guidance levels for air contaminants, including hydrogen fluoride (*Emergency and Continuous Exposure Guidance Levels for Selected Submarine Contaminants: Volume 3*) (2009).

7. I have given presentations on fluoride exposure, toxicology, and health risks to a variety of audiences, including technical (International Society for Fluoride Research, American Scientific Affiliation, International Academy of Oral Medicine and Toxicology, American Chemical Society), academic (Binghamton University, Covenant College), and lay (Metropolitan Water District of Southern California; 2nd Citizens' Conference on Fluoride; the Tennessee legislature; the town of Yellow Springs, Ohio; a citizens' group in Maryville, Tennessee). I have provided comments on fluoride-related technical reports to the U.S. Environmental Protection Agency, the U.S. Department of Health and Human Services, the California Environmental Protection Agency, the Food and Drug Administration, and the Agency for Toxic Substances and Disease Registry. I have also provided comments to a variety of state and local authorities and responded to interview requests from various news media.



8. I first became acquainted with the scientific and medical literature on fluoride exposure and toxicology in the mid-1980s, when I prepared a health issue assessment on airborne fluoride for the Environmental Protection Agency (EPA). This assessment was published in 1988 as *Summary Review of Health Effects Associated with Hydrogen Fluoride and Related Compounds: Health Issue Assessment*, Report No. EPA/600/8-89/002F (EPA 1988) and included a review of available scientific literature through January 1987. The EPA's main concern initially was hydrogen fluoride (HF). At my request, the scope of the report was expanded to include other fluoride-containing compounds. In many situations, intake of airborne fluoride is small in comparison to total intake of fluoride, but most of the toxicological effects depend on total intake of fluoride from all sources. I pointed out in this report that (1) health effects from chronic fluoride exposure are dependent on total fluoride intake from all sources; (2) people with kidney disease (renal dysfunction) are at higher risk for toxic effects due to slower clearance of fluoride from the body; (3) at least some of the decline in tooth decay attributed to fluoridated water may be due to other causes (e.g., changes in dietary patterns, changes in immune status, use of topical fluorides); and (4) the beneficial effects and adverse effects of fluoride must be weighed in determining the optimal dose for humans, and in particular, the optimal fluoride level to be maintained in public water supplies.
9. In 1998, at the request of my father, I reviewed some materials on fluoridation sent to the county school board on which he served (Lee



County, Florida) by one of the science teachers in the school system. At this time I began to be more aware of information calling into question the wisdom of water fluoridation. Some of this information was new since I had reviewed fluoride toxicity in the 1980s, and some of it was material that I had not found or had not fully appreciated in the 1980s. In particular, I learned that (1) few if any studies had examined the chemicals actually used in water fluoridation or the fluoridated water as it is consumed; (2) many human studies considered only the fluoride level in the local water supply, rather than the actual fluoride intakes experienced by individuals; (3) there was evidence for an association between water fluoridation and increased lead levels in tap water and in children's blood; (4) other countries were moving away from fluoridation of drinking water; and (5) people's fluoride intake was likely higher than had been assumed, especially for people with high water intake (e.g., athletes, outdoor workers, diabetics). I found the association between fluoridation and lead exposure especially troubling, as the connection between lead exposure and subsequent neurological and behavioral problems in children was becoming established. It also was becoming apparent to me that an association between fluoride exposure and a number of previously unacknowledged adverse health effects was plausible, but inadequately studied.

10. In 2003, I was asked to serve on a National Research Council (NRC) subcommittee charged with reviewing fluoride exposure and toxicology, and specifically with evaluating whether the EPA's drinking water



standard was sufficiently protective. As is customary for the NRC, the committee was composed of experts in several relevant disciplines and representing a variety of viewpoints. In particular, the committee included people who had expressed opinions for or against the practice of fluoridation as well as people who had expressed no opinion on fluoridation. As described in our 2006 report (*Fluoride in Drinking Water: A Scientific Review of EPA's Standards*; NRC 2006), the committee unanimously concluded that the EPA's maximum contaminant level goal (MCLG, a nonenforceable, health-based standard) which is set at 4 mg/L was not protective, and hence its maximum contaminant level (MCL, the enforceable standard, in this case equal to the MCLG) was not protective. This conclusion was based on severe dental fluorosis, stage II skeletal fluorosis, and increased risk of bone fracture, adverse effects for which sufficient information is available in the literature to consider them to be "known" adverse health effects. EPA's MCLG is supposed to be set "at a level at which no known or anticipated adverse effect on the health of persons occurs and which allows an adequate margin of safety" (EPA 2009). The NRC subcommittee also reviewed a number of other adverse health effects which can reasonably be anticipated from fluoride exposure, at the exposure levels experienced by people served with fluoridated water. The NRC subcommittee did not review the assumed benefits of fluoride exposure or of water fluoridation or make any finding that the practice of fluoridation is safe or that typical fluoride concentrations in water (concentrations corresponding to water



fluoridation) are safe. We did not evaluate the safety or benefits or efficacy of fluoridation.

11. In 2008 I was asked to serve on another NRC subcommittee, this one looking at guidance levels for air contaminants on submarines, for both acute and chronic exposures. One of the chemicals on the list was hydrogen fluoride (NRC 2009). For chronic toxicity of hydrogen fluoride, the total fluoride exposure from all sources has to be considered, as I had pointed out in 1988. The population of interest for this subcommittee was limited to healthy young men (submarine crews include no women, children, older men, or men with certain known health problems). This report provides a list of average exposure levels at which fluoride-related health effects have been reported and an estimate of the average exposure levels experienced by submarine crews on and off the submarines. The proposed 90-day Continuous Exposure Guidance Level for airborne hydrogen fluoride corresponds to a fluoride intake of 0.023-0.026 mg/kg-day, about a factor of 2 lower than a dose associated with the potential for fluoride-induced systemic toxicity (0.05 mg/kg-day), a value which is exceeded by many persons consuming fluoridated water.
12. From working on the NRC reports (2003 on), I became well acquainted with the literature on fluoride exposure and on adverse health effects from fluoride exposure. Following publication of the NRC report in 2006, I also began reviewing material on the assumed benefits of fluoridation. I have also reviewed both recent and not-so-recent

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documents from the Centers for Disease Control and Prevention (CDC), the Department of Health and Human Services (HHS), EPA, NRC, the American Dental Association (ADA), and others. From my extensive review of the scientific and medical literature, agency reports, and other publicly available information, I have identified three major areas of concern:

- 12.1 Available data do not support a role of community water fluoridation in improving dental health.
 - 12.2 A variety of adverse health effects are associated with fluoride exposures in the range experienced by people with fluoridated water.
 - 12.3 By fluoridation of drinking water, governments and water suppliers are indiscriminately administering a drug to the population, without individual evaluation of need, appropriate dose, efficacy, or side effects.
13. The following sections of this affidavit address these three areas of concern.

Available data do not support a role of community water fluoridation in improving dental health.

14. The U.S. Department of Health and Human Services (HHS) considers community water fluoridation to be important in the prevention of dental



caries (Federal Register 2011), as do governments and health agencies in a few other countries including, I understand, New Zealand. However, the question of whether water fluoridation actually produces a benefit requires further attention.

