The case against fluoridation of public water supplies is getting stronger because evidence of fluoride's harmful health impacts keeps accumulating. In July 2014 residents of Peel, Canada, filed a legal petition to end fluoridation, supported by testimony from Kathleen Thiessen, Ph.D. Dr. Thiessen, who served on the National Research Council committee that studied fluoride in water, testified that a variety of health effects are associated with fluoride exposure, including effects on the brain, bones, renal system, and endocrine system. Dr. Thiessen also testified that the benefits of fluoridated water have been poorly-documented and likely are overstated, and that fluoridated water is a drug that is administered without individual dosing or consent.

Attached is a legal opinion written for the Concerned Residents of Peel to End Fluoridation by Nader Hasan of the Canadian law firm Ruby, Shiller Chan and Hasan. Dr. Thiessen's affidavit is appended to the memo.

—Moms Against Fluoridation

MEMORANDUM

RUBY SHILLER CHAN HASAN BARRISTERS

TO: Liesa Cianchino, *Chair of Concerned Residents of Peel to End Fluoridation*

FROM: Nader R. Hasan

DATE: June 23, 2014

RE: Legal Arguments Against Artificial Water Fluoridation

SUMMARY AND OPINION

You have asked me to provide an opinion on the lawfulness of the Region of Peel's fluoridation program. In short, if an Ontario resident can properly present the existing scientific and medical evidence to an Ontario court, then there is a reasonable possibility that an Ontario court would declare the *Fluoridation Act* and municipal fluoridation programs in Ontario to be unconstitutional and thus invalid. Should that occur, there is also a real possibility that the Region of Peel would be held legally liable to residents in a lawsuit for harm caused by artificial fluoridation.

This memorandum proceeds in three parts. Part I discusses the factual background to Ontario and Peel's fluoridation programs and situates these provisions in the global context. Part II discusses the scientific evidence relating to health effects of fluoridation. While fluoridation has significant potential effects on the environment and non-human animal and plant species, I focus on the human health effects because those effects are likely to figure most prominently in a legal challenge to fluoridation. Part III discusses the potential arguments in a legal challenge to fluoridation programs in Ontario as well as other legal issues that may arise in a court challenge to fluoridation in Ontario. I have also appended to this memo an affidavit from Dr. Kathleen Thiessen, a biomedical scientist, who has served on two U.S. National Research Council subcommittees dealing with fluoride exposure and toxicology. Her affidavit was commissioned specifically in connection with the ongoing debate about fluoridation in the Region of Peel.

PART I – FACTUAL BACKGROUND OF ARTIFICIAL FLUORIDATION

Fluoride is the anionic or reduced form of fluorine and is the thirteenth most abundant element in the Earth's crust. Given that fluorine is so abundant, it is not surprising that fluoride compounds are components of minerals in rocks and soil. Due to these components, and the action of ground water acting upon them, fluoride is released into the groundwater and is the major contributor to the small amounts of fluoride present in most water sources. In general, most ground water contains low concentrations of fluoride, typically less than 0.5 mg/L.

Fluoridation is the controlled addition of fluoride ions to water that has a low fluoride concentration (sometimes called "artificial fluoridation"). In the early 1900s, significant work was done in understanding the root cause of the mottling of teeth and tooth decay. This mottling, and improved dental health, was ultimately attributed to the high fluoride concentrations in the ground water that was ingested by these individuals. Over time, additional studies were undertaken, which were purported to establish a relationship between fluoride and substantially fewer cavities, ultimately leading to four community-wide trials that were established in the mid-1940s. These trials were conducted in Grand Rapids, MI; Newburgh, NY; Brantford, ON and Evanston, Ill. Soon thereafter, the U.S. Public Health Service and many dental associations endorsed community-wide fluoridation as a practical and safe public health measure to prevent tooth decay.

Over the past 65 years, additional investigation has examined everything from the health effects of the various fluoride compounds used in the fluoridation process to the dosage levels that provide adequate dental health protection. Over this time-frame, fluoride dosage levels have on average dropped from 1.0 to 1.2 mg/L to between 0.5 and 0.8 mg/L, while the maximum acceptable concentration (MAC) has been established at 1.5 ppm. The Ontario Ministry of Health and Long Term Care, in partnership with the Ontario Ministry of the Environment, have established a guideline of 0.5-0.8 mg/L for fluoride in drinking water. The Region of Peel claims to "closely monitor" the fluoride levels in the

water supply to make sure the correct concentration is being maintained.[1] Under the *Safe Drinking Water Act Regulations*, the maximum allowable concentration of fluoride in Ontario drinking water is 1.5 mg/L.[2]

In 1961, the Province of Ontario enacted the *Fluoridation Act,*[3] which specifically provided for the establishment and maintenance of fluoridation of drinking water within the Ontario waterworks system. The *Fluoridation Act* does not require fluoridation. Under the *Act*, municipalities were given the discretionary authority, by way of the passing of a by-law "…to establish, maintain and operate, or require that the local board establish, maintain and operate, a fluoridation system in connection with the waterworks system."[4]

Cities that already had a fluoridation program in place were not required to pass a new by-law; the *Fluoridation Act* permitted the continuation of those programs.[5] Accordingly, the *Fluoridation Act* permitted the continuing fluoridation of the water supplies of the City of Mississauga and City of Brampton. In 2007, the Regional Municipality of Peel passed a by-law establishing a fluoridation system in the Town of Caledon.[6]

According to the Canadian Dental Association, approximately 45% of Canadians drink fluoridated public water.[7] However, the figures vary significantly across the country. Quebec has historically opposed artificial fluoridation, and as such, today less than 3%

---

[1] Region of Peel, Peel Public Health, "Fluoridation - Frequently Asked Questions", online: http://www.peelregion.ca/health/topics/commdisease/dental/fluoridation.htm#10.

[2] *Safe Drinking Water Act, 2002*,*Ontario Drinking Water Quality Standards*, O.R. 169/03, Schedule 2.

[3] *Fluoridation Act*, R.S.O. 1990, c. F.22.

[4] *Ibid*., s. 2(1).

[5] *Ibid*., s. 2.1(2).

[6] Regional Municipality of Peel, *A by-law to provide for the fluoridation of the Town of Caledon's communal water supply*, online: http://www.peelregion.ca/health/topics/commdisease/dental/by-law.htm.

[7] Danielle Rabby-Waytowich, "Water Fluoridation in Canada: Past and Present" (July/August 2009), 75 JCDA 451, online: http://cda-adc.ca/jcda/vol-75/issue-6/451.pdf.

Quebec's population drinks fluoridated water.[8] Only approximately 3.7% of residents of British Columbia drinks fluoridated water.[9] At 75.9%, Ontario is the most heavily fluoridated province. In recent years, however, some medium-sized municipalities, including Waterloo and Windsor, have ended their fluoridation programs.[10] The debate between pro- and anti-fluoride activists in Ontario municipalities is acrimonious, with both sides accusing the other of "cherry picking" research to boost its argument. Health Canada as well as the Canadian Medical Association and the Canadian Dental Association are staunchly pro-fluoride. The Green Party of Canada, and respected NGOs such as the Council of Canadians, Green Peace Canada and Sierra Club, oppose fluoridation of municipal water supplies.

Canada's rate of fluoridation puts it squarely in the global middle among the Organization of Economic and Cooperative Development ("OECD") countries. According to a 2002 study, approximately 69% of U.S. residents were living in communities with fluoridated water.[11] By contrast, only approximately 3% of the population in Western Europe currently consumes fluoridated water.[12] Despite this fact, the available evidence does not suggest that tooth decay rates are higher in unfluoridated Western European countries than in the United States or other fluoridated countries.

PART II – SCIENTIFIC EVIDENCE CONCERNING FLUORIDATION

The success of any legal challenge to Ontario's fluoridation program will turn on the quality of expert and scientific evidence presented. For the claimants to be successful, they will have to adduce evidence of both (1) fluoride's speculative and/or nominal

[8] Eric Tchouaket et al, "The economic value of Quebec's water fluoridation program" (June 2013), 21 J Public Health 523 at 524.

[9] *Ibid*. Danielle Rabby-Waytowich, "Water Fluoridation in Canada: Past and Present", *supra* at 452.

[10] See CBC News, Fluoride no longer to be added to Windsor water" (Jan. 29, 2013), CBC.ca online: http://www.cbc.ca/news/canada/windsor/fluoride-no-longer-to-be-added-to-windsor-water-1.1325977.

[11] Centers for Disease Control and Prevention, "Fluoridation Status: Percentage of U.S. Population on Public Water Supply Systems Receiving Fluoridated Water", CDC.gov online: http://apps.nccd.cdc.gov/nohss/FluoridationV.asp.

[12] Fluoride Action Network, "Water Fluoridation Status in Western Europe", online: http://fluoridealert.org/content/water_europe/.

benefit in reducing dental caries; and (2) the risk of harm posed by fluoride in adults and children. To date, the most comprehensive review of the existing scientific evidence on fluoride's toxicity is the study conducted by the National Research Council's Committee on Fluoride in Drinking Water, which was published in 2006.[13] The National Research Council ("NRC") is a non-profit entity in the United States, whose membership includes eminent scientists across the United States. It is funded in part by Congress and the U.S. federal agencies. Its studies are generally considered authoritative.

The review of the evidence below is not meant to be exhaustive. It is meant rather to highlight the types of evidence that could be presented in a legal challenge.

Lack of Evidence of Fluoridation's Benefits

The purpose of fluoridation is to reduce dental caries (tooth decay). Since the 1950s, it has been virtually gospel within the dental community that fluoridation of drinking water is responsible for reducing tooth decay. This belief was once thought to be unassailable. But the evidence available today makes it far from clear. We now know that tooth decay is enhanced or diminished by numerous factors, including dietary, socio-economic, environmental, hygienic and many other factors. Recent studies have shown that tooth decay rates have decreased as fast in unfluoridated areas as in fluoridated areas,[14] leading many to suggest that other factors — i.e., improved diet, modern dental care, more regular trips to the dentist and the availability of fluoridated toothpaste — are the causes of decreases in tooth decay rather than water fluoridation.

In 1999, the U.S. Centers for Disease Control and Prevention conceded what many dental researchers already had concluded: that fluoride's predominant mechanism of action was

[13] Committee on Fluoride in Drinking Water, National Research Council, *Fluoride in Drinking Water: A Scientific Review of EPA's Standards* (National Academies of Sciences Press, 2006) at 4 [hereinafter "NRC Report"].

[14] See, e.g., John Colquhuon, *Child Dental Health Differences in New Zealand*, 9 Comm. Health Stud. 85 (1987); John Yiamouyiannis, *Water Fluoridation and Tooth Decay: Results from the 1987-1987 National Survey of Schoolchildren*, 23 Fluoride 55 (1990).

topical, not *systemic*.[15] In other words, to the extent that fluoride works, it does so via direct exposure to the tooth and not from inside the body. Connett, Beck and Micklem argue persuasively that if the primary benefit of fluoride is through topical treatment on teeth, then it makes no sense to expose every tissue in the body to fluoride through ingestion in drinking water.[16]

Scientific Evidence of Fluoride's Harm

There is significant scientific evidence of harm caused by fluoridation. And even if the harms associated with fluoridation cannot be proven to a degree of scientific certainty, the existing scientific information and literature point to a variety of serious risks inherent in artificial fluoridation.

Dental Fluorosis

There is a scientific consensus that fluoridation can cause "dental fluorosis", which is a dose-related mottling of the enamel of the teeth that can range from mild discoloration of the tooth surface to severe staining and pitting. The condition is permanent after it develops in children during tooth formation. Whether to consider fluorosis to be an adverse health effect or merely a cosmetic effect has been the subject of debate. However, the U.S. National Research Council has concluded that severe fluorosis is more than a cosmetic issue because severe fluorosis can lead to enamel loss, leaving the dentin open to decay and infection and causing structural damage to the tooth.[17]

Muskoskeletal Effects

Skeletal fluorosis is a bone and joint condition associated with prolonged exposure to high concentrations of fluoride. Fluoride increases bone density and appears to exacerbate the growth of osteophytes present in the bone and joints, resulting in joint

[15] Centers for Disease Control and Prevention, "Achievements in Public Health, 1900-1999: Fluoridation of Drinking Water to Prevent Dental Caries" (Oct. 1999), 48 Mortality and Morbidity Weekly Review 933-40, online: http://www.cdc.gov/mmwr/preview/mmwrhtml/mm4841a1.htm.

[16] Paul Connett et al, *The Case Against Fluoride: How Hazardous Waste Ended Up in Our Drinking Water and the Bad Science and Powerful Politics that Keep It There*, at 13.

[17] NRC Report, *supra*, at 4

stiffness and pain.[18] There is no doubt that high concentrations of fluoride cause skeletal fluorosis. The debate within the scientific community is the extent of the risk of skeletal fluorosis at current levels of fluoridation.[19] Defenders of fluoridation argue that a concentration of 1.5mg/L is too low to present a risk of skeletal fluorosis. It should, however, be noted that the first symptoms of skeletal fluorosis are similar to the first symptoms of many forms of arthritis — stiffness and pain in the joints and pain in the bones.

There is also scientific evidence that fluoride can increase the risk of bone fractures. The NRC Report notes that "several strong observational studies indicated an increased risk of bone fracture in populations exposed to fluoride at 4 mg/L."[20] While there are fewer studies dealing with the risk of bone fracture within populations exposed to fluoride at a rate of 2 mg/L or lower, there is a peer-reviewed study from Finland that suggests an increased rate of hip fracture in populations exposed to fluoride at concentrations above 1.5 mg/L,[21] which is the maximum allowable rate of fluoridation in Ontario.

