Comments on
the U.S. EPA’s Proposed Rule,
published in the Federal Register on January 11, 2017:

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

Docket ID No. EPA-HQ-OW-2016-0627

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INDEX:

Page 3.  Introduction

Page 3.  FAN’s Response

Page 4.  FAN’s Request


Page 37.  Part 3: Cancer and its Cost Due to Fluoridation Chemicals

Page 39.  Part 4: Fluoride and Cancer, particularly Osteosarcoma

Page 40.  Part 5: Food treated with Highly Neurotoxic Fumigant

Page 42.  Part 6: Fluoride and Environmental Justice

Page 88.  Part 7: Millions of Pounds of Legally Emitted Fluoride Compounds

Page 96.  Against Forgetting: Published Fluoride Studies: 2010 – February 2017

PP 159-219.  References for Part 1: The Neurotoxicity of Fluoride

Page 195.  APPENDIX A:
Post-NRC Human Studies Investigating Fluoride’s Impact on Cognition

Page 201.  APPENDIX B:
Post-NRC Human Studies Investigating Fluoride’s Impact on Fetal Brain

Page 202.  APPENDIX C:
Post-NRC Human Studies Investigating Fluoride’s Impact on Other Parameters of Neurotoxicity

Page 203.  APPENDIX D:
Post-NRC Animal Studies Investigating Fluoride’s Neuroanatomical & Neurochemical Effects

Page 212.  APPENDIX E:
Post-NRC Animal Studies Investigating Fluoride’s Effect on Learning/Memory

Page 215.  APPENDIX F:
Post-NRC Animal Studies Investigating Fluoride’s Effect on Other Behavioral Parameters Beyond Learning/Memory

Page 217.  APPENDIX G:
Post-NRC In Vitro Studies Investigating Fluoride’s Effect on Brain Cells

PP 220-231.  References for Part 6: Fluoride and Environmental Justice

INTRODUCTION:

The Fluoride Action Network (FAN) submits the following response to the Environmental Protection Agency’s (EPA) decision to forego a Six-Year Review of fluoride.

FAN was founded in 2000 as a project of the American Environmental Health Studies Project, Inc. FAN is an organization of scientists, doctors, dentists, environmental health researchers, and concerned citizens working to raise awareness about the impact of current fluoride exposures on human health.

On January 11, 2017, the EPA published a Proposed Rule in the Federal Register on its decision to defer a Six-Year Review of fluoride for the National Primary Drinking Water Regulations (NPDWR). According to EPA, the purpose of a Six-Year Review is

> to evaluate current information for regulated contaminants to determine if there is new information on health effects, treatment technologies, analytical methods, occurrence and exposure, implementation and/or other factors that provides a health or technical basis to support a regulatory revision that will improve or strengthen public health protection.

EPA stated in the Proposed Rule:

> The Agency has determined that a revision to the NPDWR for fluoride is not appropriate at this time. EPA acknowledges information regarding the exposure and health effects of fluoride (as discussed later in the “Health Effects” and “Occurrence and Exposure” sections). However, with EPA’s identification of several other significant NPDWRs as candidates for near-term revision (see Sections VI.B.3 and VI.B.4), potential revision of the fluoride NPDWR is a lower priority that would divert significant resources from the higher priority candidates for revision that the Agency has identified, as well as other high priority work within the drinking water office.

In Table VI-1 of the Proposed Rule, EPA categorized fluoride as:

> Low priority and/or no meaningful opportunity

FAN’s RESPONSE:

FAN disagrees with EPA’s decision to defer performing a review as we find that fluoride poses unacceptable risks to the fetus, infant, child, and adult. In April 2011 FAN submitted two substantive submissions to EPA. Because EPA never responded to these submissions, we attach them as a major part of this submission as they are relevant to the risks we are concerned with and also deserving of a response. Added to those submissions we include the following:

- The CDC reported that 214,213,860 people in the U.S. were on fluoridated drinking water systems in 2014 (the 2016 statistics are not yet available). That is approximately four million more people receiving fluoridated water since FAN’s submissions in April 2011.

- The Neurotoxicity of Fluoride (pp 7-34). This new section, written by Michael Connett, includes 196 published studies that have addressed the neurotoxic effects of fluoride exposure subsequent to the National Research Council’s 2006 report, including 61 human studies.

studies, 115 animal studies, 17 cell studies, and 3 systematic reviews. In FAN’s 2011 submissions we listed 15 studies reporting an association of fluoride and reduced IQ (see pages 39-41) - today there are 50 studies. A Flash Drive containing over 300 studies referenced in this section was sent to EPA’s Docket Reading Room.

• Fluoride was labeled a developmental neurotoxin in 2014; this is discussed in the above section on the Neurotoxicity of Fluoride.

• In a 2015 study, Malin & Till found a statistically significant correlation between the prevalence of water fluoridation at the state level and Attention-Deficit Hyperactivity Disorder (ADHD). This is discussed in the above section on the Neurotoxicity of Fluoride.

• In a 2015 study, Peckham et al. reported an association or risk of higher levels of hypothyroidism in practices in fluoridated areas across England. (Approximately 10% of the population live in fluoridated areas.)

• In 2016, Hirzy et al. published a risk assessment that found U.S. children receive unsafe levels of fluoride (pp 35-36).

• In 2013, Hirzy et al. reported on the cancers due to, and their costs associated with, the chemicals used in community water fluoridation programs (pp 37-38).

• Fluoride and Cancer, particularly Osteosarcoma (p 39)

• Food treated with Highly Neurotoxic Fumigant (pp 40-41)

• Fluoridation is an Environmental Justice Issue (pp 42-85)

• According to the Toxic Release Inventory there are millions of pounds of fluoride and fluorine emissions released legally each year into the environment, yet no regulatory agency is studying or protecting the residents who live downwind of these facilities. (pp 88-94)

• Against Forgetting: Published Fluoride Studies: 2010 – February 2017 (pp 95-158). The top five categories with the most studies for this time period are: 104 Bone/Joint studies; 82 Reproductive studies; 81 Animal Brain Studies; 75 Dental Fluorosis studies; 75 Total Body Burden studies; 48 Kidney studies. This list updates the studies we submitted to EPA in 2011 in Appendix A: Selected studies published since the release of the NRC report in 2006.

• NHANES in 2014 reported dental fluorosis rates at 58.3% of U.S. surveyed adolescents, including an astonishing 21.2% with moderate dental fluorosis, and 2% with severe dental fluorosis. This represents significant over-exposure to fluoride in America’s children.

• NHANES in 2016 reported that 350,000 U.S. children (1 in 200) have serum fluoride levels in the approximate range associated with overt neurotoxic effects.

• According to the 2015 Cochrane Report, “We did not identify any evidence, meeting the review’s inclusion criteria, to determine the effectiveness of water fluoridation for preventing caries in adults.”

• In 2015, Ko and Thiessen released a study that found no cost-savings from community water fluoridation because of the costs associated with treating dental fluorosis.
FAN’s Request: An Immediate Moratorium

Because of the lack of timeliness that EPA has demonstrated in responding to the public’s concerns on fluoride, and the critical nature of those concerns, FAN requests the EPA’s Office of Water place an immediate moratorium on drinking water fluoridation in order to protect the health of the public while EPA finds the time to examine the material in this submission, as well as the submissions of others, including those submitted by Kathleen Thiessen PhD.