15. The University of York has carried out perhaps the most thorough review to date of human studies on effects of fluoridation. Their work (McDonagh et al. 2000) is often cited as showing the safety and efficacy of water fluoridation, but it actually does neither (Wilson and Sheldon 2006; Cheng et al. 2007). The report mentions a surprising lack of high quality studies demonstrating benefits, and also finds little evidence that water fluoridation reduces socioeconomic disparities:

- 15.1. Given the level of interest surrounding the issue of public water fluoridation, it is surprising to find that little high quality research has been undertaken. (McDonagh et al. 2000)

- 15.2. Water fluoridation aims to reduce social inequalities in dental health, but few relevant studies exist. The quality of research was even lower than that assessing overall effects of fluoridation. (Cheng et al. 2007)

- 15.3. Evidence relating to reducing inequalities in dental health was both scanty and unreliable. (Wilson and Sheldon 2006)

16. The apparent benefit is modest, about a 15% difference in the proportion of caries-free children (McDonagh et al. 2000). The American Dental Association (2005) states that “water fluoridation continues to be effective in reducing dental decay by 20-40%,” which would translate to

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less than 1 decayed, missing, or filled permanent tooth (DMFT) in older children and adolescents (based on U.S. data from CDC 2005).

17. Neither McDonagh et al. (2000) nor the ADA (2005) mentions that fluoride exposure appears to delay the eruption of permanent teeth, although this has been known since the 1940s (Short 1944; Weaver 1948; NRC 2006; Limeback and Robinson 2012). A delay in tooth eruption alters the curve of caries rates with respect to age and complicates the analysis of age-specific caries rates (Psoter et al. 2005; Alvarez 1995; Alvarez and Navia 1989). Specifically, “the longer the length of exposure to the oral environment the greater is the risk of the tooth becoming carious” (Finn and Caldwell 1963; citing Finn 1952). Komárek et al. (2005) have calculated that the delay in tooth eruption due to fluoride intake may explain the apparent reduction in caries rates observed when comparisons are made at a given age, as is usually done.
18. Most studies of benefits of fluoride intake or fluoridation have failed to account for a number of important variables, including individual fluoride intakes (as opposed to fluoride concentrations in the local water supplies), sugar intake, socioeconomic variables, local variations in caries rates, and the general decline in caries rates over the last several decades, independent of water fluoridation status. When World Health Organization data on oral health of children in various countries are compared, similar declines in caries over time are seen in all developed countries, regardless of fluoridation status (Cheng et al. 2007; Neurath 2005). The only peer-reviewed paper to be published from California's

major oral health survey in the 1990s reported no association between fluoridation status and risk of early childhood caries (Shiboski et al. 2003). Several studies show differences in caries rates with socioeconomic status or dietary factors but not with fluoridation status (e.g., Adair et al. 1999; Hamasha et al. 2006). Hagan (1947) reported a wide range of caries rates (DMFT = 1.41-10.64 at age 16) in children in various nonfluoridated communities in the state of Georgia.

19. In general, the role of diet and nutrition in good dental health seems to be underappreciated. For example, Cote et al. (2004) have documented a much lower rate of caries experience in refugee children from Africa than in U.S. children or refugee children from Eastern Europe, a situation that the authors attribute more to the amount of sugar in the diet than the presence of fluoride in the water. Finn (1952) provides an extensive review of dental caries in “modern primitive peoples,” concluding that they “show less dental caries than do most civilized peoples. . . . Evidence indicates, however, that primitive peoples have an increased caries attack rate when brought into contact with modern civilization and a civilized diet.”
20. A number of sources (reviewed by NRC 2006), including the Centers for Disease Control and Prevention (CDC 2001), indicate that any beneficial effect of fluoride on teeth is topical (e.g., from toothpaste), not from ingestion. Featherstone (2000) describes mechanisms by which topical fluoride has an anti-caries effect and states that “[f]luoride incorporated

during tooth development [i.e., from ingested fluoride] is insufficient to play a significant role in caries protection.” Also:

- 20.1. The fluoride incorporated developmentally—that is, systemically into the normal tooth mineral—is insufficient to have a measureable effect on acid solubility. (Featherstone 2000)
- 20.2. The prevalence of dental caries in a population is not inversely related to the concentration of fluoride in enamel, and a higher concentration of enamel fluoride is not necessarily more efficacious in preventing dental caries. (CDC 2001)
21. Fluoride concentrations in drinking water or saliva are too low to be contributing significantly to a topical anti-caries effect, especially since most drinking water is not “swished” around the teeth before being swallowed. CDC (2001) states that “The concentration of fluoride in ductal saliva, as it is secreted from salivary glands, is low—approximately 0.016 parts per million (ppm) in areas where drinking water is fluoridated and 0.006 ppm in nonfluoridated areas. This concentration of fluoride is not likely to affect cariogenic activity.”
22. The single study that has examined caries experience in relation to individual fluoride intakes at various ages during childhood (the Iowa study) has found no association between fluoride intake and caries experience; caries rates (% of children with or without caries) at ages 5 and 9 were similar for all levels of fluoride intake (Warren et al. 2009).

The authors state that “the benefits of fluoride are mostly topical” and that their “findings suggest that achieving a caries-free status may have relatively little to do with fluoride *intake*” (emphasis in the original). Most of the children with caries had “relatively few decayed or filled surfaces” (Warren et al. 2009). The authors' main conclusion:

Given the overlap among caries/fluorosis groups in mean fluoride intake and extreme variability in individual fluoride intakes, firmly recommending an “optimal” fluoride intake is problematic. (Warren et al. 2009).

23. The national data set collected in the U.S. in 1986-1987 (more than 16,000 children, ages 7-17, with a history of a single continuous residence) shows essentially no difference in caries rates in the permanent teeth of children with different water fluoride levels (Table 1; Fig. 1; data obtained from Heller et al. 1997; similar data can be obtained from Iida and Kumar 2009). Analysis in terms of mean DMFS (decayed, missing, or filled tooth surfaces) for the group (Fig. 2), as opposed to caries prevalence, shows an apparent 18% decrease between the low-fluoride (< 0.3 mg/L) and fluoridated (0.7-1.2 mg/L) groups. In absolute terms, this is a decrease of about one-half (0.55) of one tooth surface per child. One possible explanation is delayed tooth eruption, which was not considered in the study. Note that the mean DMFS for the highest fluoride group is higher than for either of the two intermediate groups, also indicating that DMFS scores are not solely a function of water fluoride concentration. When the data are examined by the distribution of DMFS scores (Fig. 3),

no real difference in caries experience with respect to water fluoride concentration is observed.

24. In my opinion as a scientist with particular expertise in reviewing toxicological and epidemiological literature and in assessing exposures and health risks, the available data, responsibly interpreted, indicate little or no beneficial effect of water fluoridation on oral health.

A variety of adverse health effects are associated with fluoride exposures.

25. For most of the U.S. population, the single largest source of fluoride exposure is municipal tap water, including tap water used directly, beverages and foods prepared with municipal tap water either at home or in restaurants, and commercial beverages and processed foods prepared with municipal tap water. For a water fluoride level of 1 mg/L (1 ppm), which is the level still used in most fluoridated U.S. cities, estimated average exposures to fluoride from all sources range from about 0.03 mg/kg/day (mg of fluoride per kg of body weight per day) for adults and nursing infants to 0.09 mg/kg/day for non-nursing infants (especially infants fed formula prepared with fluoridated tap water). Note that these are estimated *average* exposures. For individuals with high tap water consumption (discussed by NRC 2006), total fluoride exposures can exceed 0.1 mg/kg/day for some adults and may reach 0.2 mg/kg/day for some infants. In one of the few studies to evaluate individual intake of fluoride from all sources, Warren et al. (2009) report individual fluoride intakes (from all sources) in excess of 0.2 mg/kg/day for some infants.