Neurobehavioural Effects

Animal and human studies of fluoride have been published reporting adverse cognitive and behavioural effects. Epidemiological studies conducted in China have reported I.Q. deficits in children exposed to fluoride at 2.5 to 4 mg/L in drinking water. The NRC found these studies to be sufficiently alarming to call for "additional research on the effects of fluoride on intelligence."[22] In 2012, a group of scientists published a systematic review of the literature on developmental fluoride neurotoxicity. The review concluded that the consistency of pre-existing studies showing a link between fluoride

[18] NRC Report, *supra*, at 5.

[19] *Ibid*. at 6.

[20] *Ibid*.

[21] *Ibid*. at 7.

[22] *Ibid*. at 8.

and cognitive deficits shows that potential developmental neurotoxicity of fluoride should be a high research priority.[23]

The NRC also noted that fluorides "increase the production of free radicals in the brain through several different biological pathways. These changes have a bearing on the possibility that fluorides act to increase the risk of developing Alzheimer's disease."[24] The NRC has called for additional studies in this area as well.[25]

Genotoxicity and Carcinogenicity

There have been a number of studies that have suggested a link between fluoride and bone cancer. The NRC Report concludes that fluoride "appears to have the potential to initiate and promote cancers, particularly of the bone, but the evidence to date is tentative and mixed".[26] The NRC cautions readers that at the time of the publication of the NRC Report a major hospital-based study on osteosarcoma (bone cancer) and fluoride exposure was underway at the Harvard School of Dental Medicine.[27] The Harvard study, which was published in 2006, found an association between fluoride exposure in drinking water during childhood and the incidence of osteosarcoma among males (but not females).[28] This is a significant and concerning finding.

PART III – LEGAL ISSUES AND ARGUMENT

Detractors of fluoridation raise a number of policy and moral arguments. These include, *inter alia*, arguments that fluoridation may be harmful to the environment and plant and animal wildlife. They also point out that fluoridated water in much of North America is treated by using hexafluorosicilic acid (H2SiF6) and sodium silicofluoride (Na2SiF6), which are by-products of fertilizer manufacturing and which contain numerous

[23] Anna Choi et al, "Developmental Fluoride Neurotoxicity: A Systematic Review and Meta-Analysis" (2012), 120 Environmental Health Perspectives 1362 at 1367.

[24] *Ibid*. at 222.

[25] *Ibid.*

[26] *Ibid*. at 336.

[27] Ibid. at 10.

[28] Elise B. Bassin et al, "Age-specific fluoride exposure in drinking water and osteosarcoma" (2006), 17 Cancer Causes & Control 421.

contaminants, including heavy metals such as lead and chromium, nonmetals such as arsenic, and even trace amounts of radioactive isotopes.

While these and other arguments may be persuasive policy arguments against fluoridation, a legal challenge to fluoridation based on human health effects is the most likely argument to succeed in Canadian courts. More specifically, if the proper evidence, such as the medical evidence described above, can be presented in court, there is a reasonable possibility that an Ontario court will declare the *Fluoridation Act* and the municipal fluoridation programs in Ontario to be unconstitutional.

The Constitutional Argument

The most viable legal argument against Ontario's fluoridation program is that it is unconstitutional because it violates s. 7 of the *Canadian Charter and Rights and Freedoms*. Section 7 provides that "Everyone has the right to life, liberty and security of the person and the right not to be deprived thereof except in accordance with the principles of fundamental justice."[29] Legislation that conflicts with this constitutional right must be struck down.

Section 7 of the *Charter* means that everyone has the right to life, liberty and security of the person. This right, however, is not limitless. The State can limit one's rights to life, liberty and security of the person, but only if it does so in accordance with "the principles of fundamental justice." Thus, to establish a violation of s. 7 of the *Charter*, the claimant must establish: (1) that the law or State action has deprived the claimant of her or his right to life, liberty or security of the person; *and* (2) that the deprivation is inconsistent with principles of fundamental justice. There are strong arguments that a claimant challenging Ontario's *Fluoridation Act* could satisfy both of these legal requirements.

[29] Part I of the *Constitution Act, 1982*, being Schedule B to the *Canada Act 1982* (U.K.), 1982, c. 11, s. 7 [hereinafter "Charter"]. For an overview of s. 7 and its jurisprudence, see Hamish Stewart, *Fundamental Justice: Section 7 of the Canadian Charter of Rights and Freedoms* (Toronto: Irwin Law, 2012); Nader R. Hasan, "Three Theories of 'Principles of Fundamental Justice'" (2013), 63 S.C.L.R. (2d) 339.

Fluoridation Deprives Residents of the Right to Liberty and Security of the Person

The Supreme Court of Canada has found that the liberty interest protected by s. 7 includes the right to make fundamental personal choices free from state interference.[30] In the context of medical treatment, the Ontario Court of Appeal has held that the right not to be subject to medical treatment without informed consent is an aspect of the security of the person interest under s. 7.[31] Section 7 thus protects "the right to be free from unwanted medical treatment."[32] To deprive individuals of the ability to make decisions with respect to their treatment and to force them to submit to medication against their competent wishes infringes the *Charter* right to security of the person as protected under s. 7 of the *Charter*.[33]

Ontario's fluoridation programs infringe upon the s. 7 right to security of the person. Fluoridation is State-imposed mass medication. This proposition was established by the Supreme Court's 1957 decision in *Toronto (Metro) v. Forest Hill (Village)*.[34] In that case, the residents challenged a municipal by-law that authorized the City "to undertake the treatment of the …. water supply by fluoridation." At that time, the Province's enabling legislation only permitted the municipalities to ensure a "continued and abundant supply of pure and wholesome water." It did not specifically authorize fluoridation or other forms of mass medication. The City argued that the power to make the water supply "pure and wholesome" implicitly authorized fluoridation. A majority of the Supreme Court of Canada disagreed. It held that fluoridation "is not a means to an end of wholesome water for water's function but to an end of a special health purpose for which a water supply is made use of as a means." In other words, the purpose of fluoridation was not to purify the water, but to medicate the population with fluoride.

The Ontario Legislature superseded *Toronto (Metro) v. Forest Hill (Village)* when it passed the *Fluoridation Act* in 1961. But the Supreme Court of Canada's conclusion that

[30] *Blencoe v. British Columbia (Human Rights Commission)*, [2000] 2 S.C.R. 307 at, para. 54.

[31] *Fleming v. Reid*, [1991] O.J. No. 1083 at para. 31, 39-40 (C.A.).

[32] *Ibid*. at para. 31.

[33] *Ibid*. at para. 40.

[34] *Toronto (Metro) v. Forest Hill (Village)*, [1957] S.C.R. 569.

the purpose of fluoridation is not water purification but rather medication remains the finding of this country's highest court. As such, Ontario's fluoridation programs constitute medication without consent and thus deprives Ontario residents of their s. 7 liberty and security-of-the-person interests.

Fluoridation Violates the Principle of Gross Disproportionality

Given that the *Fluoridation Act* triggers the s. 7 liberty and security-of-the-person rights, the primary challenge for claimants will be in showing that the deprivation is inconsistent with the principles of fundamental justice. If that can be shown, then the claimant will have succeeded in proving that the fluoridation program is unconstitutional. The most relevant principle of fundamental justice here is the principle against *gross disproportionality*.

A law is "grossly disproportionate" if the state action or legislative response to a problem is so extreme as to be disproportionate to any legitimate government interest.[35] In other words, a law will be found to be grossly disproportionate where its benefits are grossly disproportionate to its potential harm.[36]

If a claimant can properly marshal the available scientific evidence, they ought to be able to show that the *risk* of significant harm caused by fluoridation is grossly disproportionate to the speculative benefit of reduced dental carries. As noted above, recent studies suggest that the claimed reduction in tooth decay over the past several decades is more likely attributable to improved dental care rather than fluoridated water. If true, then the benefits of fluoridated water are, at best, marginal, or, at worst, non-existent.

By contrast, the negative effects of fluoridation appear to be real and substantial. As noted above, the authoritative NRC Report concludes that dental fluorosis is more than

[35] R. v. Malmo-Levine, [2003] 3 S.C.R. 571 at para. 143.

[36] *Canada (Attorney General) v. PHS Community Services Society*, [2011] S.C.J. No. 44, [2011] 3 S.C.R. 134 at para. 153; *Canada (Attorney General) v. Bedford*, 2013 SCC 72 at para. 159.

just a cosmetic effect.[37] Peer-reviewed scientific studies show that water fluoridation can have an adverse impact on children's I.Q..[38] Other studies show that fluoride can affect bone and make fractures more likely.[39] The 2006 Harvard study shows an association between osteosarcoma and fluoridated water.[40] Even if these negative effects are not conclusively proven, the *risk* of potential harm is significant. It would be reckless to expose residents to the risk of cancer, among other things, for the marginal benefit of reduced tooth decay, particularly where, as here, it is no longer clear that fluoridated drinking water is even a significant contributor to reduced tooth decay. Marginal benefit in exchange for significant risk is the *sine qua non* of gross disproportionality.

The likelihood of success of a hypothetical legal challenge to fluoridation will turn largely on the strength of the scientific evidence presented in court because the stronger the scientific evidence of risk of harm, the greater the gross disproportionality.

Previous Legal Challenges Are Not Indicative of Likelihood of Success in Ontario

Skeptics about the viability of a successful legal challenge to Ontario's fluoridation program will point out that since the Supreme Court's 1957 decision *Toronto (Metro) v. Forest Hill (Village)*, which was superseded by legislative action (see *supra* at 10-11), all other legal challenges to fluoridation programs in North America have failed. For the following reasons, I do not regard these cases as barring a legal challenge in Ontario.

The Canadian Cases

In Canada, there have been unsuccessful challenges to fluoridation programs in Alberta and British Columbia: see, e.g., *Millership v. Kamloops (City)*;[41] *Locke v. Calgary (City)*.[42] Those cases, however, are distinguishable on at least three different grounds.

[37] *Supra* at 6.

[38] *Supra* at 7.

[39] *Supra* at 6-7.

[40] *Supra* at 7-8.

[41] [2003] B.C.J. No. 109 (B.C. Sup. Crt).

[42] [1993] A.J. No. 926 (Q.B.)).

First, those challenges were brought by self-represented litigants. While it appears that these individuals did an admirable job at marshaling the evidence and the arguments, novel constitutional challenges such as this are highly complex and require the assistance of counsel.

Second, the scientific evidence about fluoridation is improving. More information than ever before is known about fluoridation. At the time that *Millership* (2003) and *Locke* (1996) were decided, for example, the NRC Report had not yet been published. Nor had the Harvard study on the association between osteosarcoma and artificial fluoridation been completed.

Third, Canadian constitutional law under s. 7 of the *Charter* has developed significantly over the past five years. The principle of fundamental justice of "gross disproportionality" is a fairly new principle in Canadian constitutional law. Prior to the Supreme Court's recent decisions in *PHS* and *Bedford*, there was some doubt over whether this principle was indeed a principle of fundamental justice and also some doubt over what "gross disproportionality" actually meant. In my view, the best argument against fluoridation relies on the principle of gross disproportionality. This argument was not available to the claimants in *Locke* and *Millership.* Each of these factors suggests that these other cases will not bar a successful constitutional challenge to fluoridation in Ontario.

The U.S. Cases

The U.S. cases are also distinguishable, but for different reasons. There have been a handful of high-profile cases in the United States that involved challenges to municipal fluoridation programs. These challenges have failed on technical grounds, but each time the trial judge made judicial findings of fact that supported the plaintiffs' arguments that fluoridation causes harm to humans. In *Aitkendead v. Borough of West View*, the trial judge granted a preliminary hearing enjoining the municipality from continuing its fluoridation program on the basis that the plaintiffs had shown compelling evidence that fluoride may be a carcinogen.[43] That decision was superseded by legislative action,[44] but

[43] *Aitkendead v. Borough of West View*, No. GD-458578 (Allegheny County Court of Common Pleas, Pa); see also John Remington Graham and Pierre-Jean Morin, "Highlights in North American Litigation During

the factual findings spurred investigations into fluoridation in the United Kingdom and in Quebec, with the latter ultimately imposing a moratorium on fluoridation across the Province.[45]

The next important U.S. case involving a challenge to fluoridation was *Illinois Pure Water Committee v. Director of Public Health*.[46] After a lengthy trial, Judge Niemann concluded that fluoridation legislation, which "exposes the public to the risk, uncertain in its scope, of unhealthy side effects of artificial fluoridation in water supplies, is unreasonable, and [is] a violation of the due process clause of the Illinois Constitution of 1970."[47] He further noted that "[t]his record is barren of any credible and reputable scientific epidemiological studies and/or analysis of statistical data which would support the Illinois Legislature's determination that fluoridation of public water supplies is both a safe and effective means of promoting public health."[48] Accordingly, Judge Niemann entered a permanent injunction enjoining further fluoridation in Illinois. The Illinois Supreme Court granted the State's appeal, but it did not disturb any of Judge Niemann's factual findings.[49] Instead, the Illiniois Supreme Court relied on an expansive doctrine of "police powers", under which the State was granted significant deference on decisions relating to public health. The Illiniois Supreme Court wrote that the "wisdom, necessity and expediency" of the fluoridation program "are no concern of the courts, but are matters primarily for the legislative body of the municipality, and courts are without power to interfere merely because they believe a different regulation might have been wiser or better."[50] Under this heightened evidentiary burden, it was not enough that the

---

the Twentieth Century on Artificial Fluoridation of Public Water Supplies," 14:2 J. Land Use & Envtl. L. 195 at 229-232.