References:


1. INTRODUCTION

Water fluoridation began in the U.S. in the 1940s on the premise that fluoride’s primary benefit to teeth comes from ingestion. (Fejerskov 2004). It is now universally recognized by dental researchers, however, that fluoride’s predominant benefit is topical not systemic. (NRC 2006, at 13; CDC 2001, at 4; Featherstone 2000). It is also now recognized that fluoride is not an essential nutrient. (NRC 1993, at 30; NRC 1989, at 235). Fluoride does not need to be swallowed, therefore, to prevent any disease, including tooth decay. By contrast, fluoride’s risks to health come from ingestion, including the spectrum of neurotoxic effects discussed below. Accordingly, a reasonable use of fluoride for caries prevention would aim to maximize its topical contact with teeth, while minimizing its ingestion. Topical fluoride products like toothpaste are compatible with this goal; fluoridating water supplies is not.

III. FLUORIDE IN DRINKING WATER: RECENT REGULATORY BACKGROUND

In 2003, the EPA asked the National Research Council (NRC) to review the scientific merits of EPA's Maximum Contaminant Level Goal (MCLG) for fluoride, which then and now is set at 4 mg/L. In response, the NRC reviewed the existing research on fluoride toxicity and concluded, in March 2006, that the MCLG is not protective of public health and should be lowered. (NRC 2006). The NRC's conclusion was based on fluoride's adverse effects on bone and teeth, but the NRC also raised numerous concerns about the potential for fluoride to cause other systemic harm, particularly to the nervous and endocrine systems.

With respect to the nervous system, the NRC concluded: “On the basis of information largely derived from histological, chemical, and molecular studies, it is apparent that fluorides have the ability to interfere with the functions of the brain.” (NRC 2006, at 222). The NRC's conclusion about fluoride's interference with the brain rested primarily on its review of animal studies, since—at the time of NRC's review—few human studies were available. The situation today, however, is much different as many studies linking fluoride exposure to cognitive deficits in humans have now been published. The number of human studies published subsequent to the NRC review that have found significant relationships between fluoride and adverse cognitive outcomes (n = 46) dwarfs the number of such studies that were available to the NRC (n = 5).1 The evidence linking fluoride to neurotoxicity in humans, therefore, is far more compelling today than it was when NRC published its review. Indeed, in 2014, fluoride was added to the list of chemicals “known to cause developmental neurotoxicity in human beings” in a review published by Lancet Neurology. (Grandjean & Landrigan 2014, at 334, Tbl 2). Only 12 chemicals are on this list.

It has been 10 years since the NRC concluded that the MCLG for fluoride be lowered, but the EPA has yet to do so. Further, despite the voluminous post-2006 research on neurotoxicity, and despite the Safe Drinking Water Act's mandate that EPA protect against “known or anticipated adverse effects,” EPA's Office of Water (EPA OW) has indicated that it will not be considering neurotoxicity as an endpoint of concern when promulgating the new MCLG.

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1 The 46 post-NRC human cognitive studies are cited in Appendix A. The five human cognitive studies that NRC cited are: Li et al. (1995); Zhao et al. (1996); Lu et al. (2000); Xiang et al. (2003a,b); and Qin et al. (1990).  
Specifically, in its December 2010 risk assessment of fluoride’s non-cancer effects, EPA OW established a reference dose for fluoride based solely on severe dental fluorosis, and declined to add an uncertainty factor to account for the neurotoxicity hazard. (EPA 2010, at 3 & 106). EPA OW justified this decision on the grounds that NRC’s 2006 review did not draw firm conclusions about the public health relevance of fluoride neurotoxicity. (EPA 2010, at 106). Nowhere in EPA OW’s risk assessment, however, did it account for the neurotoxicity research published subsequent to NRC’s review.

The cavalier manner in which EPA’s OW dismissed the evidence of fluoride neurotoxicity stands in stark contrast to EPA’s own Guidelines for Neurotoxicity Risk Assessment [hereafter Guidelines] that EPA has stated it “will follow in evaluating data on potential neurotoxicity associated with exposure to environmental toxicants.” (EPA 1998, at 1). The Fluoride Action Network submits that application of EPA’s Guidelines to the existing database for fluoride shows that neurotoxicity is a hazard of fluoride exposure, that the weight of evidence indicates neurotoxicity is a more sensitive endpoint of fluoride exposure than severe dental fluorosis, and, further, that the reference dose for fluoride that will protect the public and susceptible subpopulations against neurotoxicity is incompatible with the doses now ingested in fluoridated areas.

IV. FLUORIDE’S NEUROTOXICITY IS SUPPORTED BY OVER 180 STUDIES PUBLISHED SINCE NRC’S 2006 REVIEW

One of the striking features of the research on fluoride neurotoxicity is the large quantity of studies—animal, cellular, and human—that have reported an effect. In a recent review of developmental neurotoxins by EPA scientists, only 22% of suspected neurotoxins were found to have any supporting human data. (Mundy et al. 2015, at 25). The EPA team thus characterized chemicals, including fluoride, whose suspected neurotoxicity is backed by human data, as “gold standard” chemicals that warrant prioritization. (Mundy et al. 2015, at 27). In the case of fluoride, not only is there human data, the data is so extensive that fluoride has been classified alongside lead, mercury, and PCBs as one of only 12 chemicals “known to cause developmental neurotoxicity in human beings.” (Grandjean & Landrigan 2014, at 334, Tbl 2). The existence of so many human studies on fluoride neurotoxicity highlights the urgent need for a diligent risk assessment, per EPA’s Guidelines, to ensure that the general public, and sensitive subpopulations, are not ingesting neurotoxic levels.

Unlike EPA’s 2010 risk assessment, a diligent evaluation of fluoride’s neurotoxicity would consider the voluminous data that has been released since the NRC published its review in March 2006. Towards this end, attached is an exhaustive list of human, animal, and cell studies of fluoride’s neurotoxicity that have become available since NRC’s review.4

In total, we have identified 196 published studies that have addressed the neurotoxic effects of fluoride exposure subsequent to the NRC’s review, including 61 human studies, 115 animal studies, 17 cell studies, and 3 systematic reviews.

3 The Guidelines state that: “If data are considered sufficient for risk assessment, and if neurotoxicity is the effect occurring at the lowest dose level (i.e., the critical effect), an oral or dermal RfD or an inhalation RfC, based on neurotoxic effects, is then derived.” (EPA 1998, at 2)

4 Included among these studies are Chinese language studies that were originally published in Chinese journals prior to 2006 but were not translated and made available in the U.S. until after the NRC’s review. Excluded from these studies are those that are only available in abstract form, and animal/cell studies that have not yet been published and/or translated into English.
The post-NRC human studies include:

- 54 studies investigating fluoride’s effect on cognition, including but not limited to IQ, with all but 8 of these studies finding statistically significant associations between fluoride exposure and cognitive deficits. (Appendix A)
- 3 studies investigating fluoride’s effect on fetal brain, with each of the 3 studies reporting deleterious effects. (Appendix B)
- 4 studies investigating fluoride’s association with other forms of neurotoxic harm, including ADHD, altered neonatal behavior, and various neurological symptoms. (Appendix C)

The post-NRC animal studies include:

- 105 studies investigating fluoride’s ability to produce neuroanatomical and neurochemical changes, with all but 2 of the studies finding at least one detrimental effect in the fluoride-treated groups. (Appendix D)
- 31 studies investigating fluoride’s effect on learning and memory, with all but one of the studies finding at least one deleterious effect in the fluoride-treated groups. (Appendix E)
- 18 studies investigating fluoride’s impact on other parameters of neurobehavior besides learning and memory, with all but one of the studies finding effects. (Appendix F)

The post-NRC cell studies include:

- 17 studies, including 2 studies that investigated and found effects at fluoride levels that chronically occur in the blood of Americans living in fluoridated communities. (Appendix G)

In addition to the above studies, FAN is submitting three post-NRC systematic reviews of the literature, including two that address the human/IQ literature, and one that addresses the animal/cognition literature. (NTP 2016; Choi et al. 2012; Tang et al. 2008).