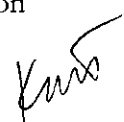


26. The NRC (2006) identified several sizeable subgroups of the U.S. population that require special consideration due to above-average fluoride exposures, increased fluoride retention, or greater susceptibility to effects from fluoride exposures. Groups known to be at risk of high fluoride intake include those with high water intake (e.g., outdoor workers, athletes, and individuals with diabetes insipidus or other medical conditions) or exposure to other sources of fluoride intake (NRC 2006). In addition, people with impaired renal function are at higher risk of adverse effects per unit intake of fluoride, due to impaired excretion of fluoride and consequent higher fluoride concentrations in the body. Tap water consumption varies among individuals by more than a factor of 10, depending on age, activity level, and the presence of certain health conditions such as diabetes insipidus (NRC 2006; see also Warren et al. 2009 for an example of estimated fluoride intakes for individual children at different ages). A substantial number of infants have water consumption rates in excess of 0.1 L/kg/day (100 mL per kg body weight per day; NRC 2006; EPA 2004a).
27. The U.S. Department of Health and Human Services (HHS) recently proposed a new recommendation regarding fluoride concentrations in drinking water (Federal Register 2011), the primary change being from a recommended range of 0.7-1.2 mg/L fluoride in drinking water (0.7-1.2 ppm) based on ambient local temperatures, to a single value of 0.7 mg/L (0.7 ppm), regardless of temperature. At the proposed fluoride concentration of 0.7 mg/L in drinking water, infants consuming at least



0.1 L/kg/day of tap water will have fluoride intakes at and above 0.07 mg/kg/day, and some will exceed 0.15 mg/kg/day (NRC 2006).

28. The HHS recommendation addresses only dental fluorosis (discussed below), while ignoring a long list of other health concerns for the U.S. population. Dental fluorosis itself has been associated with increased risks of various adverse health effects, including thyroid disease, lowered IQ, and bone fracture (Alarcón-Herrera et al. 2001; Zhao et al. 1996; Li et al. 1995; Lin et al. 1991; Desai et al. 1993; Yang et al. 1994; Jooste et al. 1999; Li et al. 2003; Susheela et al. 2005; Rocha-Amador et al. 2009). To the best of my knowledge, no studies in the U.S. or Canada have looked for associations between dental fluorosis and risk of other adverse effects. However, the failure to look for adverse health effects does not demonstrate the absence of adverse health effects.
29. The NRC (2006) indicated that the Environmental Protection Agency's (EPA's) present drinking water standards for fluoride (maximum contaminant level goal [MCLG] and maximum contaminant level [MCL], both at 4 mg/L) are not protective of human health, based on preventing severe dental fluorosis, stage II skeletal fluorosis, and increased risk of bone fractures. Given the wide range of water intake within the American population and the presence of other sources of fluoride intake, one can reasonably expect that a "safe" level of fluoride in drinking water would be at least a factor of 10 below the "unsafe" level of 4 mg/L. EPA's MCLG is defined as a "non-enforceable health goal which is set at a level at which no known or anticipated adverse effect on



the health of persons occurs and which allows an adequate margin of safety” (EPA 2009). Dental fluorosis, skeletal fluorosis, and increased risk of bone fracture are all reasonably well known and acknowledged adverse health effects from fluoride exposure. However, EPA is also required to consider the “anticipated” adverse effects (which may occur at lower levels of fluoride exposure than the “known” effects) and allow for an adequate margin of safety. The proposed HHS recommendation for water fluoridation at 0.7 mg/L is not adequate to protect against known or anticipated adverse effects and does not allow an adequate margin of safety to protect young children, people with high water consumption, people with kidney disease (resulting in reduced excretion of fluoride), and other potentially sensitive population subgroups.

30. In addition to the “known” adverse health effects of dental fluorosis, skeletal fluorosis, and increased risk of bone fracture, “anticipated” adverse health effects from fluoride exposure or community water fluoridation include (but are not limited to) carcinogenicity, genotoxicity, endocrine effects, increased blood lead levels, and hypersensitivity (reduced tolerance) to fluoride. These effects (described in more detail below) are not as well studied as the dental and skeletal effects, which should indicate that a greater margin of safety is necessary to ensure protection of the population—“in the face of uncertain evidence it is important to act in a manner that protects public health” (Tickner and Coffin 2006). In addition, it should be noted that some of these effects may occur at lower fluoride exposures than those typically associated with dental or skeletal effects, such that protection against the dental or



skeletal effects does not necessarily ensure protection against other anticipated adverse health effects. Elimination of community water fluoridation is in my opinion the best way to reduce fluoride exposures for most individuals to a level at which adverse health effects are unlikely.

31. A few comments regarding the interpretation of the available fluoride studies may be helpful. As Cheng et al. (2007) have described, a “negative” study may simply mean that the study was not sufficiently sensitive to demonstrate a moderate (as opposed to large) effect. This is often due to use of too small a sample size. In addition, study populations are often grouped by community, water source, or fluoride concentration in the water, rather than by individual intake. Due to the wide variation in drinking water intake, this approach results in study groups with overlapping intakes and makes it difficult to detect dose response relationships that do in fact exist.
32. The few studies that have looked at age-dependent exposure to fluoride have found increased risks of adverse effects (e.g., Bassin et al. 2006 for osteosarcoma; Danielson et al. 1992 for hip fracture risk); studies that have not looked at age-dependent exposure cannot be assumed to provide evidence of no effect. Similarly, studies that have used a measure of current exposure where a cumulative measure would be more appropriate, or vice versa, cannot be assumed to demonstrate lack of an effect.
33. Studies of fluoride toxicity in laboratory animals are sometimes dismissed as irrelevant because the exposures or fluoride concentrations used were



higher than those expected for humans drinking fluoridated tap water. It is important to know that animals require much higher exposures (5-20 times higher, or more; see NRC 2006; 2009) than humans to achieve the same effects or similar fluoride concentrations in bone or serum. In other words, humans are considerably more sensitive to fluoride than are most animal species that have been studied.

34. A number of adverse health effects can be expected to occur in at least some individuals when estimated average intakes of fluoride are around 0.05 mg/kg/day or higher (NRC 2006; 2009). For persons with iodine deficiency, average intakes as low as 0.01-0.03 mg/kg/day could produce effects (NRC 2006). The next few sections briefly summarize some (not all) of the adverse health effects, known and anticipated, that should be considered in any reevaluation of the drinking water standards for fluoride. Most of these effects have been reviewed in detail by the NRC (2006), although the NRC did not specifically evaluate health risks over the whole range of fluoride intakes or attempt to identify a “safe” level of fluoride exposure.