[44] *Aitkendead v. Borough of West View*, 397 A.2d 878 (Pa. Commw. Ct. 1979)

[45] See Graham and Morin, "Highlights in North American Litigation During the Twentieth Century on Artificial Fluoridation of Public Water Supplies," *supra* at 232.

[46] *Illinois Pure Water Committee v. Director of Public Health*, No. 68-E-128 (Madison County Circuit Court Ill. 1982).

[47] *Ibid*. at 32.

[48] *Ibid*. at 33.

[49] *Illinois Pure Water Committee v. Director of Public Health*, 470 N.E.2d 988 (Ill. Sup. Ct. 1984).

[50] *Ibid*. at 991-992.

plaintiffs have shown that fluoridation causes "some risk of a higher incidence of cancer."[51]

The court reached a similar result in *Safe Water Foundation of Texas v. City of Houston*, a challenge to the City of Houston's fluoridation program. After a lengthy trial, with ample expert testimony on both sides, the trial judge concluded that artificial fluoridation of public water supplies "may cause or contribute to cancer, genetic damage, intolerant reactions and chronic toxicity, including dental mottling…," and "that the value of said artificial fluoridation is in some doubt as to the reduction of tooth decay in man."[52] Still, the court denied the plaintiffs' motion for an injunction on grounds of police powers. The Texas Court of Appeals denied the appeal on similar grounds, but also acknowledged the significant evidence in the record that fluoridation caused harm. It noted that if the standard had been the normal civil standard of evidence (e.g., a balance of probabilities), the plaintiffs would have won. Indeed, the Texas Court of Appeals expressly found that a fair preponderance of evidence showed that "the injection of fluoride into the City's water system would be harmful," but saved the legislation on police power grounds.[53]

The U.S. cases would likely have reached a different result had Canadian law been applied or if those cases had been litigated in Canadian courts. The U.S. cases applied a very deferential standard to the pro-fluoridation defendants and held the plaintiffs to a nearly impossible burden of proof. A claimant bringing a constitutional challenge under s. 7 of the Charter would not face the same obstacles. In other words, the police powers doctrine would not save the Ontario *Fluoridation Act* if fluoridation was found to cause harm.

The Use Hexafluorosicilic Acid (H2SiF6)

I have been advised that the Region of Peel uses hexafluorosicilic acid to fluoridate its drinking water. Hexafluorosicilic acid is a waste product that is created in the fertilizer

[51] *Ibid*. at 992.

[52] *Safe Water Foundation of Texas v. City of Houston*, No. 80-52271, Findings of Fact, May 24, 1982, at 1-2.

[53] *Safe Water Foundation of Texas v. City of Houston*, 661 S.W.2d 190 at 192 (Tex. App. 1983).

manufacturing process.[54] When hexafluorosicilic acid is in its gaseous form (hydrogen fluoride (HF) and silicon tetrafluoride (SiF4)), it is a highly toxic substance.

Proponents of using hexafluorosicilic acid as a fluoridating agent argue that by the time it is diluated by about 180,000 to 1 (to reach acceptable fluoride concentrations), the contaminant levels will be below regulatory concern.[55] But this argument overlooks the fact that amounts of other contaminants, such as arsenic, remain in the hexafluorosicilic acid solution. The U.S. Environmental Protection Agency sets the ideal safety goal for arsenic in drinking water at zero because arsenic is a known human carcinogen.[56] While there may be trace amounts of arsenic naturally occurring in water, it is difficult to justify the *addition* of a known carcinogen.[57] Critics of hexafluorosicilic acid also point out that there are no known toxicological studies regarding the safety of using hexafluorosicilic acid to fluoridate water.

Apart from the constitutional argument described above, the use of hexafluorosicilic acid may violate the *Safe Drinking Water Act*. Section 20 of the *Safe Drinking Water Act* provides that "[n]o person shall cause or permit any thing to enter a drinking water system if it could result in … a drinking water health hazard…." or "is a contravention of a prescribed standard."[58]

The use of hexafluorosicilic acid may also violate the federal *Food and Drugs Act*. Section 4 of the *Food and Drugs Act* prohibits the sale of articles of food or drink that "has in or on it any poisonous or harmful substance." [59] To the extent that hexafluorosicilic acid contains a known carcinogen, then its addition to the water

---

[54] Paul Connett et al, *The Case Against Fluoride*, *supra* at 16.

[55] *Ibid*. at 19.

[56] United States Environmental Protection Agency, "Arsenic in Drinking Water", online: http://water.epa.gov/lawsregs/rulesregs/sdwa/arsenic/index.cfm.

[57] *Ibid*.

[58] *Safe Drinking Water Act, 2002*, S.O. 2002, ch. 32, s. 20(1)(a).

[59] *Food and Drugs Act*, R.S.C., 1985, c. F-27, s. 4.

represents the addition of a "poisonous or harmful substance", which is, in turn, sold to the residents of Peel.

Liability of the Region of Peel

A finding that the Region of Peel's fluoridation program is unconstitutional and/or that the use of hexafluorosicilic acid is illegal could have significant pecuniary implications for the Region. If a court should find that the fluoridation program was unconstitutional because of an unacceptable risk of harm, this could pave the way for lawsuits against the municipality.

The *Municipal Act, 2001* imposes a statutory duty of care on those who oversee drinking water systems and makes municipalities liable in tort for acts or omissions.[60] Moreover, as of December 31, 2012, amendments to the *Safe Drinking Water Act* clarified the standard of care for municipalities. Under this standard, municipalities must exercise the level of care, diligence and skill in respect of a municipal drinking water system that a reasonably prudent person would be expected to exercise in a similar situation.[61] The standard of care also extends to the owner of the municipal drinking water system, and to those people who, on behalf of the municipality, oversee the accredited operating authority or who exercise decision-making authority over the system.

The *Safe Drinking Water Act* puts responsibility for ensuring safe drinking water squarely on the municipalities. It also arguably makes those who make decisions about the municipal water supplies — such as Councillors — personally liable for acts or omissions.[62] It follows that if a court should find that fluoridation puts residents of Peel at risk of harm, then the Region of Peel and its Councillors may be liable to its residents for damages on the civil negligence standard.

[60] *Municipal Act, 2001,* S.O. 2001, ch. 25, ss. 448(2), 448(3).

[61] *Ibid.,* s. 19(1).

[62] *Ibid*., s. 19(2).

It is also worth noting that the Region faces potential liability not only under a potential civil suit brought by residents but may also be prosecuted by the Province. Under the *Safe Drinking Water Act Regulations*, any person resident in Ontario can ask the Ontario government to investigate the Region for an alleged violation of the *Act*.[63] Furthermore, the *Safe Drinking Water Act* provides that a violation of s. 20 — the prohibition on putting material into water that could cause a health hazard — shall be a criminal offence. Thus, if fluoride is proven to cause harm or a risk of harm, then a municipality that continues to fluoridate could theoretically face *criminal* prosecution.

Thus, a municipality that fails to discharge its duty of care under the *Safe Drinking Water Act* could face (1) civil liability to residents in a civil lawsuit; (2) prosecution by the Ontario government; and (3) potentially, criminal liability. These risks and liabilities ought to be sufficient to encourage municipalities to carefully re-examine their water fluoridation programs.

CONCLUSION AND RECOMMENDATIONS

In sum, if a resident of Peel succeeds in marshaling the available scientific evidence in court, there is a reasonable possibility that the *Fluoridation Act* and the Peel fluoridation programs could be found to be unconstitutional under s. 7 of the *Charter*. And if it is demonstrated in court that fluoridation puts the residents of Peel at risk, the Region is potentially liable in tort to every resident of the Region who drinks fluoridated municipal water.

It is recommended that the Regional Council take the following steps:

1. That the Council pass a resolution to re-examine its fluoridation program;

2. That the Council hear expert testimony from experts in the fields of medicine, epidemiology and dentistry to better understand the risks and benefits associated with water fluoridation;

[63] *Safe Drinking Water Act, 2002, Compliance and Enforcement Regulation*, O. Reg. 242/05, s. 7(1).

3. That the Council hear expert testimony both from experts who support fluoridation and those who oppose fluoridation; and

4. That the Council require that experts presenting their opinions also provide the Council with the underlying data and studies on which they are relying for their opinions. There is enough competing opinion in the scientific community that it will be important for municipalities to understand the bases for scientific opinion as they re-examine this important issue.

I look forward to discussing the foregoing with you further.

Nader R. Hasan

Affidavit of Kathleen M. Thiessen, Ph.D.

Kathleen M. Thiessen, Ph.D.

Senior Scientist

Oak Ridge Center for Risk Analysis, Inc.

102 Donner Drive

Oak Ridge, TN 37830

Tel.: (865) 483-6111

Fax: (865) 481-0060

E-mail: kmt@orrisk.com

April 29, 2014

I, KATHLEEN THIESSEN, of the City of Oak Ridge, in the State of Tennessee, **HEREBY MAKE OATH AND SAY AS FOLLOWS:**

I have been asked to prepare an affidavit concerning the health effects associated with water fluoridation in connection with the Region of Peel's reconsideration of its water fluoridation policies. I make this affidavit for no improper purpose.

Background and experience on the fluoridation issue

I hold a Ph.D. degree in Biomedical Sciences (concentration, genetics) from the University of Tennessee-Oak Ridge Graduate School of Biomedical Sciences and a B.A. degree in Biology and Chemistry from Covenant College. 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, 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*) and one dealing with guidance levels for air contaminants, including hydrogen fluoride (*Emergency and Continuous Exposure Guidance Levels for Selected Submarine Contaminants: Volume 3*). I am currently a Senior Scientist with Oak Ridge Center for Risk Analysis, Inc., where my projects have involved a variety of assessments of contaminant transport, human exposures, toxicity, and health risks for both radiological and chemical contaminants.

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), academic (Binghamton University, Covenant College), and lay (Metropolitan Water District of Southern California; 2nd Citizens' Conference on Fluoride; the Tennessee legislature; the towns of Yellow Springs, Ohio, and Maryville, Tennessee). I have provided comments on fluoride-related technical reports to Health Canada, the Committee on Health and Social Services of Québec, 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.

Attached hereto as Exhibit "A" to this Affidavit is a true and correct copy of my curriculum vitae.

Introduction

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.

In 1998, I reviewed some materials on fluoridation sent to the county school board on which my father 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 tap 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.

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 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) 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 from fluoride exposure. EPA's MCLG is supposed to be set "at a level at which no known or anticipated adverse effect on the health of persons is expected to occur and which allows an adequate margin of safety" (EPA 2012). 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, nor did it specifically evaluate the safety of water fluoridation.

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.

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 documents from the Centers for Disease Control and Prevention, the Department of Health and Human Services, the U.S. Environmental Protection Agency, the National Research Council, Health Canada, the American Dental Association (ADA), the Canadian Dental Association (CDA), 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:

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

(2) A variety of adverse health effects are associated with fluoride exposures in the range experienced by people with fluoridated water.

(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..

The following three sections of this affidavit address these three areas of concern. The fourth section of this affidavit summarizes the typical fluoride intakes that can be expected in fluoridated communities in Ontario and compares them with estimated levels of intake associated with specified adverse health effects.

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

Health Canada "supports water fluoridation as a public health measure to prevent dental decay" (Health Canada 2011a), and the Chief Medical Officer of Health for Ontario has "urge[d] all Ontarians to continue to support the fluoridation of their municipal drinking water systems so that everyone can enjoy the lasting health benefits" (OMHLTC 2011). 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), and the CDC has listed it among the "ten great public health achievements of the 20th century" (CDC 1999; cited by Health Canada 2011a; OMHLTC 2011). Governments and health agencies in several other countries also

consider water fluoridation to be important and beneficial. However, the question of whether water fluoridation actually produces a benefit requires further attention.

The University of York's thorough review of human studies on effects of water fluoridation (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:

> 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)

> 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)

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

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 less than 1 decayed, missing, or filled permanent tooth (DMFT) in older children and adolescents (based on U.S. data from CDC 2005). Health Canada (2010a) cites the York review (McDonagh et al. 2000) and a major U.S. study by Heller et al. (1997), among others, as support for the effectiveness of water fluoridation. Heller et al. (1997), described in more detail below, is used as the basis for Health Canada's determination of an "optimal" concentration of fluoride in drinking water of 0.7 mg/L (Health Canada 2010a).

Neither McDonagh et al. (2000), the ADA (2005), nor Health Canada (2010a) mentions that fluoride exposure appears to delay the eruption of permanent teeth, although this has been known since the 1940s (Short 1944; Feltman 1956; 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.

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, and the general decline in caries rates over the last several decades, independent of water fluoridation status (e.g., Diesendorf 1986; Colquhoun 1993). 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., Barnes et al. 1992; Adair et al. 1999; Hamasha et al. 2006).

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."

A number of sources (reviewed by NRC 2006), including the 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:

> The fluoride incorporated developmentally—that is, systemically into the normal tooth mineral—is insufficient to have a measureable effect on acid solubility. (Featherstone 2000)

> 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)

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."

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). This paper, which is not mentioned by Health Canada (2010a), reports 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).

Health Canada (2010a) bases its "optimal" concentration of fluoride in drinking water (0.7 mg/L) on a 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), as reported by Heller et al. (1997). However, these data actually show 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.

Overall, the available data, responsibly interpreted, indicate little or no beneficial effect of water fluoridation on oral health.