V. FLUORIDE POSES NEUROTOXIC RISKS AT LEVELS RELEVANT TO U.S. POPULATION

A frequent claim made by those who continue to promote fluoridation is that the doses of fluoride associated with neurotoxicity in humans and animals so vastly exceed the levels which Americans drinking fluoridated water receive as to be entirely irrelevant. In support of
this claim, proponents of fluoridation often point to the highest levels that have been linked to neurotoxicity, while ignoring the lowest levels (and even the typical levels) that have been associated with harm. This focus on the highest levels that cause harm as the starting point for analysis, rather than the lowest levels, clashes with standard tenets of risk assessment, including EPA’s Guidelines, where the starting point for risk characterization analysis is to determine the Lowest Observable Adverse Effect Level (LOAEL) or No Observable Adverse Effect Level (NOAEL).

A. Fluoride Repeatedly Linked to Reduced IQ at “Safe” Water Fluoride Levels

Contrary to the oft-repeated claim that fluoride neurotoxicity is only found at irrelevantly high doses, the existing studies of fluoride-exposed human populations have consistently found neurotoxic effects at water fluoride levels well below the current MCLG. To help clarify this issue, we examined the IQ studies that were included in the meta-review by Choi, et al. (2012). Proponents of fluoridation have dismissed the relevance of the Choi meta-review on the grounds that the IQ studies it included were in communities with fluoride levels that ranged as high as 11 ppm. As can be seen in the following table, however, the majority of waterborne fluoride studies (i.e., 13 of 18) that Choi reviewed included communities with fluoride levels below the 4 mg/L MCLG. Further, each of the 13 studies that investigated the effect of fluoride levels below 4 mg/L (average F = 2.3 mg/L) found these communities to have a lower average IQ than the control (average reduction = 6.3 IQ points), with the difference reaching statistical significance in 10 of the 13 studies.

<table>
<thead>
<tr>
<th>Study</th>
<th>Water F Level</th>
<th>IQ Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zhang et al. 1998</td>
<td>0.8 mg/L</td>
<td>-2.1 g</td>
</tr>
<tr>
<td>Lin et al. 1991</td>
<td>0.9 mg/L a</td>
<td>-7.0 a</td>
</tr>
<tr>
<td>Xu et al. 1994</td>
<td>2.0 mg/L a</td>
<td>-5.6 d</td>
</tr>
<tr>
<td>Yao et al. 1996</td>
<td>2.0 mg/L</td>
<td>-3.6 d</td>
</tr>
<tr>
<td>Yao et al. 1997</td>
<td>2.0 mg/L</td>
<td>-5.1 d</td>
</tr>
<tr>
<td>Pourleslami et al. 2011</td>
<td>2.4 mg/L</td>
<td>-6.4 a</td>
</tr>
<tr>
<td>Xiang et al. 2003</td>
<td>2.5 mg/L</td>
<td>-8.2 d</td>
</tr>
</tbody>
</table>

Another common misconception is that the endemic fluorosis/IQ studies prove the safety of fluoridated water because the control populations in these studies often have 0.7 to 1.0 mg/L fluoride in their water. Using areas with 0.7 to 1.0 mg/L as the control, however, says nothing about the safety of these levels since they are not compared against communities with lower fluoride levels.

As the Guidelines note, “Typically, estimates of the NOAEL/LOAEL are taken from the lowest part of the dose-response curve associated with impaired function or adverse effect.” (EPA 1998, at 58). Similarly, when the Benchmark Dose (BMD) approach is utilized instead of the NOAEL/LOAEL methods, EPA’s point of departure is the low end of the dose-response curve, not the high end.

We excluded any waterborne-fluoride exposure studies that did not report the water fluoride levels in the endemic fluorosis area(s). We excluded Li et al. (2010) because it did not compare a high fluoride community against a low-fluoride community, but simply looked at whether children with dental fluorosis in the high-fluoride community (2.5 mg/L) had lower IQ than children without dental fluorosis in the same community. We treated the Wang et al. 2001 and Yang et al. 1994 papers as a single study because it is apparent from the IQ data in the two papers that they are based on the same underlying IQ study. For the 18 qualifying studies, we reviewed the manuscripts to determine the lowest average fluoride concentration in each of the studies that was associated with reduced IQ. In studies with multiple exposure groups (e.g., Yao et al. 1996; Yao et al. 1997), we selected the lowest exposure group that had a reduction in IQ. For studies that only provide a range of fluoride levels for a given exposure group, we selected the midpoint in the range to represent the average fluoride concentration for the group.

As set forth in the accompanying table, one of the two studies that failed to find a statistically significant difference in average IQ (Wang et al. 2001) found an “obvious” increase in the rate of children with IQ scores lower than 80 (36.7% vs. 16.7%).

Seraj et al. 2006 2.5 mg/L -11.0b
An et al. 1992 2.7 mg/L -7.9f
Hong et al. 2001 2.9 mg/L 0 7.2d
Wang 2001/Yang 1994d 3.0 mg/L -5.0b
Lu et al. 2000 3.2 mg/L -10.9e
Fan et al. 2007 3.2 mg/L -2.3g
Zhao et al. 1996 4.1 mg/L -7.5c
Chen et al. 1991 4.6 mg/L -3.8d
Wang et al. 1996 4.8 mg/L -5.6a
Wang et al. 2006 5.5 mg/L -4.1d
Wang et al. 2007 8.3 mg/L -6.0a

a p<0.05; b p=0.025; c p<0.02; d p<0.01; e p<0.005; f Statistical significance not reported; g Not statistically significant; h Not statistically significant when analyzed in terms of average IQ, but “obvious” difference seen when analyzed in terms of percentage with low IQ; Omega High-fluoride + low-iodine versus low-fluoride + low-iodine; ¶ These two papers appear to be the same study.

B. Fluoride Linked to Cognitive Deficits at Levels of Individual Exposure Seen in Western Fluoridated Populations

Although the water fluoride levels associated with IQ reductions are modestly higher than the levels currently used in artificially water fluoridation programs, it is important to distinguish between the concentration of fluoride in a community’s water supply and the dose of fluoride that an individual ingests. For example, in rural China (where most of the IQ studies have been conducted), fluoridated toothpaste is rarely used, with less than 10% of children using any fluoride toothpaste at all.12 By contrast, in the United States, over 95% of toothpastes are fluoridated and research shows that toothpaste can contribute more fluoride to a child’s daily intake than fluoridated water. (CDC 2013c; Zohoori et al. 2013, Zohoori et al. 2012; Levy et al. 1999). As noted by a review in the Journal of Public Health Dentistry, “Virtually all authors have noted that some children could ingest more fluoride from dentrifice alone than is recommended as a total daily fluoride ingestion.”13 (Levy and Guda-Chowdhury 1999, at

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11 The 23 studies include the 10 studies listed in Table 1, the 11 studies listed in the paragraph above, and the studies by Eswar et al. 2011 and Shivaprakash et al. 2011.