Dental fluorosis

35. The main reason for the change in fluoridation levels proposed by HHS is the prevention of dental fluorosis, a condition ranging from mild spotting of the teeth to severe pitting and staining. Dental fluorosis is caused by excessive fluoride ingestion during the early years of childhood, before the permanent teeth erupt. The HHS recommendation is




intended to limit the risk of dental fluorosis while maintaining caries protection (Federal Register 2011). The most recent data indicate a fluorosis prevalence in the U.S. (all levels of severity) of 40.7% in 1999-2004 vs. 22.6% in 1986-1987 for children ages 12-15 (Beltrán-Aguilar et al. 2010). The proposed change in water fluoridation level will put the U.S. in agreement with Canada, which in 2009 recommended a fluoride concentration of 0.7 mg/L for all parts of the country (Health Canada 2009).

36. Based on the 1986-1987 data set (as reported by Heller et al. 1997), which included water fluoride concentrations, fluoridating at 0.7 mg/L can be expected to bring the fluorosis prevalence in the U.S. down to about 27%. Elimination of fluoridation entirely, for the whole population, would be expected to bring the fluorosis prevalence down to that of the current low-fluoride population (to around 13% based on Heller et al. 1997; Fig. 4).
37. The only U.S. study to have looked at dental fluorosis and individual fluoride intake at various ages (the Iowa study) reported that for children with fluoride intakes above 0.06 mg/kg/day during the first 3 years of life, fluorosis rates were as high as 50% (Hong et al. 2006b). As mentioned above, at a fluoride concentration of 0.7 mg/L in drinking water, many infants will have fluoride intakes at and above 0.07 mg/kg/day, and some will exceed 0.15 mg/kg/day (NRC 2006). Thus a large fraction of infants and young children fed formula made with



fluoridated tap water can be expected to develop dental fluorosis even at a water fluoride concentration of 0.7 mg/L.

38. The National Research Council considers severe dental fluorosis to be an adverse health effect and reports the general consensus in the literature that both severe and moderate dental fluorosis should be prevented (NRC 2006). Health Canada (2009) considers moderate dental fluorosis to be an adverse effect. The Iowa study indicates that high fluoride intake during the first 2 years of life is most important with respect to development of dental fluorosis of the permanent maxillary central incisors (the “top front teeth”)—the teeth that most affect a person's appearance—although fluoride intake up to at least 4 years old was also important (Hong et al. 2006a). The American Dental Association has issued a brief statement to the effect that parents should not prepare infant formula with fluoridated water if they are concerned about the possibility of their child developing dental fluorosis (ADA 2007). This is an admission that dental fluorosis is undesirable, and that fluoridated tap water is not “safe” for all individuals. The CDC (2005) reports a higher likelihood of moderate and severe fluorosis for minority and low-income children. While for a variety of reasons it is appropriate for governments and health agencies to encourage breastfeeding of infants, in many family situations breastfeeding is not possible (e.g., in cases of adoption or of ill-health or death of the mother). It is therefore essential that tap water be safe for use in infant formula, without putting infants at increased risk of dental fluorosis.
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Skeletal fluorosis

39. Bone fluoride concentrations in the ranges reported for stage II and III skeletal fluorosis will be reached by long-term fluoride exposures of 0.05 mg/kg/day or higher (estimated from NRC 2006). Bone fluoride concentrations, radiologic changes, and symptoms are not clearly correlated (Franke et al. 1975), and most U.S. studies do not categorize cases by stage. Recent case reports include fluorosis attributed to excessive ingestion of tea or toothpaste (Whyte et al. 2005; Hallanger Johnson et al. 2007; Kurland et al. 2007). Most of the literature addresses high fluoride exposures over a few years; there has been essentially no investigation of effects of low exposures over many years and no effort to identify fluorosis of any stage in the U.S. “Arthritis” (defined as painful inflammation and stiffness of the joints) is the leading cause of disability in the U.S., currently affects at least 46 million adults in the U.S. (including 50% of the population > 65 years old), and is expected to affect 67 million adults in the U.S. by 2030 (CDC 2006). The possibility that a sizeable fraction of “bone and joint pain” or “arthritis” in U.S. adults is attributable to fluoride exposure has not been addressed, although it is plausible, given what is known about fluoride intakes.

Increased risk of bone fractures

40. The NRC (2006) concluded that lifetime exposure to fluoride at an estimated average daily intake of 0.08 mg/kg/day (average adult fluoride

intake with water at 4 mg/L) is likely to result in higher bone fracture rates, and the available information suggests an increased likelihood of bone fracture for daily fluoride intakes of 0.05 mg/kg/day (average adult fluoride intake at 2 mg/L). The Agency for Toxic Substances and Disease Registry (ATSDR) has identified a chronic-duration Minimal Risk Level (MRL) for oral exposure to fluoride of 0.05 mg/kg/day, based on an increased risk of bone fracture (ATSDR 2003). The NRC's findings (NRC 2006) indicate that the ATSDR's MRL is not protective enough. The available studies consider fluoride intake only in terms of the concentration in the local drinking water, and most use fluoridated water (1 mg/L, corresponding to an average daily intake of 0.03 mg/kg/day for adults) as a control. Thus there is probably considerable overlap in exposures between groups, making effects more difficult to distinguish, and the entire dose response range of interest has not been well studied. The findings in humans are consistent with animal studies that have found increased brittleness of bones with increased fluoride exposure (Clark and Mann 1938; Turner et al. 1997; 2001).

41. Danielson et al. (1992) reported an increased relative risk for hip fracture in a fluoridated area of 1.27 (95% CI 1.08-1.46) for women and 1.41 (95% CI 1.00-1.81) for men. These authors reported a difference between women exposed to fluoride prior to menopause and those exposed afterwards. For women exposed prior to menopause, the fracture risk was considerably higher than for those not exposed to fluoride. Many studies of fracture risk have not looked at age-specific

exposure, or have involved women exposed only after menopause, when fluoride uptake into bone is probably substantially lower.

42. The Iowa study reported effects on bone mineral concentration and bone mineral density with average childhood fluoride intakes of 0.02-0.05 mg/kg/day (Levy et al. 2009). Linear correlation between dental fluorosis and risk of bone fracture has been reported for children and adults (Alarcón-Herrera et al. 2001; Fig. 5). Bone fracture rates in children in the U.S. may be increasing (e.g., Khosla et al. 2003), but fluoride exposure has not been examined as a possible cause or contributor.

Carcinogenicity

43. Three U.S. courts have found water fluoridation to be injurious to human health, specifically that it may cause or contribute to the cause of cancer and genetic damage (described in detail by Graham and Morin 1999). The NRC's committee on fluoride toxicology unanimously concluded that "Fluoride appears to have the potential to initiate or promote cancers," even though the overall evidence is "mixed" (NRC 2006). Referring to the animal studies, the committee also said that "the nature of uncertainties in the existing data could also be viewed as supporting a greater precaution regarding the potential risk to humans." The committee discussed the limitations of epidemiologic studies, especially ecologic studies (those in which group, rather than individual, measures of exposure and outcome are used), in detecting small increases in risk—

in other words, the studies are not sensitive enough to identify small or moderate increases in cancer risk; therefore a “negative” study does not necessarily mean that there is no risk (see also Cheng et al. 2007).