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

For most Canadians in fluoridated areas (45% of Canadians, 76% of Ontario residents; Health Canada 2011a), 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 0.7 mg/L (0.7 ppm), considered the "optimal" level by Health Canada (2010a,b; 2011b), estimated average exposures to fluoride from all sources range from about 0.02 mg/kg/day (mg of fluoride per kg of body weight per day) for adults and nursing infants to 0.065 mg/kg/day for non-nursing infants (especially infants fed formula prepared with fluoridated tap water; based on NRC 2006). Note that these are estimated *average* exposures. For individuals with high tap water consumption (discussed by NRC 2006), total fluoride exposures at 0.7 mg/L can exceed 0.1 mg/kg/day for some adults and may approach 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.

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; these groups can reasonably be expected to exist in Canada as well. 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 U.S. 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), and a similar situation can be expected in Canada.

Canada recently reduced its "optimal" concentration of fluoride in drinking water from a range of 0.8-1.0 mg/L to a single value of 0.7 mg/L (Health Canada 2011b). In 2011, The U.S. Department of Health and Human Services (HHS) proposed a similar new recommendation (Federal Register 2011; still not official) of a single value of 0.7 mg/L (0.7 ppm), consistent with the Canadian recommendation. Both the Canadian and U.S. recommendations address 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; Susheela et al. 2005), although this is not addressed by either the Canadian or U.S. recommendations. To the best of my knowledge, no published 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.

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 is expected to occur and which allows an adequate margin of safety" (EPA 2012). 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.

Thus, based on the NRC's review of the EPA standards and EPA's own requirements, neither the Canadian "optimal" fluoride concentration nor the proposed U.S. recommendation for water fluoridation, both at 0.7 mg/L, can be considered adequate to protect against known or anticipated adverse effects, and neither allows 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. The Canadian Maximum Acceptable Concentration (MAC) for fluoride in drinking water, 1.5 mg/L (Health Canada 2010a), is less than a factor of 3 below the value (4 mg/L) that the NRC (2006) concluded is not safe.

According to the Canadian Dental Association (CDA 2009), an "additive" to drinking water "should not add more than 10% of the EPA-established MCL (Maximum Contaminant Level) of any regulated drinking water substance in order to ensure the protection of the public." Fluoride is a regulated drinking water substance, and ten percent of the EPA-established MCL for fluoride (4 mg/L) is 0.4 mg/L. Canada's equivalent to the MCL in the U.S. is its MAC, which for fluoride is 1.5 mg/L (Health Canada 2010a); ten percent of the MAC is 0.15 mg/L. Nevertheless, Health Canada recommends an "optimal" concentration of 0.7 mg/L, thus contradicting the guidelines used for most other regulated substances in drinking water.

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, neurotoxicity, 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.

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.

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.

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.

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 have been documented for fluoride exposure. 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

The main reason for the recent changes in fluoridation levels (instituted in Canada and proposed in the U.S.) 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 Canadian and proposed U.S. recommendations are intended to limit the risk of moderate (Canada) or severe (U.S.) dental fluorosis while maintaining caries protection (Health Canada 2010a; 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). Canada reported a fluorosis prevalence of 16.4% (very mild and mild, with "very low levels of moderate and severe") among children ages 6-12 surveyed in 2007-2009 (Health Canada 2010a;c). Neither the more recent U.S. data nor the Canadian data report dental fluorosis prevalence with respect to local water fluoride concentrations. If the Canadian survey was representative with respect to local water fluoride concentrations, given a fluoridation rate of nearly one-half the population, one could reasonably expect that the fraction of children with fluorosis in fluoridated areas exceeds 20%.

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.

Health Canada (2010a) considers moderate dental fluorosis to be an adverse effect. 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). Neither the Canadian nor U.S. authorities have addressed the costs to

treat the cosmetic appearance of fluorosed teeth, apart from whether dental fluorosis is considered "adverse" in terms of health.

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.

Skeletal fluorosis

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). Chachra et al. (2010) recently reported bone fluoride content for residents of Toronto (fluoridated for 32-36 years at the time of the study) and Montreal (not fluoridated) who were undergoing total hip replacement surgery; most of the individuals had a diagnosis of osteoarthritis. Two of the 53 individuals in Toronto had bone fluoride concentrations in the range reported for skeletal fluorosis (NRC 2006), although both individuals would have been well into adulthood when exposure to fluoridated water began. The study did not include

exposure histories; nevertheless, it does indicate that bone fluoride concentrations in fluoridated Canadian cities can be in the range reported for skeletal fluorosis.

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. or Canada. "Arthritis" (defined as painful inflammation and stiffness of the joints) is a leading cause of disability in Canada and currently affects approximately 16.6% of Canadian adults (4.5 million people); more than half of Canadians with arthritis are less than 65 years old (Arthritis Society 2013). The possibility that a sizeable fraction of "bone and joint pain" or "arthritis" in Canadian (or 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

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).

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.

The Iowa study has 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

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).

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.

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.

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. Kim et al. (2011) actually point out that "if risk is related to exposures at a specific time in life, rather than total accumulated dose, this metric [bone fluoride levels at the time of treatment] would not be optimal." In addition, given that the median age of the controls used by Kim et al. (2011) was

more than twice the median age of the cases, and that the "median cumulative lifetime water fluoride" calculated for each group was similar, the findings of Kim et al. (2011) actually indicate higher average fluoride exposure among cases than controls, by a factor of about 2, supporting an association between fluoride exposure and osteosarcoma.

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.

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

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).

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.

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., 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

Health Canada (2010a) claims that there is no evidence that fluoride is an endocrine disruptor. However, based on an extensive review, 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).

Thyroid dysfunction and Type II diabetes presently pose substantial health concerns in both the U.S. and Canada (NRC 2006; PHAC 2011). More than 2 million Canadians (7% of the population) are diabetic (PHAC 2011), and some 10% of Canadians have some form of thyroid disease (TFC 2014). Of particular concern is an inverse correlation between subclinical maternal hypothyroidism and the IQ of the offspring (NRC 2006). 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

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). Most fluoridated water systems in Canada and the U.S. use silicofluorides (NRC 2006; CDA 2009). 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 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).

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 2012).

Neurotoxicity

Grandjean and Landrigan (2006) listed fluoride as an "emerging neurotoxic substance" that needed further in-depth studies. In a follow-up paper (Grandjean and Landrigan 2014), they list fluoride as a documented developmental neurotoxicant. 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). Fluoride is known to cross the placenta in humans (Feltman 1956; Feltman and Kosel 1961; Gedalia et al. 1964; Hanhijärvi et al. 1974; Ron et al. 1986; Malhotra et al. 1993; Gupta et al. 1993; Shimonovitz et al. 1995), and several studies have shown changes in brain chemistry in fetuses due to maternal fluoride exposures (Dong et al. 1997; Du et al. 2008; He et al. 2008; Yu et al. 2000; 2008).

Additional adverse health effects

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.

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).

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 1956; 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).

Summary

The available data, responsibly interpreted, indicate a variety of possible adverse health effects in humans associated with fluoride exposures, at the levels experienced by people with fluoridated drinking water.

(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.

Health Canada (2013) includes as "drug products" several toothpastes and mouthwashes that contain sodium fluoride as an active ingredient. 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 in the U.S. 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; Health Canada 2011b; CDA 2009). EPA's recent reference (Federal

Register 2010) to a "treated population" acknowledges this use of drinking water systems to deliver a drug to entire populations. The Canadian Dental Association (CDA 2009) claims that "Adding fluoride to water is the best way to provide fluoride protection to a large number of people. . . it benefits all residents in a community." This approach, in both the U.S. and Canada, 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.

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 beryllofluorides vs. fluoride alone, as well as the different mechanisms of action of the different chemical combinations. 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 (Brown et al. 2004; Weng et al. 2000; Casale 2001; Mullenix 2014). The U.S. EPA has set MCLGs of zero for both lead and arsenic (EPA

2012). Health Canada (2006; 2012) states that levels of arsenic in drinking water should be as low as reasonably achievable and exposures to lead should be kept to a minimum. Thus, by adding fluoridation chemicals, a water supplier is also adding contaminants for which the ideal maximum amount in drinking water is zero.

In summary, 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.

(4) Expected fluoride intakes in fluoridated communities in Ontario, compared with "no-effect" levels for adverse health effects.

Table 2 summarizes the estimated intake of water from community (municipal) water sources. These estimates are based on U.S. data (EPA 2004a), but are expected to be reasonably representative of the Canadian population as well. Intakes are summarized in terms of the volume per day (mL per day) and the volume per unit body weight per day (mL per kg body weight per day). Data are summarized by age group (both sexes included) and include both direct and indirect intake for consumers only (people who actually consume municipal water). Data are summarized in terms of an average intake, a typical range of intake among consumers, and a value representative of high consumers (but not necessarily a maximum value).

Table 3 provides a summary of estimated fluoride intakes from community water sources for four concentrations of fluoride in drinking water relevant to the situation in Ontario (targeted range of 0.5-0.8 mg/L, Health Canada's "optimal" level of 0.7 mg/L, maximum allowable concentration of 1.5 mg/L). These estimates are based on the water consumption rates (mL per kg per day) in Table 2. Note that these fluoride intakes represent only fluoride from municipal water sources; they do not include fluoride intakes from other sources (e.g., toothpaste, tea, food). Thus, total fluoride intakes would be expected to be higher than the values provided in Table 3 for a given situation.

Figures 6 and 7 summarize estimated fluoride intakes (from community water alone) from Table 3, together with "no-effect" levels identified for various adverse health effects. Note that for the entire population to be protected against a particular adverse health effect, the upper end of the intake range for all subsets (e.g., age groups) of the population must be at or below the "no-effect" level. Note also that these "no-effect" levels do not include any margin of safety for protection of individuals with greater susceptibility or higher exposure.

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

Water fluoride concentration	Children with no caries	Mean DMFS score[b]	Children with fluorosis[c]	Mean severity of fluorosis[d]
mg/L	%		%	
< 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.

Table 2. Estimated intake of water from community sources by age group.[a]

Age group	Average consumption	Typical Range	High consumers
	Intake (mL per day)		
Infants < 1 year	502	28-1147	1517
Children 2-10 years	431	29-1137	1722
Youth 11-19 years	736	58-1973	3689
Adults 20+ years	1176	103-2848	4631
	Intake per unit body weight (mL per kg per day)		
Infants < 1 year	71	3-185	261
Children 2-10 years	21	1-57	92
Youth 11-19 years	13	1-34	60
Adults 20+ years	16	1-39	62

[a] Based on U.S. data (EPA 2004a). Intakes include both direct and indirect intake for consumers only, both sexes combined.

Table 3. Estimated intake of fluoride from fluoridated community water sources (mg F per kg body weight per day), for selected concentrations of fluoride in community water, by age group (both sexes combined) and level of water consumption.[a]

Age group	Average consumption	Typical Range	High consumers
	0.5 mg/L fluoride		
Infants < 1 year	0.036	0.0015-0.093	0.131
Children 2-10 years	0.011	0.0005-0.029	0.046
Youth 11-19 years	0.0065	0.0005-0.017	0.030
Adults 20+ years	0.0080	0.0005-0.020	0.031
	0.7 mg/L fluoride		
Infants < 1 year	0.050	0.0021-0.130	0.183
Children 2-10 years	0.015	0.0007-0.040	0.064
Youth 11-19 years	0.0091	0.0007-0.024	0.042
Adults 20+ years	0.011	0.0007-0.027	0.043
	0.8 mg/L fluoride		
Infants < 1 year	0.057	0.0024-0.148	0.209
Children 2-10 years	0.017	0.0008-0.046	0.074
Youth 11-19 years	0.010	0.0008-0.027	0.048
Adults 20+ years	0.013	0.0008-0.031	0.050
	1.5 mg/L fluoride		
Infants < 1 year	0.107	0.0045-0.278	0.392
Children 2-10 years	0.032	0.0015-0.086	0.138
Youth 11-19 years	0.020	0.0015-0.051	0.090
Adults 20+ years	0.024	0.0015-0.059	0.093

[a] Based on U.S. data (EPA 2004a) for water consumption as summarized in Table 2.