12 According to a 1996 national oral health survey in China, 75% of 12-year-old children use toothpaste, and of the children who use toothpaste, only 11% use fluoride-containing varieties. (Zhu et al. 2003, at 291, Tbl 1.)

13 FAN recognizes that the FDA has jurisdiction over fluoride toothpaste, but any assessment of the safe level of a contaminant in drinking water cannot be conducted in a vacuum, and must consider the additive effect of waterborne exposures with identifiable non-water sources of exposure. When considering the neurologic safety of fluoridated water, therefore, it is critical to consider the aggregate dose of fluoride in fluoridated communities from all sources, including toothpaste. EPA has recognized this principle in its “relative source contribution” Submission by the Fluoride Action Network on EPA’s Proposed Rule for a 6-Year-Review of Fluoride under the National Primary Drinking Water Regulations. Docket No. EPA-HQ-OW-2016-0627, March 13, 2017.
The abundance of fluoridated toothpaste in the U.S., versus its relative scarcity in rural China, will therefore lessen the difference in total daily fluoride intake between these populations. In fact, as set forth below, available evidence suggests that the (i) daily fluoride doses, (ii) urine fluoride levels, (iii) serum fluoride levels, and (iv) dental fluorosis levels associated with IQ reductions in the Chinese studies are seen in children and adults in western countries living in fluoridated areas. Each of these four metrics of fluoride exposure provide a more direct assessment of individual fluoride exposure than water fluoride concentration, and are thus more probative for risk assessment purposes.

(i) Daily Fluoride Intake

The overlap between the daily fluoride intake associated with significant IQ loss in China and the daily doses American children now receive is highlighted by the recent studies from Wang et al. (2012) and Das et al. (2016). In the study by Wang, researchers investigated the impact of total daily intake of fluoride on IQ among the same group of 512 rural Chinese 8-to-13 year old children studied by the Xiang team in 2003. (Xiang et al. 2003a,b). As the following table shows, the Wang study found a clear dose response relationship between daily fluoride dose and reduced IQ.

**FIGURE 1: Relationship Between Daily Fluoride Dose and IQ**  
*(SOURCE: Wang et al. 2012, Tbl. 4)*

Wang found that a daily intake of just 2.61 mg F/day was associated with a large, statistically significant 7.28-point drop in average IQ. Assuming an average weight of 32 kg, a daily intake of 2.61 mg would provide a dosage of approximately **0.08 mg/kg/day**, which is lower analyses, which the EPA OW conducts when calculating the drinking water equivalent level (DWEL) of a reference dose. EPA (2016). TSCA also specifically contemplates consideration of aggregate and sentinel exposures in Section 6 risk evaluations. See 15 U.S.C. § 2605(b)(4)(F).

14 The authors did not provide data on the average weight of the children in the study, and we could not find data on the average weight of rural Chinese children between the ages of 8 and 13. We did, however, find published data on the weight of rural Chinese children ages 0 to 7, as well as average weight data on U.S. children between the ages of 2 and 20. (Li et al. 2011; CDC 2000a,b) A comparison of these two datasets shows that rural Chinese children weigh approximately 4 kg less than U.S. children (18.7 kg vs. 23 kg) between the ages of 6 and 7. We thus determined the average weight of 8-to-13 year old rural Chinese children by calculating the average weight of 8-to-13 year old U.S. children from the CDC growth charts (=36 kg) and subtracting 4 kg (=32 kg).

15 It bears noting that 0.08 mg/kg/day is EPA’s new reference dose for fluoride, which the Agency established to protect solely against severe dental fluorosis (without the protection of a single uncertainty factor to account for potential neurotoxic risks). (EPA 2010)
than the average daily intake (0.087 mg/kg/day) for non-nursing infants in the United States, as estimated by the NRC, and just two times greater than the average daily dose for 8-12 year old American children.\textsuperscript{16} (NRC 2006, at 65, Tbl. 2-13). Moreover, recent research has found that 10 to 15% of children under the age of 6 ingest over 0.05 mg/kg/day from toothpaste alone, with some children ingesting as much as 0.159 mg/kg/day from this single source. (Strittholt et al. 2016 at 70 tbl. 2; Zohoori et al. 2012 at 418 tbl 2; Zohoori et al. 2013 at 460 tbl 1; Levy & Guha-Chowdhury 1999 at 217 tbl 3). In one study, published by Procter & Gamble scientists (Strittholt et al. 2016), 5% of pre-schoolers were found to ingest at least 0.49 mg fluoride per brushing, which, at two brushings per day, will produce a daily dosage of 0.07 mg/kg/day from toothpaste alone for the average-weighing 2-year-old. (CDC 2000a,b). Other studies are consistent with these estimates. (Oliveria et al. 2007; Bentley et al. 1999; Levy 1993; Naccahe et al. 1992). For the many pre-school children ingesting these dosages from toothpaste, the consumption of fluoridated water will readily push them over the daily dosage (0.08 mg/kg/day) associated with sharp reductions in IQ among rural Chinese children.

Finally, as with other forms of fluoride toxicity, the potential for fluoride neurotoxicity is magnified among children with suboptimal nutrient intake. (Sun et al. 2016; Ge et al. 2011; Hong et al. 2008; Ge et al. 2005; Wang et al. 2004; Ekambaram & Paul 2002; Xu et al. 1994; Lin et al. 1991; Ren et al. 1989; Guan et al. 1988). This is highlighted by the recent study by Das and Mondal which assessed the relationship between fluoride intake and IQ among a population with a high prevalence of underweight children suggestive of an area with pervasive malnutrition. In this population, Das and Mondal confirmed a significant correlation between total fluoride intake and reduced IQ ($r = -0.343$, $p < 0.01$), as plotted in the following figure:

**FIGURE 2: Relationship Between Total Daily Intake and IQ**

*(SOURCE: Das & Mondal 2016, Fig. 6)*

Notably, Das and Mondal found a sharp 15-point drop in IQ among underweight children with mild dental fluorosis who were consuming average total daily fluoride exposures of just 0.06 mg/kg/day. (Das & Mondal 2016, at 218, Tbl. 3). As discussed above, this is a dose that many infants and children in the U.S. are estimated to exceed.