44. While the NRC did not assign fluoride to a specific category of carcinogenicity (i.e., known, probable, or possible), the committee did not consider either “insufficient information” or “clearly not carcinogenic” to be applicable. The committee report (NRC 2006) includes a discussion of how EPA establishes drinking water standards for known, probable, or possible carcinogens; such a discussion would not have been relevant had the committee not considered fluoride to be carcinogenic. The question becomes one of how strongly carcinogenic fluoride is, and under what circumstances.
45. The case-control study by Bassin et al. (2006) is the only published study thus far to have looked at age-dependent exposure to fluoride. This study reported a significantly elevated risk of osteosarcoma in boys as a function of estimated age-specific fluoride intake. Osteosarcoma is a bone cancer that commonly results in amputation of an affected limb and may result in death. At the very least, this study indicates that similar studies of pediatric osteosarcoma that have not looked at age-dependent intake cannot be considered to show “no effect.” A recent review of osteosarcoma risk factors (Eyre et al. 2009) lists fluoride among “a number of risk factors that emerge with some consistency” and considers fluoride exposure to have a “plausible” role in etiology of osteosarcoma.



46. While a few other studies (e.g., Gelberg et al. 1995; Kim et al. 2011) have looked at individual fluoride exposure (as opposed to group or ecologic measures of exposure), these have looked at total fluoride exposure until time of diagnosis or treatment. Given that there is a “lag time” of a few years between onset of a cancer and its diagnosis, use of cumulative fluoride exposure until time of diagnosis is potentially misleading, as fluoride exposure during the last several years (during the “lag time”) cannot have contributed to the initiation of a cancer but could have a significant effect on the estimate of cumulative fluoride exposure.
47. The 1990 National Toxicology Program (NTP) study on sodium fluoride officially concluded that “there was *equivocal evidence of carcinogenic activity* of sodium fluoride in male F344/N rats, based on the occurrence of a small number of osteosarcomas in dosed animals” (NTP 1990; italics in the original). According to the published report, a “small number of osteosarcomas occurred in mid- and high-dose male rats. These neoplasms occurred with a significant dose response trend, but at a rate within the upper range of incidences previously seen in control male rats in NTP studies” (NTP 1990). It is important to realize that the historic controls from previous studies had not had the special low-fluoride diet used for this study, and therefore more properly constitute a low- to mid-range exposed group rather than a control group. This and other concerns were described in a memo within the Environmental Protection Agency (Marcus 1990) and reported in the press (Hileman 1990). These concerns and the testimony before the U.S. Senate of the union representing EPA scientists (Hirzy 2000) should be taken seriously.



48. In humans, osteosarcomas tend to occur most commonly in young people (pediatric cases) or the very old (adult or geriatric cases), with a higher incidence in males than in females (Bassin et al. 2006). Sergi and Zwerschke (2008) indicate that 60-75% of cases are in patients between 15 and 25 years old. In the NTP 2-year study, fluoride exposure was begun when the animals were 6 weeks old, as is typical for NTP and similar studies (Hattis et al. 2004). Puberty in the rat typically occurs at about 32 days of age in females and 42 days in males (e.g., Gray et al., 2004; Evans 1986). Thus, the age of 6 weeks in the NTP study probably corresponds to pubertal or post-pubertal animals. The cases of osteosarcoma in the rats were reported in the late stages of the test, and probably corresponded to geriatric osteosarcomas in humans. In Bassin's study, the age range for which the fluoride-osteosarcoma association was most apparent was for exposures at ages 4-12 years, with a peak for exposures at age 6-8 years (Bassin et al. 2006). Very likely, the fluoride exposures in most of the animal studies have started after the age corresponding to the apparent most susceptible age in humans, and thus these animal studies may have completely missed the most important exposure period with respect to initiation of the majority of human osteosarcomas. Therefore, this animal study cannot be interpreted as showing no evidence of causation for pediatric osteosarcoma, although, properly interpreted, it does show evidence for causation of geriatric osteosarcoma.



Genotoxicity

49. Genotoxicity, or the ability to damage the genetic material (genes and chromosomes) of cells, is considered indicative of potential carcinogenicity. A number of mammalian *in vitro* systems have shown dose-dependent cytogenetic or cell transformational effects from fluoride exposure (reviewed by NRC 2009). Several reports suggest an indirect or promotional mechanism, e.g., inhibition of DNA synthesis or repair enzymes, rather than a direct mutagenic effect (Lasne et al. 1988; Aardema et al. 1989; Aardema and Tsutsui 1995; Meng and Zhang 1997). Human cells seem to be much more susceptible to chromosome damage from fluoride than are rodent cells (Kishi and Ishida 1993).
50. A recent paper by Zhang et al. (2009) describes a new testing system for potential carcinogens, based on induction of a DNA-damage response gene in a human cell line. Sodium fluoride tests positive in this system, as do a number of other known carcinogens, representing a variety of genotoxic and nongenotoxic carcinogenic mechanisms. Known noncarcinogens—chemicals not associated with carcinogenicity—did not test positive. The system described by Zhang et al. (2009) is considerably more sensitive than the older systems for most chemicals examined; a positive effect was seen at a fluoride concentration of about 0.5 mg/L, or a factor of 10 lower than in other systems.
51. A fluoride concentration of 0.5 mg/L in urine will routinely be exceeded by many people consuming fluoridated water (NRC 2006); for people with substantial fluoride intake, serum fluoride concentrations may also

reach or exceed 0.5 mg/L. Acute fluoride exposures (e.g., accidental poisoning, fluoride overfeeds in drinking water systems) have resulted in fluoride concentrations in urine well in excess of 5 mg/L in a number of cases (e.g., Gessner et al. 1994; Penman et al. 1997; Björnhagen et al. 2003; Vohra et al. 2008). Urine fluoride concentrations can also exceed 5 mg/L if chronic fluoride intake is above about 5-6 mg/day (0.07-0.09 mg/kg/day for an adult; based on NRC 2006). Thus, kidney and bladder cells are probably exposed to fluoride concentrations in the ranges at which genotoxic effects have been reported *in vitro*, especially when the more sensitive system of Zhang et al. (2009) is considered. Based on the results of Zhang et al. (2009), most tissues of the body are potentially at risk if serum fluoride concentrations reach or exceed 0.5 mg/L. In addition, cells in the vicinity of resorption sites in fluoride-containing bone are potentially exposed to very high fluoride concentrations in extracellular fluid (NRC 2006) and thus are also at risk for genotoxic effects.

Endocrine effects

52. The NRC (2006) concluded that fluoride is an endocrine disruptor. Endocrine effects include altered thyroid function or increased goiter prevalence (at fluoride intakes of 0.05-0.1 mg/kg/day, or 0.01-0.03 mg/kg/day with iodine deficiency), impaired glucose tolerance (at fluoride intakes above 0.07 mg/kg/day), a decrease in age at menarche in girls in fluoridated towns, and disruptions in calcium metabolism

(calcitonin and parathyroid function, at fluoride intakes of 0.06-0.15 mg/kg/day or higher). ATSDR's toxicological profile for fluoride (ATSDR 2003) refers to an animal study of thyroid function that would give a lower MRL (value not given) than the MRL derived for bone fracture risk (0.05 mg/kg/day).

53. Thyroid dysfunction and Type II diabetes presently pose substantial health concerns in the U.S. (NRC 2006). Of particular concern is an inverse correlation between subclinical maternal hypothyroidism and the IQ of the offspring. In addition, maternal subclinical hypothyroidism has been proposed as a cause of or contributor to development of autism in the child (Román 2007; Sullivan 2009). Steingraber (2007) has described the decrease in age at puberty of U.S. girls and the associated increased risk of breast cancer. Calcium deficiency induced or exacerbated by fluoride exposure may contribute to other health effects (NRC 2006).