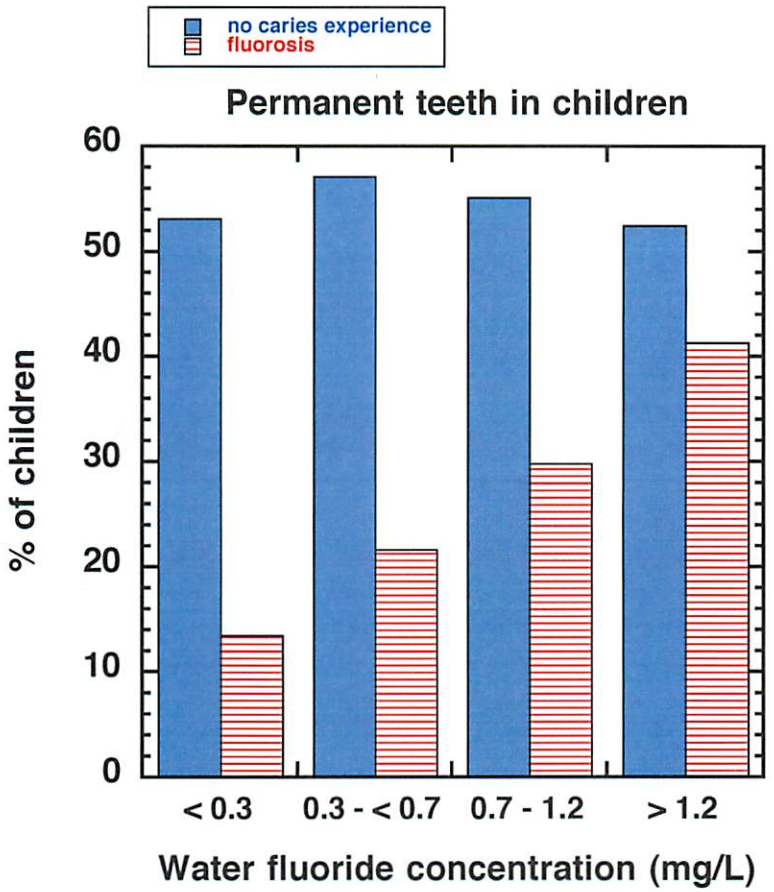

Fig. 1. Percent of children with no caries experience in the permanent teeth (DMFS = 0) and with fluorosis, with respect to water fluoride concentration. Data are shown as % of total children having no caries experience (blue) or having fluorosis (very mild, mild, moderate, or severe, but not questionable;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

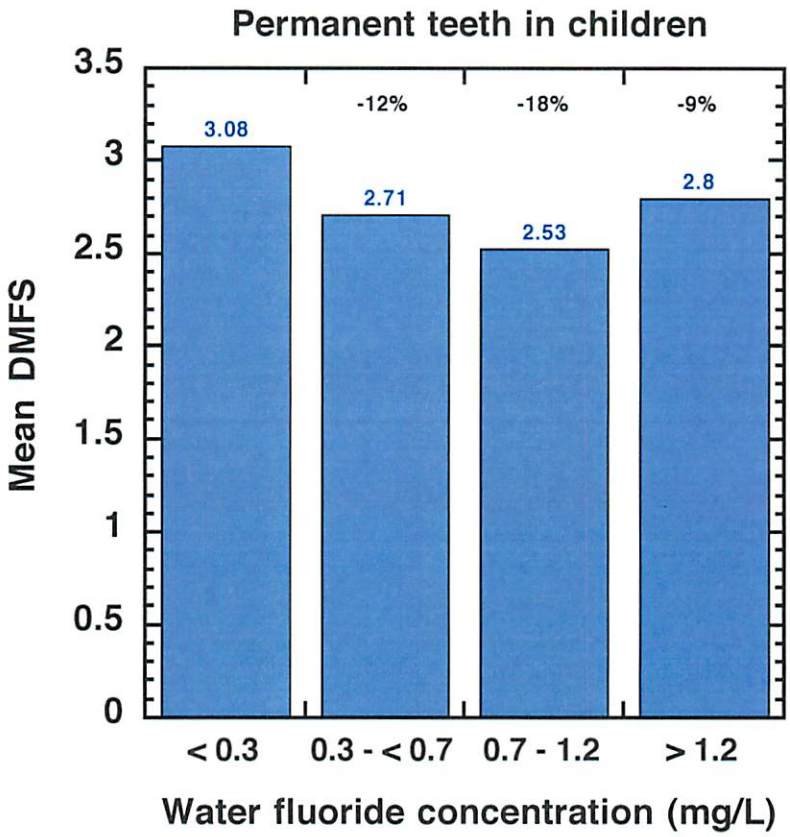

Fig. 2. Mean DMFS score (decayed, missing, or filled permanent tooth surfaces in permanent teeth), with respect to water fluoride concentration. Numerical values are provided in Table 1 of these comments and were obtained from Table 2 of Heller et al. (1997). The percent difference with respect to the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

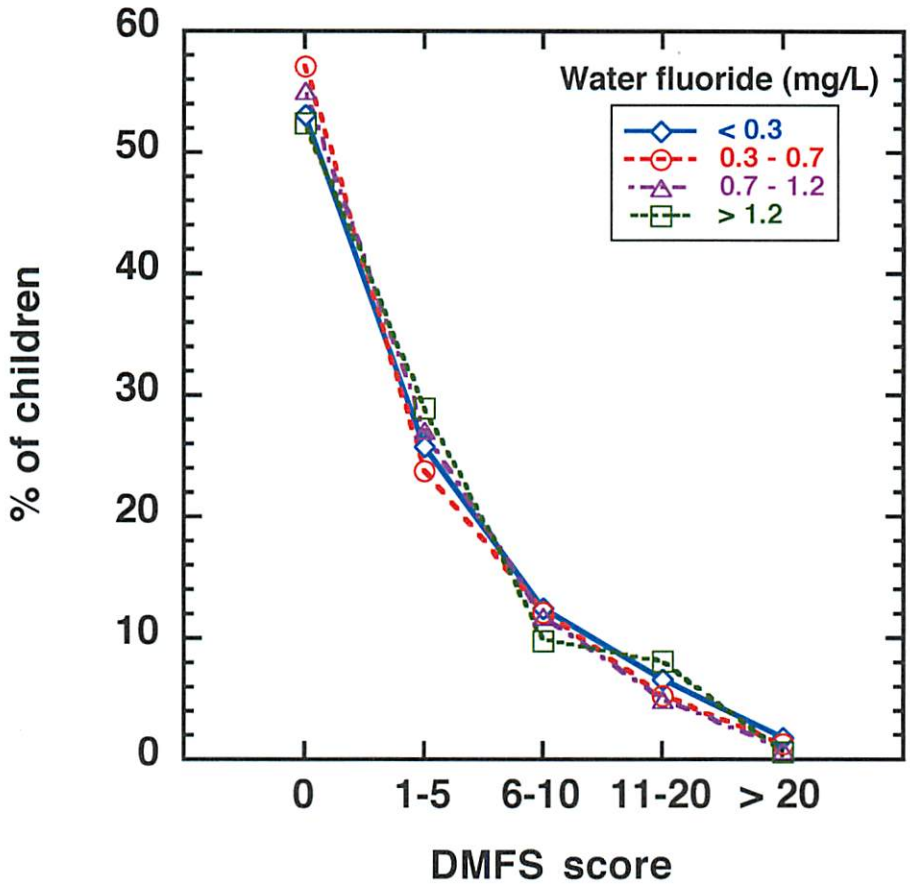

Fig. 3. Percent of children by DMFS score, with respect to water fluoride concentration. Data are shown as % of total children in a given group according to the number of decayed, missing, or filled tooth surfaces in the permanent teeth (DMFS). Data were obtained from Table 2 of Heller et al. (1997).

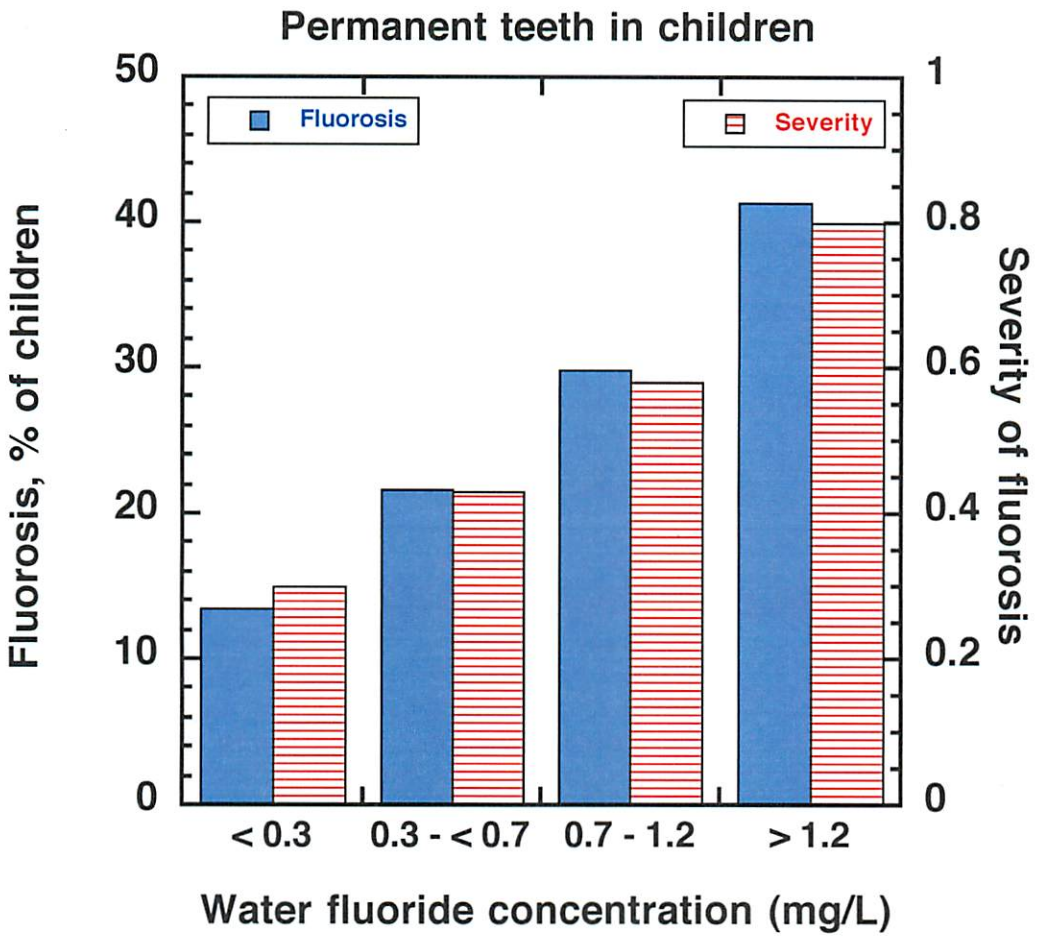

Fig. 4. Fluorosis prevalence and severity with water fluoride concentration for children ages 7-17 with a history of a single continuous residence. Data are shown as (left) % of total children having fluorosis (very mild, mild, moderate, or severe, but not questionable) or (right) severity of fluorosis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

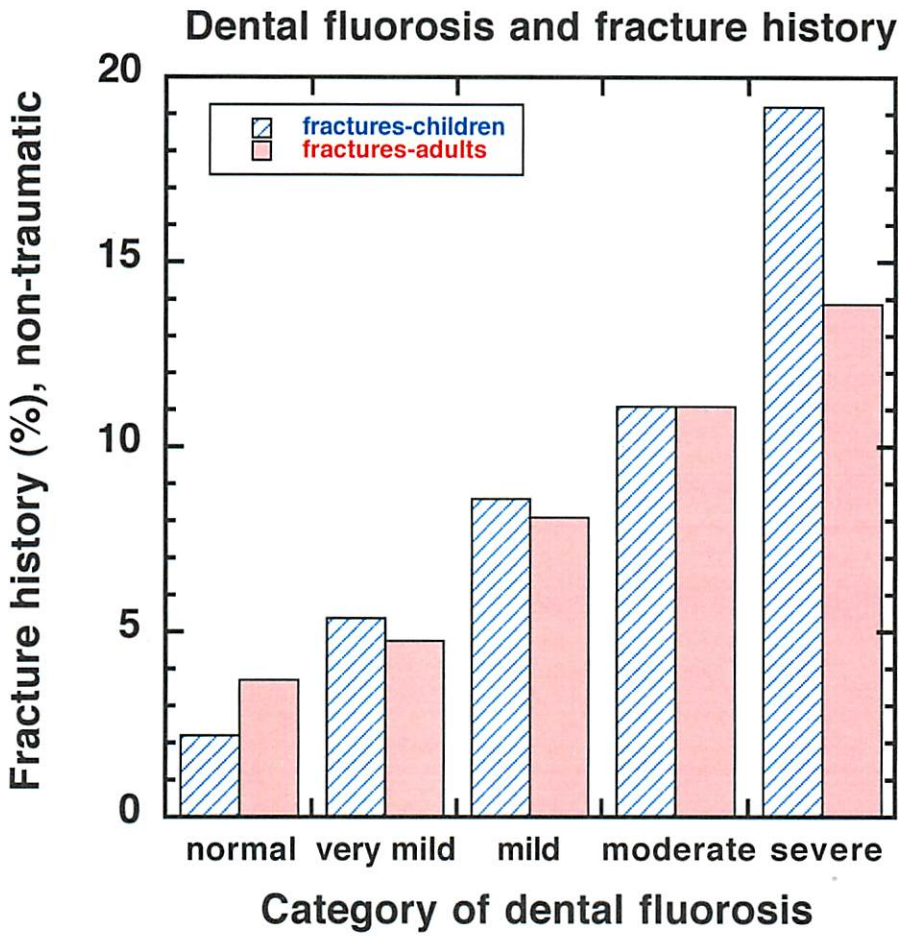

Fig. 5. Fracture history with category of dental fluorosis for children (ages 6-12) and adults (ages 13-60). Numerical values were obtained from information in Tables 5 and 6 of Alarcón-Herrera et al. (2001).