\textsuperscript{16} A recent national analysis of urinary fluoride levels in the United Kingdom UK concluded that over 65% of adults living in fluoridated areas consume more than 0.057 mg/kg/day. (Mansfield 2010)
Many of the studies on fluoride and IQ have measured the concentration of fluoride in children’s urine as a marker of individual fluoride exposure. As summarized in a 2011 review, these studies have repeatedly found significant, often large reductions in IQ when the average urinary fluoride level exceeds 2.5 mg/L, (Spittle 2011), and multiple regression analyses have repeatedly found that increased urinary fluoride correlates with reduced IQ, (Das et al. 2016; Zhang S. et al. 2015; Wang et al. 2007), even when controlling for other key risk factors. (Rocha Amador et al. 2009). While urinary fluoride levels exceeding 2.5 mg/L present a clear risk for neurotoxicity, recent studies have also found decrements in IQ at urinary fluoride concentrations well below this level. Most notable in this regard is the study by Ding et al., which examined the correlation between urinary fluoride and IQ among children with urinary fluoride levels ranging from just 0.25 mg/L to 3 mg/L. As shown in the following figure, a clear dose response trend was found within this urinary fluoride range ($p < 0.0001$), with the downward trend becoming apparent at roughly 1 mg/L. When adjusted for age, each 1 mg/L increment in urinary fluoride correlated with an average drop of 0.59 IQ points ($p < 0.0001$).

![FIGURE 3: Relationship Between Urinary Fluoride and IQ](SOURCE: Ding et al. 2011, Fig. 2)

The dose-response trend found by Ding is consistent with more recent data published by Zhang et al. 2015, which is displayed in the following figure. As can be seen, the Zhang study found a clear drop in IQ at urinary fluoride levels between 0.5 and 1.5 mg/L.

![FIGURE 4: Relationship Between Urinary Fluoride and IQ](SOURCE: Zhang S. et al. 2015, Fig. 1)
More recently, researchers have investigated the prevalence of cognitive impairment among elderly individuals living in an endemic fluorosis region of China. (Li et al. 2016). The researchers found a very high prevalence of cognitive impairment (81.2%) in the fluorosis region, and, in a case-control analysis, found a significantly elevated urinary fluoride level (2.5 mg/L vs. 1.5 mg/L, p < 0.05) in the cognitive impairment group. (Li et al. 2016, at 57, Tbl. 3). The data from this case-control analysis is presented in the following table:

TABLE 2: Urinary Fluoride & Cognitive Impairment in Elderly
(SOURCE: Li et al. 2016, Tbl 3 )

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Normal group (n=38)</th>
<th>Cognitive impairment group (n=38)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male/female</td>
<td>26/12</td>
<td>26/12</td>
<td>0.920</td>
</tr>
<tr>
<td>Age (years)</td>
<td>64.95±4.60</td>
<td>65.05±4.40</td>
<td>0.000</td>
</tr>
<tr>
<td>MMSE score</td>
<td>27.79±0.96</td>
<td>21.50±4.37</td>
<td>0.028</td>
</tr>
<tr>
<td>Total daily water fluoride intake (mg)</td>
<td>2.23±2.23</td>
<td>3.62±6.71</td>
<td>0.128</td>
</tr>
<tr>
<td>Urinary fluoride (mg/L)</td>
<td>1.46±1.04</td>
<td>2.47±2.88</td>
<td>0.046</td>
</tr>
<tr>
<td>Fluorosis score</td>
<td>0.74±0.98</td>
<td>1.29±1.01</td>
<td>0.018</td>
</tr>
<tr>
<td>Serum Hcy (μmol/L)</td>
<td>19.97±8.88</td>
<td>20.14±9.29</td>
<td>0.934</td>
</tr>
</tbody>
</table>

a Values are n/n for gender and mean±SD for other indices.
b The original values were log-transformed before comparison. The difference between two groups was tested using Student's t test.

Although there is a paucity of published data on urinary fluoride levels in the United States, a study from England found that the average urinary fluoride level among 88 adults living in a fluoridated area was 1.28 mg/L, with 16% of the tested individuals having over 2 mg/L, and 6% of individuals having over 3 mg/L. (Mansfield 1999, at 28, Tbl. 1). These levels overlap those that have been associated in endemic fluorosis areas with both reduced IQ in children and cognitive impairment in the elderly. (Li et al. 2016; Zhang S. et al. 2015; Ding et al. 2011).

17 A clear dose-response relationship between urinary fluoride and cognitive impairment was not detected in the non-case control component of Li et al.’s analysis, although urinary fluoride was found to be elevated in the population with severe cognitive impairment.

18 These urinary fluoride levels exceeded those that were found among individuals (n = 165) living in non-fluoridated areas. The average urinary fluoride level in the non-fluoridated areas was 0.96 mg/L; with 8% having more than 2 mg/L; and 4% having more than 3 mg/L. (Mansfield 1999, at 28, Tbl. 1)
A more recent study from Canada found that 5 percent of children had > 1.3 mg/L fluoride in their urine, which is well within the range of urinary fluoride levels associated with reduced IQ in the Ding and Zhang studies. (Saravanabhavan et al. 2016). A separate Canadian study found that the average urinary fluoride concentration in fluoridated areas was 0.76 mg/L, which was almost twice the concentration (0.47 mg/L) found in non-fluoridated areas. (McLaren 2016).

(iii) Serum Fluoride Level

In 2011, Xiang et al. published a paper which assessed the relationship between IQ and serum fluoride levels in the same group of 512 children studied in Wang’s daily dose analysis discussed above. As with the daily dose analysis, the authors found a significant dose-response relationship between serum fluoride level and reduced IQ. As shown in the following table, children with just 0.05 to 0.08 mg/L fluoride in their serum had a statistically significant 4.2-point drop in IQ when compared against children with less than 0.05 mg/L.\(^\text{19}\)

\[
\begin{array}{|c|c|c|c|c|c|}
\hline
\text{Serum fluoride level quantiles} & \text{N} & \text{Mean IQ} & \text{SD IQ} & \text{p}\(^b\) & \text{OR (95% CI) for IQ<80} \\
\hline
\text{Q1 and Q2} & 259 & 100.1 & 13.4 & <0.001 & 7.0 & 1 \\
(<0.05 \text{ mg/L}) & & & & & & \\
\text{Q3} & 126 & 95.9 & 13.7 & 0.004 & 2.22 (1.42–3.47) \\
(0.05–0.08 \text{ mg/L}) & & & & & & \\
\text{Q4} & 127 & 92.1 & 13.4 & 2.48 (1.85–3.32) & p \text{ trend<0.001}\(^d\) \\
(>0.08 \text{ mg/L}) & & & & & & \\
\hline
\end{array}
\]

\(^a\)Adjusted for age and gender using Logistic regression analysis. The data from two villages were combined.

\(^b\)NOVA.

\(^c\)Chi-square test.

\(^d\)Tests of linear trend were computed using ordinal scoring.

Abbreviations: CI Confidence Interval, IQ Intelligence Quotient, OR Odds Ratio, SD Standard Deviation.