Increased blood lead levels

54. An increased likelihood of elevated blood lead levels is associated with use of silicofluorides (usually H_2SiF_6 or Na_2SiF_6) as the fluoridating agent (NRC 2006; Coplan et al. 2007). Approximately 90% of people on fluoridated water are on systems using silicofluorides (NRC 2006). The chemistry and toxicology of these agents, especially at low pH (e.g., use of fluoridated water in beverages such as tea, soft drinks, or reconstituted fruit juices), have not been adequately studied (NRC 2006). Associations between silicofluoride use and biological effects in humans have been reported, in particular, elevated levels of blood lead in children and

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inhibition of acetylcholinesterase activity (reviewed by Coplan et al. 2007). A recent study in rats found significantly higher concentrations of lead in both blood and calcified tissues of animals exposed to both silicofluorides and lead (Sawan et al. 2010).

55. In addition to biological effects of silicofluorides, the interaction of silicofluorides (as the fluoridating agent) and disinfection agents (specifically, chloramines) also increases the leaching of lead from plumbing fixtures into drinking water (Maas et al. 2005; 2007). For example, the interaction of silicofluorides and chloramines is the probable explanation for the high lead levels in drinking water and children's blood in Washington, D.C. a few years ago (Maas et al. 2005; 2007; Leonnig 2010). EPA considers lead to be a probable human carcinogen and to have no practical threshold with respect to neurotoxicity (EPA 2004b)—in other words, there is considered to be no safe level of lead exposure, and the MCLG for lead is zero (EPA 2009).

Neurotoxicity

56. Grandjean and Landrigan (2006) list fluoride as an “emerging neurotoxic substance” that needs further in-depth studies. The major concern is neurotoxic effects during human development. The NRC (2006) concluded that “it is apparent that fluorides have the ability to interfere with the functions of the brain and the body by direct and indirect means.” A number of studies indicate an association of fluoride exposure with lower IQ in children and with other measures of neuropsychological

development (reviewed by NRC 2006; Connett et al. 2010; Choi et al. 2012; see also Zhao et al. 1996; Lu et al. 2000; Xiang et al. 2003; Rocha-Amador et al. 2007; 2009; Saxena et al. 2012; Seraj et al. 2012).

Additional adverse health effects

57. Fluoride intake is likely to affect the male reproductive-hormone environment, beginning at intakes of around 0.05 mg/kg/day (reviewed by NRC 2009). A “safe” intake with respect to male reproductive effects is probably somewhere below 0.03 mg/kg/day.
58. The NRC has reviewed the possible association between exposure to fluoridated water (approximately 0.02 mg/kg/day for adults) and increased risk of Down syndrome (trisomy 21) in children of young mothers, discussed a possible mechanism, and recommended further study (NRC 2006). Fetuses with Down syndrome are less likely to survive to birth, due both to higher natural fetal loss and to a high rate of pregnancy termination (Buckley and Buckley 2008; Forrester and Merz 1999; Siffel et al. 2004; Biggio et al. 2004).
59. Hypersensitivity or reduced tolerance to fluoride has been reported for exposure to fluoridated water (approximately 0.02 mg/kg/day for adults) or use of fluoride tablets (approximately 1 mg/day). Symptoms include skin irritation, gastrointestinal pain and symptoms (nausea, vomiting, diarrhea, constipation), urticaria, pruritus, stomatitis, chronic fatigue, joint pains, polydipsia, headaches, and other complaints (Waldbott 1956; 1958; Feltman and Kosel 1961; Grimbergen 1974; Petraborg 1977; Spittle 2008;

reviewed by NRC 2006). Patients were often unaware that their drinking water contained fluoride. Symptoms improved with avoidance of fluoridated water and recurred with consumption of fluoridated water or with experimental challenge with sodium fluoride. Double-blind tests of patients have confirmed hypersensitivity to fluoride (Grimbergen 1974; Waldbott 1956; 1958). Many of the observed symptoms represent true allergic phenomena, while others (e.g., gastrointestinal symptoms) could be due to a lower level of tolerance for fluoride (intoxication at lower exposure; Waldbott 1956; 1958).

By fluoridation of drinking water, governments and water suppliers are indiscriminately administering a drug to the population, without individual evaluation of need, appropriate dose, efficacy, or side effects.

60. The U.S. Food and Drug Administration (FDA) considers fluoride in toothpaste to be a non-prescription drug (e.g., FDA undated-a; undated-b) and fluoride “supplements” (usually tablets or lozenges) to be prescription drugs (e.g., Medline Plus 2008). Most prescription fluoride supplements are considered unapproved drugs (for example, see DailyMed 2011a,b,c), meaning that they “may not meet modern standards of safety, effectiveness, quality, and labeling” (FDA 2011). The goal of community water fluoridation is to provide a dental health benefit to individuals and to the population generally (Federal Register 2010), and EPA's recent reference (Federal Register 2010) to a “treated population”

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acknowledges this use of drinking water systems to deliver a drug to entire populations. This in effect puts local governments and water treatment personnel in charge of administering a chemical (i.e., a drug) to the population in an effort to improve individual and population health (Cross and Carton 2003; Cheng et al. 2007). Many people consume more fluoride from tap water than from either non-prescription (toothpaste) or prescription (tablets or lozenges) fluoride sources, without any monitoring for either efficacy or side effects, without the “drug information” or warning labels generally provided for drugs, and without any semblance of informed consent.

61. In addition, most fluoridation operations use fluorosilicates (usually H_2SiF_6 or Na_2SiF_6) rather than sodium fluoride (NaF). The chemistry and toxicology of these compounds have not been adequately studied, although important differences in biological effects between silicofluorides and simple fluorides (e.g., NaF) have been reported (Coplan et al. 2007; NRC 2006; Masters et al. 2000; Masters and Coplan 1999). The NRC (2006) discussed the increased toxicity of aluminofluorides and beryllifluorides vs. fluoride alone, as well as the different mechanisms of action of the different chemical combinations. In my opinion it is irresponsible to recommend addition of fluoride, or a particular concentration of fluoride to be added, without a comprehensive review of the substances (H_2SiF_6 or Na_2SiF_6) that are actually added. In addition, fluoridation chemicals often contain impurities such as lead and arsenic (e.g., Weng et al. 2000; Brown et al. 2004), for which EPA has set MCLGs of zero (EPA 2009), such that a

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water supplier is actually adding contaminants for which the ideal maximum amount in drinking water is zero.

62. In summary, in my opinion it is irresponsible to promote or encourage uncontrolled exposure of any population to a drug that, at best, is not appropriate for many individuals (e.g., those who do not want it, those whose water consumption is high, formula-fed infants, people with impaired renal function) and for which the risks are inadequately characterized and inadequately disclosed to the public. Elimination of community water fluoridation at the earliest possible date would be in the best interest of public health.

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Table 1. Caries prevalence and fluorosis prevalence with water fluoride concentration.^a

Water fluoride concentration mg/L	Children with no caries %	Mean DMFS score ^b	Children with fluorosis ^c %	Mean severity of fluorosis ^d
< 0.3	53.2	3.08	13.5	0.30
0.3 - < 0.7	57.1	2.71	21.7	0.43
0.7 - 1.2	55.2	2.53	29.9	0.58
> 1.2	52.5	2.80	41.4	0.80

^a Data for permanent teeth of children ages 5-17 (caries experience and DMFS score) or 7-17 (dental fluorosis), with a history of a single residence, from Tables 2 and 5 of Heller et al. (1997).