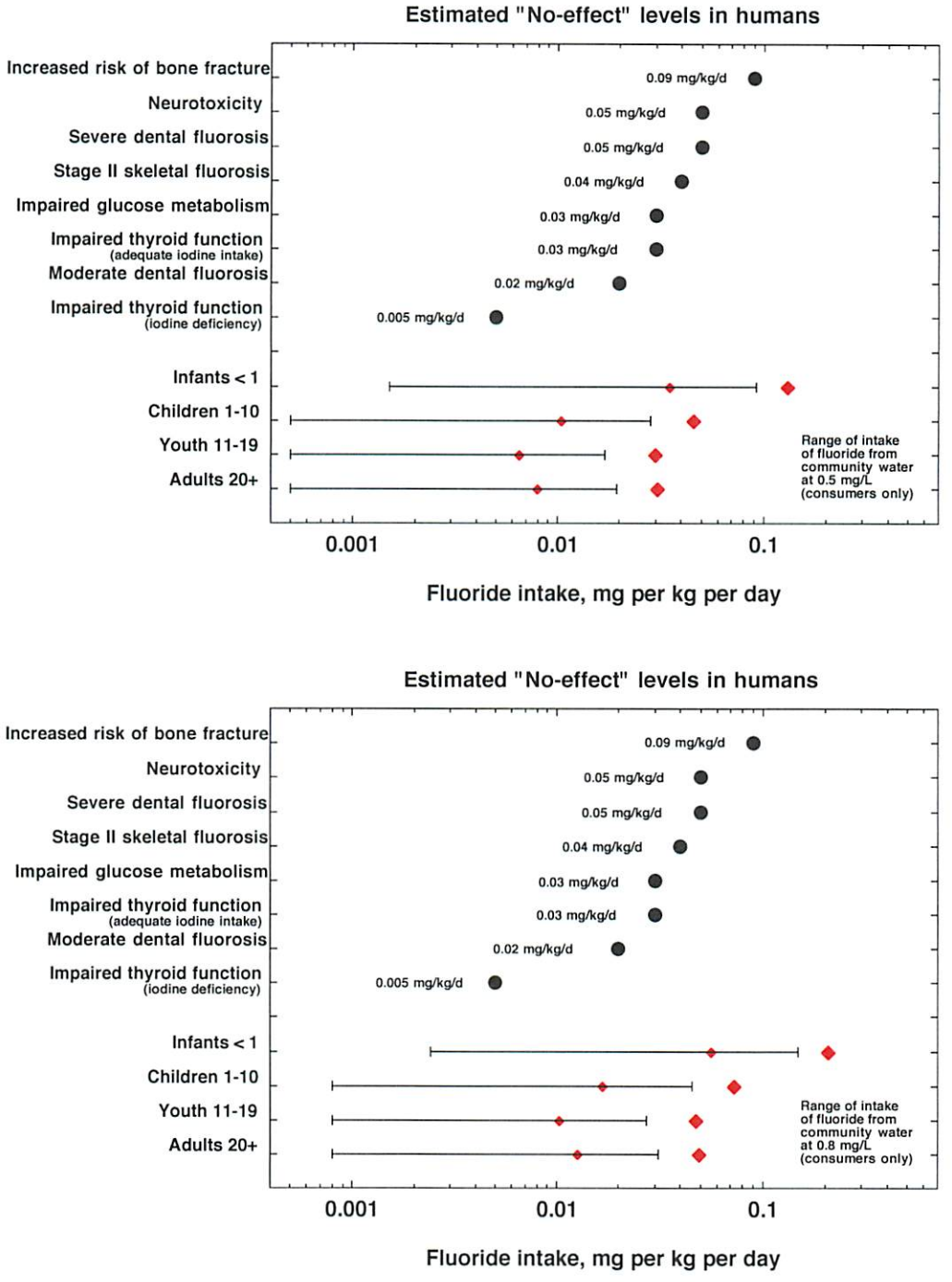

Fig. 6. Comparison of estimated fluoride intakes from community water alone (from Table 3) at the lower and upper limits of the targeted fluoride concentrations for Ontario, 0.5 mg/L (top) and 0.8 mg/L (bottom), with estimated "no-effect" levels of fluoride intake in humans.

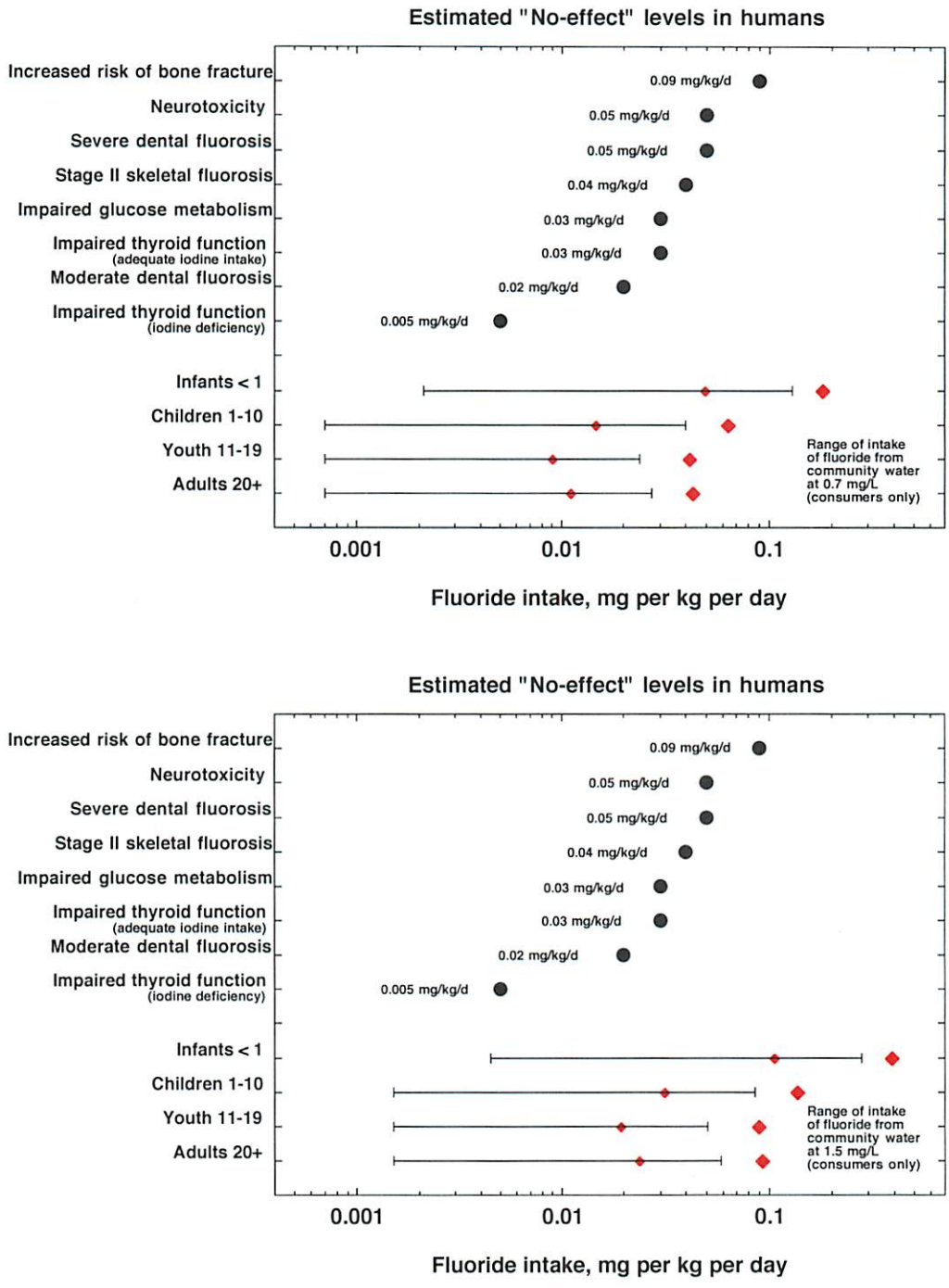

Fig. 7. Comparison of estimated fluoride intakes from community water alone (from Table 3) at Health Canada's "optimal" fluoride concentration, 0.7 mg/L (top), and Maximum Allowable Concentration, 1.5 mg/L (bottom), with estimated "no-effect" levels of fluoride intake in humans.

References

Aardema, M.J., Gibson, D.P., and LeBoeuf, R.A. 1989. Sodium fluoride-induced chromosome aberrations in different stages of the cell cycle: A proposed mechanism. Mutation Research 223:191-203.

Aardema, M.J., and Tsutsui, T. 1995. Sodium fluoride-induced chromosome aberrations in different cell cycle stages. Mutation Research 331:171-172.

ADA (American Dental Association). 2005. Fluoridation facts. Chicago, IL: American Dental Association. [Available: http://www.ada.org/sections/professionalResources/pdfs/fluoridation_facts.pdf]

ADA (American Dental Association). 2007. Infants, formula and fluoride. JADA 138:132. [Available: http://www.ada.org/sections/scienceAndResearch/pdfs/patient_70.pdf]

Adair, S.M., C.M. Hanes, C.M. Russell, and G.M. Whitford. 1999. Dental caries and fluorosis among children in a rural Georgia area. Pediatr. Dent. 21(2):81-85.

Alarcón-Herrera, M.T., Martín-Domínguez, I.R., Trejo-Vázquez, R., and Rodriguez-Dozal, S. 2001. Well water fluoride, dental fluorosis, and bone fractures in the Guadiana Valley of Mexico. Fluoride 34:139-149.

Alvarez, J.O. 1995. Nutrition, tooth development, and dental caries. Am. J. Clin. Nutr. 61S:410S-416S.

Alvarez, J.O., and Navia, J.M. 1989. Nutritional status, tooth eruption, and dental caries: a review. Am. J. Clin. Nutr. 49:417-426.

Arthritis Society. 2013. Arthritis in Canada. Prepared by the Arthritis Community Research and Evaluation Unit (ACREU) for the Arthritis Society. July 2013. [Available: https://www.arthritis.ca/document.doc?id=903]

ATSDR (Agency for Toxic Substances and Disease Registry). 2003. Toxicological Profile for Fluorides, Hydrogen Fluoride, and Fluorine. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA, September 2003.

Barnes, G.P., Parker, W.A., Lyon, T.C., Drum, M.A., and Coleman, G.C. 1992. Ethnicity, Location, Age, and Fluoridation Factors in Baby Bottle Tooth Decay and Caries Prevalence of Head Start Children. Public Heath Reports 107(2):167–173.

Bassin, E.B., Wypij, D., Davis, R.B., and Mittleman, M.A. 2006. Age-specific fluoride exposure in drinking water and osteosarcoma (United States). Cancer Causes Control. 17(4):421-428.

Beltrán-Aguilar, E.D., Barker, L., and Dye, B.A. 2010. Prevalence and severity of dental fluorosis in the United States, 1999–2004. NCHS data brief, no 53. Hyattsville, MD: National Center for Health Statistics.

Biggio, J.R. Jr., Morris, T.C., Owen, J., and Stringer, J.S.A. 2004. An outcomes analysis of five prenatal screening strategies for trisomy 21 in women younger than 35 years. Am. J. Obstet. Gynecol. 190:721-729.

Björnhagen, V., Höjer, J., Karlson-Stiber, C., Seldén, A.I., and Sundbom, M. 2003. Hydrofluoric acid-induced burns and life-threatening systemic poisoning: Favorable outcome after hemodialysis. Clinical Toxicology 41(6):855-860.

Brown, R.A., Cornwell, D.A., and MacPhee, M.J. 2004. Trace contaminants in water treatment chemicals: Sources and fate. Journal AWWA 96(12):111–125.

Buckley, F., Buckley, S. 2008. Wrongful deaths and rightful lives—screening for Down syndrome. Down Syndrome Res Practice 12:79-86.

Casale, R.J. 2001. Improving chemical handling procedures can help reduce associated treatment problems. Journal AWWA 93(9):95–106.

CDA (Canadian Dental Association). 2009. Fluoride Q & A's. July 2009. [Available: http://www.fairview.ca/Fluoride-QA-with-the-CDA1.pdf]

CDC (Centers for Disease Control and Prevention). 1999. Ten great public health achievements—United States, 1900-1999. MMWR 48(12):241-243. [Available: http://www.cdc.gov/mmwr/preview/mmwrhtml/00056796.htm]

CDC (Centers for Disease Control and Prevention). 2001. Recommendations for Using Fluoride to Prevent and Control Dental Caries in the United States. Morbidity and Mortality Weekly Report 50(RR-14). Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

CDC (Centers for Disease Control and Prevention). 2005. Surveillance for Dental Caries, Dental Sealants, Tooth Retention, Edentulism, and Enamel Fluorosis—United States, 1988-1994 and 1999-2002. Morbidity and Mortality Weekly Report 54(SS3):1-43. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

Chachra, D., Limeback, H., Willitt, T.L., and Grynpas, M.D. 2010. The long-term effects of water fluoridation on the human skeleton. J. Dent. Res. 89(11):1219-1223.

Cheng, K.K., Chalmers, I., and Sheldon, T.A. 2007. Adding fluoride to water supplies. BMJ 335:699-702.

Choi, A.L., Sun, G., Zhang, Y., and Grandjean, P. 2012. Developmental fluoride neurotoxicity: A systematic review and meta-analysis. Environmental Health Perspectives 120(10):1362-1368.

Clark, J.D., and Mann, E.H. 1938. A study of the occurrence of fluorine in the drinking water of New Mexico and the menace of fluorine to health. The University of New Mexico Bulletin, Chemistry Series 2(5):3-23.

Colquhoun, J. 1993. Fluorides and the decline in tooth decay in New Zealand. Fluoride 26:125–134.

Connett, P., J. Beck, and H.S. Micklem. 2010. *The Case Against Fluoride*. White River Junction, VT: Chelsea Green Publishing.

Coplan, M.J., Patch, S.C., Masters, R.D., and Bachman, M.S. 2007. Confirmation of and explanations for elevated blood lead and other disorders in children exposed to water disinfection and fluoridation chemicals. NeuroToxicology 28:1032-1042.