The Xiang team’s findings are consistent with the findings of other recent studies, including Guo Z. et al. (2008), which found impairment in neurobehavioral function among adult industrial workers with average serum fluoride levels of 0.066 mg/L, and Zhang S. et al. (2015), which found significant reductions in IQ among children with just over 0.05 mg/L fluoride in their blood when compared to children with the lowest levels. The Zhang study plotted the serum data in the following figure:

**FIGURE 5: Relationship Between Serum Fluoride and IQ**

(SOURCE: Zhang S. et al. 2015, Fig. 1)

\(^{19}\) As the authors emphasize, their finding of a 4-point IQ drop in children with more than 0.05 mg/L fluoride in their serum does not mean that serum levels lower than 0.05 mg/L are safe.
To put these serum fluoride levels in the context of U.S. exposures, typical serum fluoride levels for adults in the U.S. have been stated to range from about 0.01 to 0.076 mg/L (0.5 to 4 μM/L). (CDC 2014, at 2; see also Kissa 1987). In one study of infants, an average concentration of 0.08 mg/L was found among healthy 4-to-6 month old infants, while an average concentration of 0.10 to 0.18 mg/L was found among 4-to-18 month old infants receiving peritoneal dialysis. (Warady et al. 1989). A study by Ekstrand found that infants ingesting 0.25 mg in supplement form have spikes in their blood ranging as high as 0.092 mg/L, and averaging 0.063 mg/L. (Ekstrand 1994, at 159 tbl 3). Ekstrand’s study did not measure the impact of ingesting fluoride in the form of infant formula reconstituted with fluoridated water, but the resulting daily peaks in serum fluoride levels may be comparable, since Ekstrand estimates that infants consuming fluoridated formula receive doses (up to five times a day) that are comparable to a supplement (i.e., 20-30 ug/kg of fluoride per formula feeding vs. 32 ug/kg per supplement). (Ekstrand 1994, at 162).

While there has long been a paucity of serum fluoride data available for children in the U.S., a recent NHANES survey found that roughly 1 in 200 American children between the ages of 3 to 19 have serum fluoride levels exceeding 0.04 mg/L. (NHANES 2016). Since there are approximately 70 million American children in this age range, (US Census Bureau 2011), the NHANES data indicates that approximately 350,000 American children have serum fluoride levels in the approximate range associated with overt neurotoxic effects.

(iv) Dental Fluorosis Level

EPA OW’s 2010 risk assessment of the non-cancer effects of fluoride rests on the implicit assumption that severe dental fluorosis is the most sensitive adverse endpoint of fluoride exposure. This assumption, however, is at odds with a number of studies which have found significant associations between fluoride exposure and cognitive deficits among children with non-severe forms of fluorosis. Most notably, the study by Ding et al. (2011) found a dose-dependent relationship between reduced IQ and urinary fluoride concentration in a population where severe dental fluorosis was completely absent. The Ding study thus suggests that the doses of fluoride that impair cognitive ability are lower than the doses that cause severe fluorosis. Other recent studies have found impairment in cognitive abilities among children with mild fluorosis, moderate fluorosis, and moderate/severe fluorosis when compared with children with no fluorosis, thus suggesting that the doses of fluoride associated with the milder forms of fluorosis are sufficient to impair brain development.20 (Das & Mondal 2016 at tbl 3;

20 Some studies, however, including Ding, have not found a clear relationship between IQ and dental fluorosis status, thus suggesting that a person’s susceptibility to fluoride-induced neurotoxicity may be distinct from their susceptibility to dental fluorosis. (Asawa et al. 2014; Li et al. 2010)
Consistent with the above studies of human populations, studies of rodents have repeatedly found significant impairments in learning ability as well as other neurotoxic harms among rats with only mild forms of fluorosis.\textsuperscript{21} (Liu et al. 2011; Pereira et al. 2011; Niu et al. 2008; Chioca et al. 2008). As noted by Niu et al., “these findings indicate that fluoride . . . can influence spontaneous behaviors and lower the learning ability of rats before the appearance of dental lesions.”\textsuperscript{22} (Angmar-Mansson & Whitford 1982).

Taken together, the available human and animal studies suggest that fluoride can impair cognitive abilities prior to the development of severe fluorosis. This has obvious public health relevance in the United States, since recent studies show that the prevalence of dental fluorosis is now at historically unprecedented levels. In CDC’s 1999-2004 NHANES survey, for example, 41% of adolescents were diagnosed with dental fluorosis, including 8.6% with mild fluorosis, and 4% with moderate and severe. These rates are considerably higher than what was found in the 1986-87 national survey by the National Institute of Dental Research. (Beltran et al. 2010; Heller et al. 1997). Moreover, the rates appear to have increased yet further since the 1999-2004 NHANES survey. Specifically, the 2011-2012 NHANES survey found dental fluorosis in 58.3% of the surveyed adolescents, including an astonishing 21.2% with moderate fluorosis, and 2% with severe. (NHANES 2014). Since there are an estimated 42 million adolescents currently living in the U.S.,\textsuperscript{23} the NHANES data suggests that up to 24 million adolescents now have some form of dental fluorosis, with over 8 million adolescents having moderate fluorosis, and 840,000 having severe fluorosis.

The NHANES surveys do not provide data on the respective rates of fluorosis in fluoridated vs. non-fluoridated communities, but research has repeatedly confirmed that both the prevalence and severity of dental fluorosis are greater in U.S. communities with fluoridated water than in communities without. (Heller et al. 1997; Jackson et al. 1995; Williams & Zwemer 1990). Ending fluoridation will thus reduce the number of children developing dental fluorosis, and the accompanying neurotoxic risks associated with the doses that produce fluorosis.\textsuperscript{24}

\textsuperscript{21} Consistent with this, Zhou Z. et al. (2016) recently reported that biochemical changes occur in rats at doses well below those that cause dental fluorosis.

\textsuperscript{22} While rodent teeth undergo constant remodeling, thus distinguishing them from human teeth, research has found that rat teeth develop dental fluorosis at the same serum fluoride levels that produce fluorosis in humans. According to Angmar-Mansson & Whitford, “It is well known that, in fluoridated drinking water studies with rats, a water fluoride concentration of 10-25 ppm is necessary to produce minimal disturbances in enamel mineralization. Because of the high water concentrations required, the rat has been regarded as more resistant to this adverse effect of fluoride. However, when the associated plasma levels are considered, the rat and the human appear to develop enamel fluorosis at very nearly the same concentrations.” (Angmar-Mansson & Whitford 1982, at 339) Based on this finding, Angmar-Mansson & Whitford concluded that “the rat is a better model for the study of human enamel fluorosis than previously believed.” (Id. at 334)

\textsuperscript{23} This estimate is based on the number of Americans between the ages of 10 and 19. It comes from the Office of Adolescent Health, which is part of the Department of Health & Human Services. (DHHS 2016).

\textsuperscript{24} Decreases in dental fluorosis have been documented following temporary suspensions of fluoridation as short as 11 months. (Burt et al. 2000)
VI. NEUROTOXIC RISK OF LOW DOSE FLUORIDE IS FURTHER SUPPORTED BY ANIMAL AND CELL STUDIES

The studies linking fluoride exposure with neurotoxic effects in humans are consistent with research on both experimental animals and cell cultures. Studies on rodents, for example, have found neurotoxic effects, including learning impairments, at water fluoride levels less than 15 mg/L, with 8 studies published since the NRC review reporting neurotoxic effects at water fluoride levels less than 5 mg/L. These are notably low fluoride levels for rodents, since it is generally estimated that rats require approximately 5 times more fluoride in their water to achieve the same level of fluoride in their blood as humans, and over 10% of children living in fluoridated areas receive the same waterborne dosage of fluoride (mg/kg/day) as rats drinking water with up to 9 mg F/L. (NTP 2016, at 56-57)

The following table lists the water fluoride concentrations associated with neurotoxic effects in rodents:

<table>
<thead>
<tr>
<th>Study</th>
<th>F Concentration (F⁻)</th>
<th>Duration of Treatment</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chouhan (2010)</td>
<td>1 mg/L</td>
<td>4 months</td>
<td>Oxidative stress; alterations in neurotransmitters</td>
</tr>
<tr>
<td>Wu (2008)</td>
<td>1 mg/L</td>
<td>Gestation</td>
<td>Behavioral alterations</td>
</tr>
<tr>
<td>Gao (2009)</td>
<td>2.3 mg/L</td>
<td>6 months</td>
<td>Enzyme inhibition; impaired cognition; oxidative stress</td>
</tr>
<tr>
<td>Liu (2014)</td>
<td>2.3 mg/L</td>
<td>1 month</td>
<td>Impaired learning</td>
</tr>
<tr>
<td>Liu (2010)</td>
<td>2.3 mg/L</td>
<td>6 months</td>
<td>Impaired cognition; alterations in neurotransmitters</td>
</tr>
<tr>
<td>Sandeep (2013)</td>
<td>2.3 mg/L</td>
<td>3 months</td>
<td>Behavioral alterations; enzyme inhibition</td>
</tr>
<tr>
<td>Zhang (2015)</td>
<td>2.3 mg/L</td>
<td>6 months</td>
<td>Oxidative stress; activation of AGE/RAGE system</td>
</tr>
<tr>
<td>Zhang Z. (2008)</td>
<td>4.5 mg/L</td>
<td>10 weeks</td>
<td>Impaired learning; pathological changes in synaptic structure</td>
</tr>
<tr>
<td>Zhu (2011);</td>
<td>6.8 mg/L</td>
<td>9 months</td>
<td>Trend towards decreased synaptic membrane fluidity &amp; PSD-95 expression level; altered expression of CaMKIIα, c-fos, Bax, and Bcl-2 (statistically significant at 13.6 mg/L)</td>
</tr>
<tr>
<td>Zhang (2011);</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zhang J. (2013)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bhatnagar (2011)</td>
<td>8 mg/L</td>
<td>1 month</td>
<td>Morphological changes in neurons</td>
</tr>
<tr>
<td>Banala (2015)</td>
<td>9 mg/L</td>
<td>Gestation + 30 days postnatal</td>
<td>Impaired learning; loss of motor control; &amp; oxidative stress</td>
</tr>
<tr>
<td>Reddy (2014)</td>
<td>9 mg/L</td>
<td>3 months</td>
<td>Alterations in neurotransmitters; altered immunological parameters; oxidative stress</td>
</tr>
</tbody>
</table>
Fluoride’s ability to cause neurotoxic effects at low levels of exposure is further corroborated by in vitro cell studies conducted subsequent to the NRC review. While most of the in vitro studies used high levels of fluoride (≥10 mg/L), two of the studies investigated the effects of concentrations that are found in the bloodstream of many Americans. Both of these low-concentration studies detected adverse effects. As displayed in the following figure, Gao et al. (2008) found that just 0.5 μM of fluoride (i.e., 0.009 mg/L) caused lipid peroxidation in SH-SY5Y cells after 48 hours of exposure. Most individuals living in fluoridated areas in the United States have fluoride levels in their blood that exceed this level. (CDC 2014; Kissa 1987).

The Gao study also found that 0.5 μM had an effect on the level of a7 nAChR protein in the SH-SY5Y cells, as displayed in the following figure:

Consistent with the findings of these two brain cell studies, the in vitro studies by Gutowska have repeatedly found that concentrations of just 1 to 3 μM (i.e., 0.019 to 0.057 mg/L) are sufficient to affect inflammatory responses. (Gutowska et al. 2015, 2012, 2010). The Gutowska team’s findings underscore the biologically active nature of even micromolar concentrations of fluoride, and warrant consideration for their implications to neuroinflammation. (Louveau et al. 2011).
Flores-Mendez et al. (2014) also investigated the effect of 0.5 uM, and, per the following figure, found a suggestive trend towards an increase in eEF2 phosphorylation in cultured Bergmann glia cells (BGC) after 15 minutes of treatment.

**FIGURE 8: eEF2 Phosphorylation in BGC Cultures Treated with Fluoride**

(Source: Flores-Mendez et al. 2014, Fig. 4b)

Flores-Mendez also found a suggestive trend towards an increased influx of calcium into the cell after 3 minutes of treatment with 5 uM fluoride (i.e., 0.095 mg/L). (Flores-Mendez et al. 2014, at 130 Fig. 5c) This concentration can be found chronically in the blood of children with kidney disease living in fluoridated areas, (Warady et al. 1989), and is intermittently exceeded by children ingesting fluoride supplements, fluoridated toothpaste, and other dental products.26

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26 While there is a paucity of research on the serum fluoride levels following use of fluoride tablets and toothpaste, Ekstrand found that, among a group of 5 preschool children, ingestion of 0.5 mg fluoride tablets caused serum Submission by the Fluoride Action Network on EPA’s Proposed Rule for a 6-Year-Review of Fluoride under the National Primary Drinking Water Regulations. Docket No. EPA-HQ-OW-2016-0627, March 13, 2017.
VII. RECENT EPIDEMIOLOGICAL STUDIES CORROBORATE NEUROTOXIC RISK FROM FLUORIDATED WATER IN WESTERN POPULATIONS

The overlap between the internal doses of fluoride experienced in western populations and the internal doses associated with neurotoxic effects in humans, animals, and cell cultures, is cause for public health concern. Although there has been a notable lack of epidemiological research into fluoride's neurotoxic effects in the U.S., a 2015 study by Malin and Till found a statistically significant correlation between the prevalence of water fluoridation at the state level and Attention-Deficit Hyperactivity Disorder (ADHD). Fluoridation prevalence significantly correlated with ADHD even after controlling for socioeconomic status (SES), and fluoridation “appeared to be the more robust predictor.” As Malin and Till note, their findings “are consistent with prior epidemiological studies that have associated high and low fluoride concentration exposure with neurodevelopmental effects in children.”

Another epidemiological study from 2015, by Peckham et al., provides further corroborative evidence that fluoridation can cause neurotoxic effects. Peckham’s study examined the relationship between water fluoride levels and hypothyroidism in the United Kingdom, and found that fluoride levels ≥ 0.7 mg/L significantly correlated with higher rates of hypothyroidism. This correlation was strengthened, not weakened, when controlling for the covariates of age, gender, and index of deprivation.

The correlation between fluoridation and hypothyroidism reported by Peckham is (i) plausible and (ii) adds further support for the capacity of fluoridated water to cause neurotoxic effects. First, the correlation is plausible because, as summarized by the NRC, multiple lines of research indicate that fluoride can lower thyroid function, including the fact that fluoride was once used as a drug for this precise purpose, at doses as low as 2 to 5 mg/day. (NRC 2006; Galletti & Joyet 1958). Second, the correlation between fluoridation and hypothyroidism adds further support for fluoridation’s neurotoxic potential because, as recognized in EPA’s Guidelines, “the development of the nervous system is intimately associated with the presence of circulating hormones such as thyroid hormone.” (EPA 1998, at 50). Since both clinical and subclinical hypothyroidism during pregnancy have been associated with reduced IQ in offspring, (Korevaar et al. 2016; Murphy et al. 2015; Klein et al. 2001), the relationship between fluoridation and hypothyroidism provides a mechanism by which fluoridation can reduce IQ, even absent a direct neurotoxic effect.