^b Decayed, missing, or filled tooth surfaces (permanent teeth).

^c Includes very mild, mild, moderate, and severe fluorosis, but not "questionable."

^d Dean's Community Fluorosis Index.

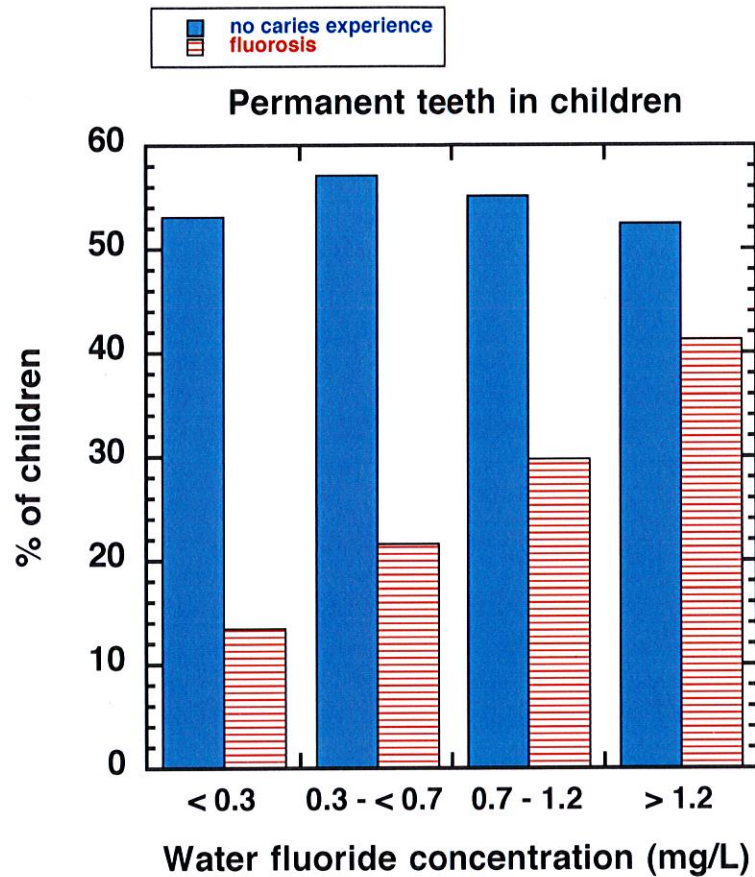


Fig. 1. Percent of children with no caries experience in the permanent teeth (DMFS = 0) and with fluorosis, with respect to water fluoride concentration. Data are shown as % of total children having no caries experience (blue) or having fluorosis (very mild, mild, moderate, or severe, but not questionable; red). Numerical values are provided in Table 1 and were obtained from Tables 2 and 5 of Heller et al. (1997).

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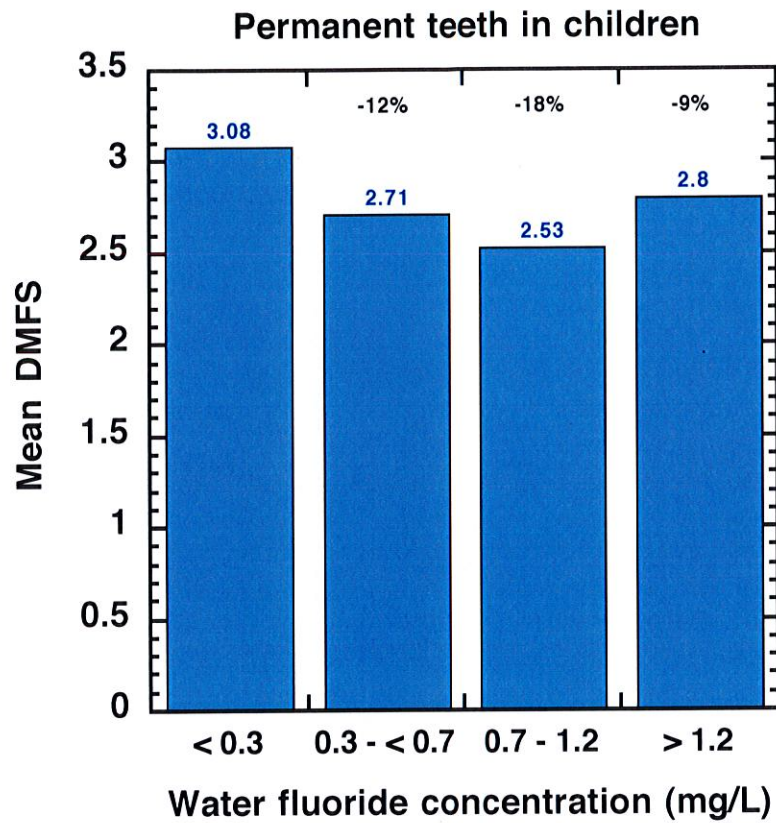


Fig. 2. Mean DMFS score (decayed, missing, or filled permanent tooth surfaces in permanent teeth), with respect to water fluoride concentration. Numerical values are provided in Table 1 and were obtained from Table 2 of Heller et al. (1997). The percent difference with respect to the lowest fluoride group is also provided.

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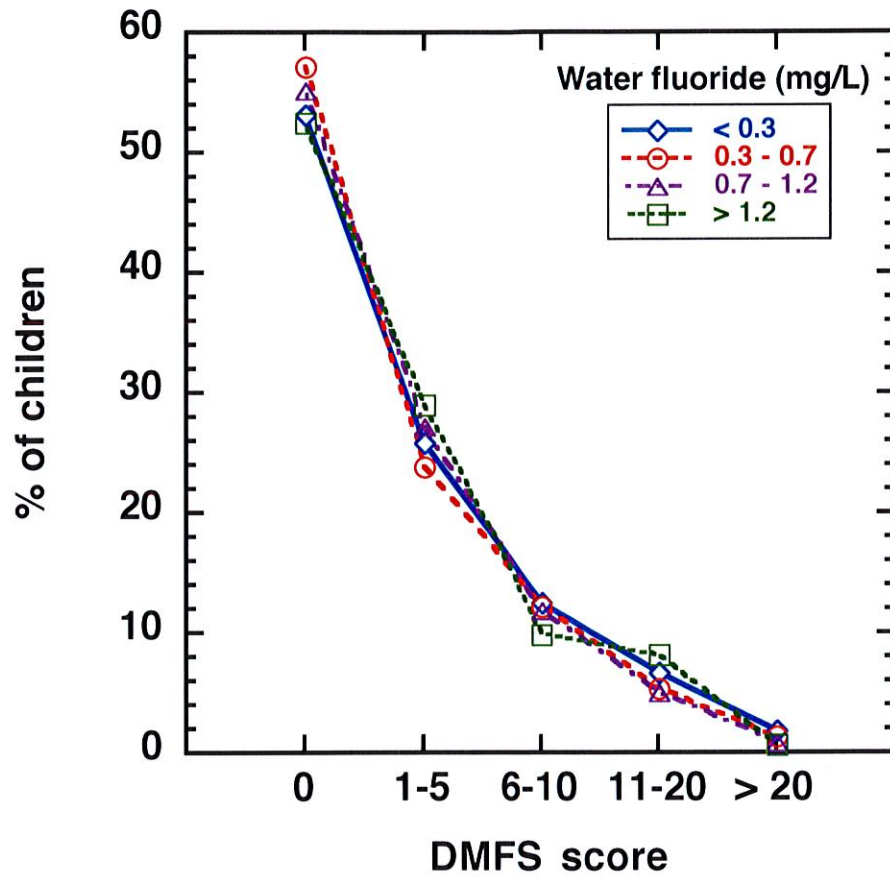


Fig. 3. Percent of children by DMFS score, with respect to water fluoride concentration. Data are shown as % of total children in a given group according to the number of decayed, missing, or filled tooth surfaces in the permanent teeth (DMFS). Data were obtained from Table 2 of Heller et al. (1997).