Cote, S., P. Geltman, M. Nunn, K. Lituri, M. Henshaw, and R.I. Garcia. 2004. Dental caries of

refugee children compared with US children. Pediatrics 114:e733–e740. [Available at www.pediatrics.org/cgi/doi/10.1542/peds.2004-049]

Cross, D.W., and R.J. Carton. 2003. Fluoridation: A violation of medical ethics and human rights. Int. J. Occup. Environ. Health 9(1):24-29.

DailyMed (2011a). Fluoride (sodium fluoride) tablet, chewable. Available at http://dailymed.nlm.nih.gov/dailymed/lookup.cfm?setid=58b3e7ff-c032-4e42-823a-8ed7b93d606f (revised May 2011).

DailyMed (2011b). Fluoride drops (sodium fluoride) liquid. Available at http://dailymed.nlm.nih.gov/dailymed/lookup.cfm?setid=2e05a9bf-f8e7-41b1-b382-2dc9d8dafee4 (revised May 2011).

DailyMed (2011c). Epiflur TM (sodium fluoride) tablet. Available at http://dailymed.nlm.nih.gov/dailymed/lookup.cfm?setid=97a2ab7a-5a2c-4c35-9bb4-f3cd37dcd180 (revised September 2009).

Danielson, C., Lyon, J.L., Egger, M., and Goodenough, G.K. 1992. Hip fractures and fluoridation in Utah's elderly population. JAMA 268:746-748.

Desai, V.K., Solanki, D.M., and Bansal, R.K. 1993. Epidemiological study on goitre in endemic fluorosis district of Gujarat. Fluoride 26:187-190.

Diesendorf, M. 1986. The mystery of declining tooth decay. Nature 322:125–129.

Dong, Z., Wan, C., Zhang, X., and Liu, J. 1997. Determination of the contents of amino acid and monoamine neurotransmitters in fetal brains from a fluorosis endemic area. Journal of Guiyang Medical College 18(4):241-245.

Du, L., Wan, C., Cao, X., and Liu, J. 2008. The effect of fluorine on the developin human brain. Fluoride 41(4):327-330.

EPA (Environmental Protection Agency). 1988. Summary Review of Health Effects Associated with Hydrogen Fluoride and Related Compounds: Health Issue Assessment. Research Triangle Park, NC: Office of Health and Environmental Assessment, Report No. EPA/600/8-89/002F.

EPA (Environmental Protection Agency). 2004a. Estimated Per Capita Water Ingestion and Body Weight in the United States—An Update: Based on Data Collected by the United States Department of Agriculture's 1994-96 and 1998 Continuing Survey of Food Intakes by Individuals. EPA-822-R-00-001. Office of Water, U.S. Environmental Protection Agency. October 2004.

EPA (Environmental Protection Agency). 2004b. Lead and compounds (inorganic) (CASRN 7439-92-1). Integrated Risk Information System, U.S. Environmental Protection Agency [online]. [Available: http://www.epa.gov/ncea/iris/subst/0277.htm]

EPA (Environmental Protection Agency). 2012. 2012 Edition of the Drinking Water Standards and Health Advisories. Washington, DC: U.S. Environmental Protection Agency, Office of Water, EPA 822-S-12-001. [Available: http://water.epa.gov/drink/standards/hascience.cfm]

Evans, A.M. 1986. Age at puberty and first litter size in early and late paired rats. Biology of Reproduction 34:322-326.

Eyre, R., R.G. Feltbower, E. Mubwandarikwa, T.O.B. Eden, and R.J.Q. McNally. 2009. Epidemiology of bone tumours in children and young adults. Pediatr. Blood Cancer 53:941-952.

FDA (U.S. Food and Drug Administration). Undated-a. Medicines in My Home. Information for students on the safe use of over-the-counter medicines. [Available: http://www.fda.gov/downloads/Drugs/ResourcesForYou/Consumers/BuyingUsingMedicineSafely/UnderstandingOver-the-CounterMedicines/UCM093965.pdf or http://www.fda.gov/downloads/Drugs/ResourcesForYou/Consumers/BuyingUsingMedicineSafely/UnderstandingOver-the-CounterMedicines/UCM094872.pdf]

FDA (U.S. Food and Drug Administration). Undated-b. Medicines in My Home. Information for adults on using over-the-counter medicines safely. [Available: http://www.fda.gov/downloads/Drugs/ResourcesForYou/Consumers/BuyingUsingMedicineSafely/UnderstandingOver-the-CounterMedicines/UCM094872.pdf]

FDA (U.S. Food and Drug Administration). 2011. Unapproved Drugs: Drugs marketed in the United States that do not have required FDA approval. Available at http://www.fda.gov/Drugs/GuidanceComplianceRegulatoryInformation/EnforcementActivitiesbyFDA/SelectedEnforcementActionsonUnapprovedDrugs/default.htm (updated August 5, 2011).

Featherstone, J.D.B. 2000. The science and practice of caries prevention. JADA 131:887-899.

Federal Register. 2010. National Primary Drinking Water Regulations; Announcement of the Results of EPA's Review of Existing Drinking Water Standards and Request for Public Comment and/or Information on Related Issues. Federal Register 75(59):15500-15572, March 29, 2010.

Federal Register. 2011. Proposed HHS Recommendation for Fluoride Concentration in Drinking Water for Prevention of Dental Caries. Federal Register 76(9):2383-2388, January 13, 2011.

Feltman, R. 1956. Prenatal and postnatal ingestion of fluorides: A progress report. Dental Digest 62:353-357.

Feltman, R., and Kosel, G. 1961. Prenatal and postnatal ingestion of fluorides—Fourteen years of investigation. Final report. J. Dent. Med. 16:190-198.

Finn, S.B. 1952. Prevalence of dental caries. In: A Survey of the Literature of Dental Caries. Washington, DC: National Academy of Sciences, Publication 225, pp. 117-173.

Finn, S.B., and R.C. Caldwell. 1963. Dental caries in twins. I. A comparison of the caries experience of monozygotic twins, dizygotic twins and unrelated children. Archives of Oral Biology 8:571-585.

Forrester, M.B., and Merz, R.D. 1999. Prenatal diagnosis and elective termination of Down syndrome in a racially mixed population in Hawaii, 1987-1996. Prenat. Diagn. 19:136-141.

Franke, J., Rath, F., Runge, H., Fengler, F., Auermann, E., and Lenart, G.L. 1975. Industrial fluorosis. Fluoride 8:61-85.

Gedalia, I., Brzezinski, A., Zukerman, H., and Mayersdorf, A. 1964. Placental transfer of fluoride in the human fetus at low and high F-intake. J. Dent. Res. 43(5):669-671.

Gelberg, K.H., Fitzgerald, E.F., Hwang, S., and Dubrow, R. 1995. Fluoride exposure and childhood osteosarcoma: A case-control study. American Journal of Public Health 85(12):1678-1683.

Graham, J.R., and Morin, P.J. 1999. Highlights in North American litigation during the twentieth century on artificial fluoridation of public water supplies. J. Land Use & Environmental Law 14(2):195-242.

Grandjean, P., and Landrigan, P.J. 2006. Developmental neurotoxicity of industrial chemicals. Lancet 368:2167-2178.

Grandjean, P., and Landrigan, P.J. 2014. Neurobehavioural effects of developmental toxicity. Lancet Neurology 13:330-338.

Gray, L.E., Jr., Wilson, V., Noriega, N., Lambright, C., Furr, J., Stoker, T.E., Laws, S.C., Goldman, J., Cooper, R.L., and Foster, P.M.D. 2004. Use of the laboratory rat as a model in endocrine disruptor screening and testing. ILAR Journal 45(4):425-437.

Grimbergen, G.W. 1974. A double blind test for determination of intolerance to fluoridated water. Preliminary Report. Fluoride 7:146-152.

Gupta, S., Seth, A.K., Gupta, A., and Gavane, A.G. 1993. Transplacental passage of fluoride. The Journal of Pediatrics 123(1):139-141.

Hallanger Johnson, J.E., Kearns, A.E., Doran, P.M., Khoo, T.K., and Wermers, R.A. 2007. Fluoride-related bone disease associated with habitual tea consumption. Mayo Clin Proc 82:719-724.

Hamasha, A.A., J.J. Warren, S.M. Levy, B. Broffitt, and M.J. Kanellis. 2006. Oral health behaviors of children in low and high socioeconomic status families. Pediatr. Dent. 28(4):310-315.

Hanhijärvi, H., Kanto, J., and Ruponen, S. 1974. Human free ionized plasma fluoride concentrations during pregnancy, toxemia, and lactation. Fluoride 7(3):143-146.

Hattis, D., Goble, R., Russ, A., Chu, M., and Ericson, J. 2004. Age-related differences in susceptibility to carcinogenesis: A quantitative analysis of empirical animal bioassay data. Environmental Health Perspectives 112:1152–1158.

He, H., Cheng, Z., and Liu, W. 2008. Effects of fluorine on the human fetus. Fluoride 41(4)321-326.

Health Canada. 2006. Guidelines for Canadian Drinking Water Quality: Guideline Technical Document—Arsenic. Water Quality and Health Bureau, Healthy Environments and Consumer Safety Branch, Health Canada, Ottawa, Ontario. May 2006. [Available: http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/water-eau/arsenic/arsenic-eng.pdf]

Health Canada. 2010a. Guidelines for Canadian Drinking Water Quality: Guideline Technical Document—Fluoride. Water, Air and Climate Change Bureau, Healthy Environments and Consumer Safety Branch, Health Canada, Ottawa, Ontario. December 2010. [Available: http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/water-eau/2011-fluoride-fluorure/2011-fluoride-fluorure-eng.pdf]

Health Canada. 2010b. It's Your Health: Fluoride and Human Health. October 2010. [Available: http://www.hc-sc.gc.ca/hl-vs/alt_formats/pacrb-dgapcr/pdf/iyh-vsv/environ/fluor-eng.pdf]

Health Canada. 2010c. Report on the Findings of the Oral Health Component of the Canadian Health Measures Survey 2007-2009. [Available: http://www.fptdwg.ca/assets/PDF/CHMS/CHMS-E-tech.pdf]

Health Canada. 2011a. Office of the Chief Dental Officer Projects. June 2011. [Available: http://www.hc-sc.gc.ca/ahc-asc/branch-dirgen/fnihb-dgspni/ocdo-bdc/project-eng.php#a6]

Health Canada. 2011b. Environmental and Workplace Health. Fluoride in Drinking Water. June 2011. [Available: http://www.hc-sc.gc.ca/ewh-semt/water-eau/drink-potab/health-sante/faq_fluoride-fluorure-eng.php]

Health Canada. 2012. Guidelines for Canadian Drinking Water Quality—Summary Table. Water, Air and Climate Change Bureau, Healthy Environments and Consumer Safety Branch, Health Canada, Ottawa, Ontario. August 2012. [Available: http://www.hc-sc.gc.ca/ewh-semt/alt_formats/pdf/pubs/water-eau/2012-sum_guide-res_recom/2012-sum_guide-res_recom-eng.pdf]

Health Canada. 2013. Drug Product Database Online Query. [Available: http://webprod5.hc-sc.gc.ca/dpd-bdpp/start-debuter.do?lang=eng]

Heller, K.E., Eklund, S.A., and Burt, B.A. 1997. Dental caries and dental fluorosis at varying water fluoride concentrations. J. Public Health Dentistry 57(3):136-143.

Hileman, B. 1990. Fluoride bioassay study under scrutiny. Chemical & Engineering News 68(38):29-30 (September 17, 1990).

Hirzy, J.W. 2000. Statement of Dr. J. William Hirzy, National Treasury Employees Union Chapter 280, before the Subcommittee on Wildlife, Fisheries and Drinking Water, United States Senate, June 29, 2000. [Available: http://www.nteu280.org/Issues/Fluoride/629FINAL.htm or http://www.fluoridealert.org/HirzyTestimony.pdf]

Hong, L., Levy, S.M., Broffitt, B., Warren, J.J., Kanellis, M.J., Wefel, J.S., and Dawson, D.V. 2006a. Timing of fluoride intake in relation to development of fluorosis on maxillary central incisors. Community Dent. Oral Epidemiol. 34:299-209.

Hong, L., Levy, S.M., Warren, J.J., Broffitt, B., and Cavanaugh, J. 2006b. Fluoride intake levels in relation to fluorosis development in permanent maxillary central incisors and first molars. Caries Research 40:494-500.

Iida, H., and Kumar, J.V. 2009. The association between enamel fluorosis and dental caries in U.S. schoolchildren. JADA 140:855-862.

Jooste, P.L., Weight, M.J., Kriek, J.A., and Louw, A.J. 1999. Endemic goitre in the absence of iodine deficiency in schoolchildren of the Northern Cape Province of South Africa. Eur. J. Clin. Nutr. 53:8-12.

Khosla, S., Melton, L.J. III, Dekutoski, M.B., Achenbach, S.J., Oberg, A.L., Riggs, B.L. 2003. Incidence of childhood distal forearm fractures over 30 years. JAMA 290:1479-1485.

Kim, F.M., C. Hayes, P.L. Williams, G.M. Whitford, K.J. Joshipura, R.N. Hoover, C.W. Douglass, and the National Osteosarcoma Etiology Group. 2011. An assessment of bone fluoride and osteosarcoma. J. Dent. Res. Epub ahead of print, July 28, 2011.

Kishi, K., and Ishida, T. 1993. Clastogenic activity of sodium fluoride in great ape cells. Mutation Research 301:183-188.

Komárek, A., Lesaffre, E., Härkänen, T., Declerck, D., and Virtanen, J.I. 2005. A Bayesian analysis of multivariate doubly-interval-censored dental data. Biostatistics 6(1):145-155.

Kurland, E.S., Schulman, R.C., Zerwekh, J.E., Reinus, W.R., Dempster, D.W., and Whyte, M.P. 2007. Recovery from skeletal fluorosis (an enigmatic, American case). J Bone Mineral Res. 22:163-170.

Lasne, C., Lu, Y.-P., and Chouroulinkov, I. 1988. Transforming activities of sodium fluoride in cultured Syrian hamster embryo and BALB/3T3 cells. Cell Biology and Toxicology 4(3):311-324.

Leonnig, C.D. 2010. CDC misled District residents about lead levels in water, House probe finds. The Washington Post, May 20, 2010 [Available: http://www.washingtonpost.com/wp-dyn/content/article/2010/05/19/AR2010051902599_pf.html]

Levy, S.M., Eichenberger-Gilmore, J., Warren, J.J., Letuchy, E., Broffitt, B., Marshall, T.A., Burns, T., Willing, M., Janz, K., and Torner, J.C. 2009. Associations of fluoride intake with children's bone measures at age 11. Community Dent. Oral Epidemiol. 37:416-426.

Li, X.S., Zhi, J.L., and Gao, R.O. 1995. Effect of fluoride exposure on intelligence in children. Fluoride 28:189-192.

Limeback, H., and Robinson, C. 2012. Fluoride Therapy. Ch 16 in: Comprehensive Preventive Dentistry, Limeback, H.(Ed.). Ames, Iowa, West Sussex, and Oxford, John Wiley & Sons, Ltd., pp. 251-282.

Lin, F.F., Aihaiti, Zhao, H.X., Lin, J., Jiang, J.Y., Maimaiti, and Aiken. 1991. The relationship of a low-iodine and high-fluoride environment to subclinical cretinism in Xinjiang. IDD Newsletter 7:24-25.

Lu, Y., Sun, Z.R., Wu, L.N., Wang, X., Lu, W., and Liu, S.S. 2000. Effect of high-fluoride water on intelligence in children. Fluoride 33(2):74-78.

Maas, R.P., Patch, S.C., and Smith, A.M. 2005. Effects of Fluorides and Chloramines on Lead Leaching from Leaded-Brass Surfaces. Technical Report 05-142. Environmental Quality Institute, University of North Carolina, Asheville, NC. June 2005.

Maas, R.P., Patch, S.C., Christian, A.-M., and Coplan, M.J. 2007. NeuroToxicology 28:1023-1031.

Malhotra, A., Tewari, A., Chawla, H.S., Gauba, K., and Dhall, K. 1993. J. Indian Soc. Pedo. Prev. Dent. 11:1-3.

Marcus, W.L. 1990. Fluoride Conference to review the NTP Draft Fluoride Report. Memorandum to A.B. Hais, Acting Director, Criteria & Standards Division, Office of Drinking Water, Environmental Protection Agency.

Masters, R.D., and Coplan, M. 1999. Water treatment with silicofluorides and lead toxicity. Int. J. Environ. Sci. 56:435-449.

Masters, R.D., Coplan, M.J., Hone, B.T., and Dykes, J.E. 2000. Association of silicofluoride treated water with elevated blood lead. Neurotoxicology 21(6):1091-1100.

McDonagh, M., Whiting, P., Bradley, M., Cooper, J., Sutton, A., Chestnutt, I., Misso, K., Wilson, P., Treasure, E., and Kleijnen, J. 2000. A Systematic Review of Public Water Fluoridation. NHS Centre for Reviews and Dissemination, University of York, York, UK.

Medline Plus. 2008. Fluoride. U.S. National Library of Medicine and National Institutes of Health. [Available: http://www.nlm.nih.gov/medlineplus/druginfo/meds/a682727.html]

Meng, Z., and Zhang, B. 1997. Chromosomal aberrations and micronuclei in lymphocytes of workers at a phosphate fertilizer factory. Mutation Research 393:283-288.

Mullenix, P.J. 2014. A new perspective on metals and other contaminants in fluoridation chemicals. IJOEH 20(2):157–166.

Neurath, C. 2005. Tooth decay trends for 12 year olds in nonfluoridated and fluoridated countries. Fluoride 38:324-325.

NRC (National Research Council). 2006. Fluoride in Drinking Water: A Scientific Review of EPA's Standards. [Available: http://www.nap.edu/catalog/11571.html]

NRC (National Research Council). 2009. Emergency and Continuous Exposure Guidance Levels for Selected Submarine Contaminants: Volume 3. [Available: http://www.nap.edu/catalog.php?record_id=12741]

NTP (National Toxicology Program). 1990. Toxicology and carcinogenesis studies of sodium fluoride (CAS No. 7681-49-4) in F344/N rats and B6C3F$_1$ mice (drinking water studies). National Toxicology Program Technical Report Series No. 393.

OMHLTC (Ontario Ministry of Health and Long-Term Care). 2011. Drinking Water Fluoridation. Statement from Dr. Arlene King, Chief Medical Officer of Health. April 2011. [Available: http://www.health.gov.on.ca/en/news/bulletin/2011/hb_20110404_2.aspx]

Penman, A.D., Brackin, B.T., and Embrey, R. 1997. Outbreak of acute fluoride poisoning caused by a fluoride overfeed, Mississippi, 1993. Public Health Reports 112:403-409.

Petraborg, H.T. 1977. Hydrofluorosis in the fluoridated Milwaukee area. Fluoride 10:165-169.

PHAC (Public Health Agency of Canada). 2011. Diabetes in Canada: Facts and figures from a public health perspective. Public Health Agency of Canada, Ottawa. [Available: http://www.phac-aspc.gc.ca/cd-mc/publications/diabetes-diabete/facts-figures-faits-chiffres-2011/pdf/facts-figures-faits-chiffres-eng.pdf]

Psoter, W.J., Reid, B.C., and Katz, R.V. 2005. Malnutrition and dental caries: A review of the literature. Caries Res. 39:441-447.

Rocha-Amador, D., Navarro, M.E., Carrizales, L., Morales, R., and Calderón, J. 2007. Decreased intelligence in children and exposure to fluoride and arsenic in drinking water. Cad. Saúde Pública (Rio de Janeiro) 23 Sup 4:S579-S587.

Rocha-Amador, D., Navarro, M., Trejo-Acevedo, A., Carrizales, L., Pérez-Maldonado, I., Díaz-

Barriga, F., and Calderón, J. 2009. Use of the Rey-Osterrieth Complex Figure Test for neurotoxicity evaluation of mixtures in children. NeuroToxicology 30:1149-1154.

Román, G.C. 2007. Autism: Transient in utero hypothyroxinemia related to maternal flavonoid ingestion during pregnancy and to other environmental antithyroid agents. J. Neurological Sciences 262:15-26.

Ron, M., Singer, L., Menczel, J., and Kidroni, G. 1986. Fluoride concentration in amniotic fluid and fetal cord and maternal plasma. Eur. J. Obstet. Gynecol. Reprod. Biol. 21:213-218.

Sawan, R.M.M., Leite, G.A.S., Saraiva, M.C.P., Barbosa, F. Jr., Tanus-Santos, J.E., and Gerlach, R.F. 2010. Fluoride increases lead concentrations in whole blood and in calcified tissues from lead-exposed rats. Toxicology 271(1-2):21-26.

Saxena, S., Sahay, A., and Goel, P. 2012. Effect of fluoride exposure on the intelligence of school children in Madhya Pradesh, India. J. Neurosci. Rural Pract. 3(2):144-149.

Seraj, B., Shahrabi, M., Shadfar, M., Ahmadi, R., Fallahzadeh, M., Eslamlu, H.F., and Kharazifard, M.J. 2012. Effect of high water fluoride concentration on the intellectual development of children in Makoo/Iran. J. Dentistry (Tehran) 9(3):221-229.

Sergi, C., and Zwerschke, W. 2008. Osteogenic sarcoma (osteosarcoma) in the elderly: Tumor delineation and predisposing conditions. Experimental Gerontology 43:1039-1043.

Shiboski, C.H., Gansky, S.A., Ramos-Gomez, F., Ngo, L., Isman, R., Pollick, H.F. 2003. The association of early childhood caries and race/ethnicity among California preschool children. J. Public Health Dentistry 63:38-46.

Shimonovitz, S., Patz, D., Ever-Hadani, P., Singer, L., Zacut, D., Kidroni, G., and Ron, M. 1995. Umbilical cord fluoride serum levels may not reflect fetal fluoride status. J. Perinat. Med. 23:279-282.

Short, E.M. 1944. Domestic water and dental caries: VI. The relation of fluoride domestic waters to permanent tooth eruption. J. Dent. Res. 23:247-255.

Siffel, C., Correa, A., Cragan, J., Alverson, C.J. 2004. Prenatal diagnosis, pregnancy terminations and prevalence of Down syndrome in Atlanta. Birth Defects Res. A Clin. Mol. Teratol. 70:565-571.

Spittle, B. 2008. Dyspepsia associated with fluoridated water. Fluoride 41(1):89-92.

Steingraber, S. 2007. The Falling Age of Puberty in U.S. Girls: What We Know, What We Need to Know. San Francisco: The Breast Cancer Fund.

Sullivan, K.M. 2009. Iodine deficiency as a cause of autism. J. Neurological Sciences 276:202.

Susheela, A.K., Bhatnagar, M., Vig, K., and Mondal, N.K. 2005. Excess fluoride ingestion and thyroid hormone derangements in children living in Delhi, India. Fluoride 38:98-108.

TFC (Thyroid Foundation of Canada). 2014. About Thyroid Disease. [Available: http://www.thyroid.ca/thyroid_disease.php]

Tickner, J., and Coffin, M. 2006. What does the precautionary principle mean for evidence-based dentistry? Journal of Evidence-Based Dental Practice 6(1):6-15.

Turner, C.H., Garetto, L.P., Dunipace, A.J., Zhang, W., Wilson, M.E., Grynpas, M.D., Chachra, D., McClintock, R., Peacock, M., and Stookey, G.K. 1997. Fluoride treatment increased serum IGF-1, bone turnover, and bone mass, but not bone strength, in rabbits. Calcif. Tissue Int. 61:77-83.

Turner, C.H., Hinckley, W.R., Wilson, M.E., Zhang, W., and Dunipace, A.J. 2001. Combined effects of diets with reduced calcium and phosphate and increased fluoride intake on vertebral bone strength and histology in rats. Calcif. Tissue Int. 69:51-57.

Vohra, R., Velez, L.I., Rivera, W., Benitez, F.L., and Delaney, K.A. 2008. Recurrent life-threatening ventricular dysrhythmias associated with acute hydrofluoric acid ingestion: Observations in one case and implications for mechanism of toxicity. Clinical Toxicology 46:79-84.

Waldbott, G.L. 1956. Incipient chronic fluoride intoxication from drinking water. II. Distinction between allergic reactions and drug intolerance. Int. Arch. Allergy 9:241-249.

Waldbott, G.L. 1958. Allergic reactions from fluorides. Int. Arch. Allergy 12:347-355.

Warren, J.J., Levy, S.M., Broffitt, B., Cavanaugh, J.E., Kanellis, M.J., and Weber-Gasparoni, K. 2009. Considerations on optimal fluoride intake using dental fluorosis and dental caries outcomes—A longitudinal study. J. Public Health Dentistry 69:111-115.

Weng, C.-N., Smith, D.B., and Huntley, G.M. 2000. Treatment chemicals contribute to arsenic levels. Opflow 10:6–7.

Whyte, M.P., Essmyer, K., Gannon, F.H., and Reinus, W.R. 2005. Skeletal fluorosis and instant tea. Am. J. Med. 118:78-82.

Wilson, P.M., and Sheldon, T.A. 2006. Muddy waters: evidence-based policy making, uncertainty and the "York review" on water fluoridation. Evidence & Policy 2(3):321-331.

Xiang, Q., Liang, Y., Chen, L., Wang, C., Chen, B., Chen, X., and Zhou, M. 2003. Effect of fluoride in drinking water on children's intelligence. Fluoride 36(2):84-94.

Yang, Y., Wang, X., and Guo, X. 1994. Effects of high iodine and high fluorine on children's intelligence and the metabolism of iodine and fluorine [in Chinese]. Zhonghua Liu Xing Bing Xue Za Zhi 15:296-298.

Yu, Y. 2000. Effects of fluoride on the ultrastructure of glandular epithelial cells of human fetuses. Chinese Journal of Endemiology 19(2):81-83.

Yu, Y., Yang, W., Dong, Z., Wan, C., Zhang, J., Liu, J., Xiao, K., Huang, Y., and Lu, B. 2008. neurotransmitter and receptor changes in the brains of fetuses from areas of endemic fluorosis. Fluoride 41(2):134-138.

Zhang, R., Niu, Y., Du, H., Cao, X., Shi, D., Hao, Q., and Zhou, Y. 2009. A stable and sensitive testing system for potential carcinogens based on DNA damage-induced gene expression in human HepG2 cell. Toxicology in Vitro 23:158-165.

Zhao, L.B., Liang, G.H., Zhang, D.N., and Wu, X.R. 1996. Effect of a high fluoride water supply on children's intelligence. Fluoride 29:190-192.

Sworn before me at the City of Oak Ridge)
in the State of Tennessee this 29th day)
of April, 2014.)
)

NOTARY

KATHLEEN THIESSEN


KIMBERLY H. DISNEY
STATE OF TENNESSEE
NOTARY PUBLIC
ANDERSON COUNTY