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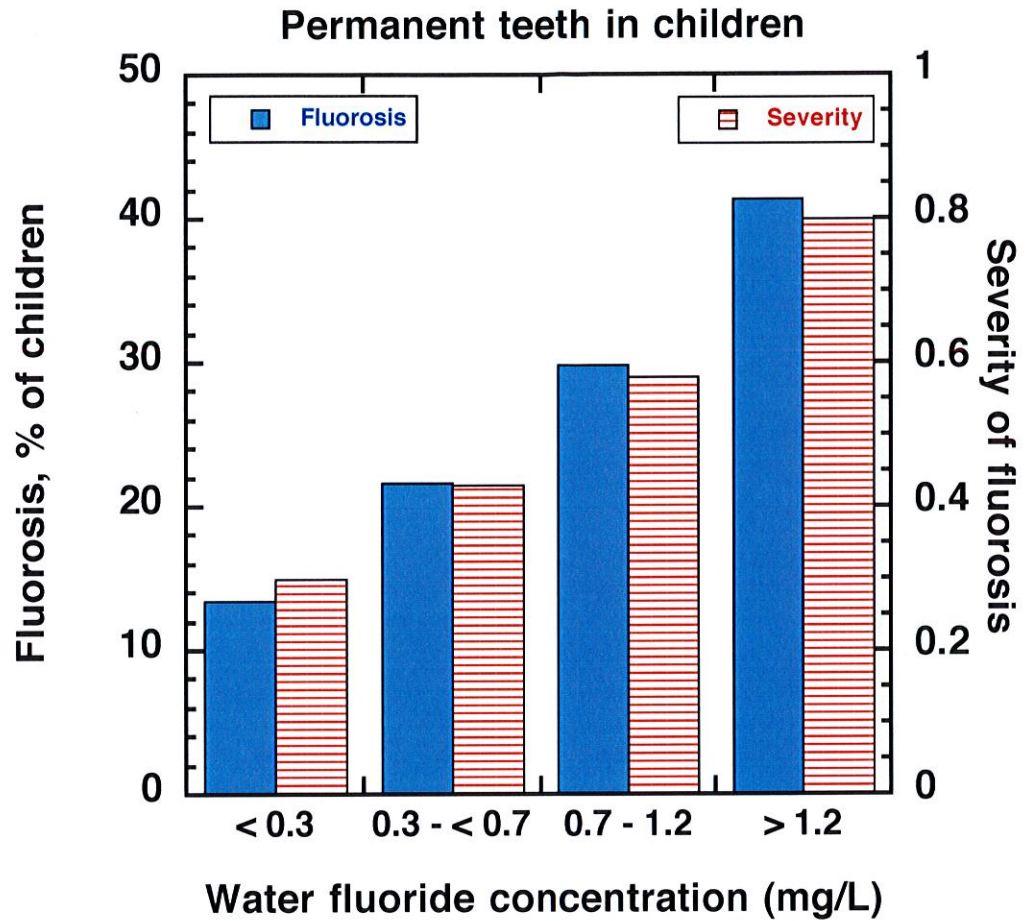


Fig. 4. Fluorosis prevalence and severity with water fluoride concentration for children ages 7-17 with a history of a single continuous residence. Data are shown as (left) % of total children having fluorosis (very mild, mild, moderate, or severe, but not questionable) or (right) severity of fluorosis by Dean's Community Fluorosis Index. Numerical values are provided in Table 1 and were obtained from Table 5 of Heller et al. (1997).

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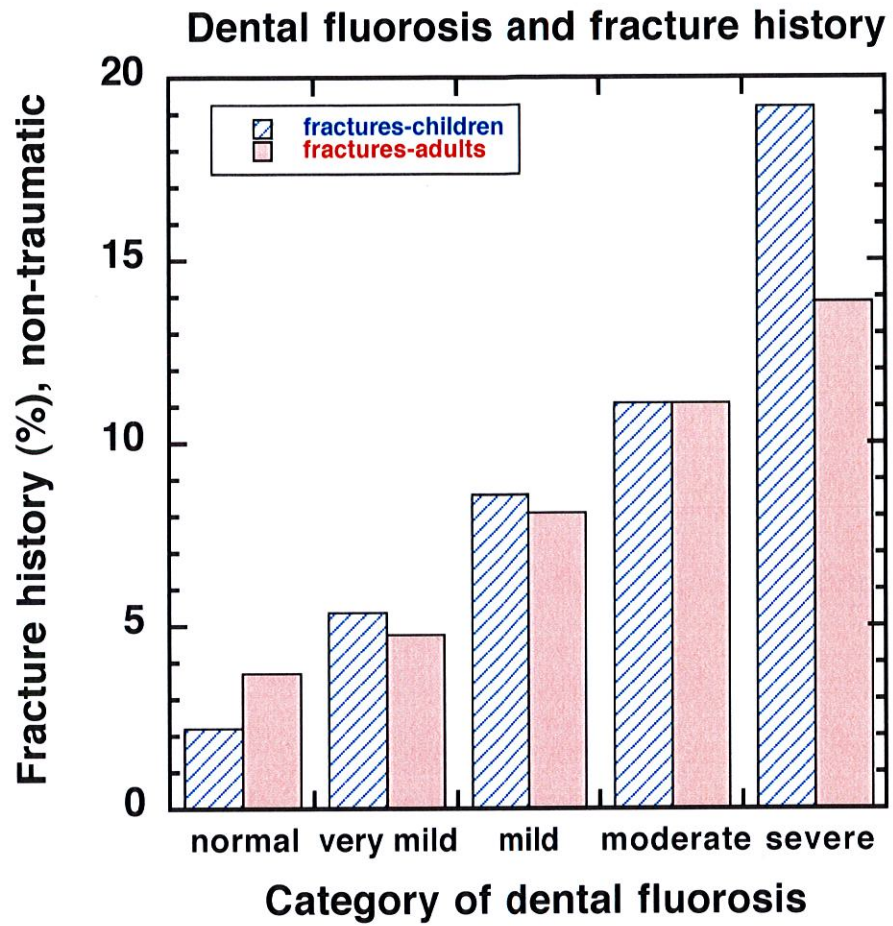


Fig. 5. Fracture history with category of dental fluorosis for children (ages 6-12) and adults (ages 13-60). Numerical values were obtained from information in Tables 5 and 6 of Alarcón-Herrera et al. (2001).

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Kathleen M. Thiesen

AFFIRMED at *Oak Ridge, TN* this *29th*
day of *Oct* 2013

before me:

Kimberly H. Disney



A person who is authorised to administer oaths by the law of the state of Tennessee

Commission Expires 4/26/14