VIII. SUSCEPTIBLE SUBPOPULATIONS ARE AT HEIGHTENED RISK OF FLUORIDE NEUROTOXICITY AND NEED PROTECTION

EPA’s Guidelines recognize that individual susceptibility to the neurotoxicity of environmental toxicants can vary by a factor of ten or more, and is influenced by factors such as nutritional status, age, genetics, and disease. (EPA 1998, at 63-65, 78). Each of these factors—

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27 “In general, it is assumed that an uncertainty factor of 10 for intrapopulation variability will be able to accommodate differences in sensitivity among various subpopulations, including children and the elderly. However, in cases where it can be demonstrated that a factor of 10 does not afford adequate protection, another uncertainty factor may be considered in conducting the risk assessment.” (EPA 1998, at 65)

nutritional status, age, genetics, and disease—are known to influence an individual's susceptibility to chronic fluoride toxicity. Any factor that can predispose an individual to chronic fluoride toxicity should be suspected as a factor that will predispose to fluoride neurotoxicity as well. In fact, recent research in both humans and animals has specifically demonstrated that nutrient deficiencies (i.e., iodine and calcium) amplify fluoride's neurotoxicity. Further, Zhang S. et al. (2015) reported that certain COMT gene polymorphism greatly influences the extent of IQ loss resulting from fluoride exposure, which is consistent with research on other neurotoxins, including methyl mercury. (Julvez & Grandjean 2013).

While the full range of individual susceptibility to fluoride neurotoxicity in the U.S. cannot be precisely calculated, some subpopulations can be identified as being at elevated risk, including infants, the elderly, and individuals with deficient nutrient intake (particularly

28 Studies have repeatedly confirmed that genetic factors can significantly increase susceptibility to fluoride toxicity, (Everett 2011), including effects on bone (Kobayashi et al. 2014; Yan et al. 2007; Mousny et al. 2006); teeth (Buzalaf et al. 2014; Ba et al. 2011; Huang et al. 2008; Everett et al. 2002); and reproductive hormones (Zhou et al. 2016).
29 See, e.g., Irigoyn-Camacho ME et al. (2016); Simon et al. (2014); Ravula et al. (2012); Itai et al. (2010); Schiff (2008); NRC (2006); Teotia et al. (1998); Torra et al. (1998); Warady et al. (1989); and Turner et al. (1995). For additional citations and discussion, see http://www.fluoridealert.org/studies/skeletal_fluorosis03.
30 See, e.g., Ge et al. (2011); Hong et al. (2008); Ge et al. (2005); Wang et al. (2004); Xu et al. (1994); Lin et al. (1991); Ren et al. (1989); Guan et al. (1988).
31 Sun et al. (2016); Ekambaram & Paul (2002).
32 As discussed earlier, the study by Das & Mondal (2016) examined the impact of fluoride on IQ in a population with a high prevalence of underweight children, suggestive of an area with chronic malnutrition. In this population, a daily fluoride dose of just 0.06 mg/kg/day was associated with a sharp 15-point drop in IQ among children with mild fluorosis. (Das & Mondal 2016, at 218, Tbl. 3).
33 Although breast fed infants receive the lowest fluoride intake by bodyweight (<0.001 mg/kg/day) of all age-groups (Ekstrand et al. 1981), this situation is flipped on its head when infants are fed formula reconstituted with fluoridated water. As noted by the NRC, “On a per-body-weight basis, infants and young children have approximately three to four times greater exposure than do adults.” (NRC 2006, at 3). Not only do formula-fed infants receive an unnaturally high dose, they have an impaired ability to excrete the fluoride they ingest, retaining up to 87% of the absorbed dose. Ekstrand et al. (1994). Infants exposed to formula made with fluoridated water are at significantly higher risk for developing dental fluorosis on their permanent front teeth. Hong et al. (2006). In light of the research linking dental fluorosis and modest levels of fluoride exposure with reduced IQ, infants are a susceptible subpopulation of critical concern for fluoride neurotoxicity.
34 As noted in the Guidelines, “[T]he aged population is considered to be at particular risk [of neurotoxicity] because of the limited ability of the nervous system to regenerate or compensate to neurotoxic insult.” (EPA 1998, at 65). This is of concern because the brain will be more exposed to fluoride in older age due to the (1) increased level of fluoride circulating in the serum from both age-related decreases in renal function and age-related increases in bone resorption (particularly in post-menopausal women), and (2) increased permeability of the blood-brain barrier. Rosenberg (2014); Ravula et al. (2012); Itai et al. (2010); Torra et al. (1998). This may help explain the very high prevalence of cognitive impairment (82%) found among elderly individuals in an endemic fluorosis area. Li et al. (2016); see also Shao et al. (2003).
iodine and calcium), (B) certain COMT gene polymorphisms, and (C) kidney disease. Various factors suggest that African Americans may also suffer disproportionate risks as well, including elevated use of infant formula, elevated exposure to lead, depressed calcium and anti-oxidant intake, and significantly higher rates of dental fluorosis, including in its moderate and severe forms.

Any risk assessment on the neurotoxicity of fluoride must thus be mindful of the need to protect susceptible subpopulations; anything less would be inconsistent with EPA’s Guidelines. In fact, even where there is no specific information to indicate differential susceptibility to a neurotoxin, EPA’s Guidelines state that a margin of safety (i.e., “uncertainty factor”) should still be incorporated to account for “potential differences in susceptibility.” (EPA 1998, at 78). In the case of fluoride, there is uncontroverted evidence indicating substantial differences in susceptibility, and thus the basis for applying an uncertainty factor is especially strong.

IX. A REFERENCE DOSE PROTECTIVE AGAINST FLUORIDE NEUROTOXICITY IS INCOMPATIBLE WITH WATER FLUORIDATION IF STANDARD RISK ASSESSMENT PROCEDURES ARE APPLIED

35 According to a consensus paper in the Journal of the National Medical Association, “Eighty-six percent of African Americans get just more than half of the daily recommended amount of calcium, and only half consume one or more servings of dairy a day. Of particular concern, 83% of African-American children 2-17 years of age are not getting enough calcium.” Wooten & Price (2004). Insufficient nutrient intakes in the United States are severe enough in some individuals to qualify as nutrient deficiencies. Recent NHANES data, for example, found that 6% of Americans have a vitamin C deficiency. CDC (2012). Vitamin C deficiency has been found to exacerbate fluoride’s toxicity in humans, while vitamin C supplementation has been found to ameliorate fluoride’s neurotoxic effects in animals. Nabavi et al. (2013; Basha & Madhusudhan (2010); Pandit et al. (1940). With respect to iodine, NHANES data shows that women of child bearing age (20 to 39 years old) have "median urine iodine concentrations bordering on insufficiency." Pfeiffer et al. (2013). Children born to women with insufficient iodine levels should be considered a susceptible subpopulation for fluoride neurotoxicity due to fluoride’s ability to exacerbate the neurological effects of inadequate iodine.

36 The study by Zhang S. et al. (2015) suggests that children with the COMT val/val genotype suffered a five-fold larger drop in IQ than children with the COMT val/met and met/met genotypes. As noted by Zhang, “In the subpopulation carrying the COMT reference genotype (Model 3), 1 unit increase in urinary fluoride (1 mg/l) was associated with a decrease of 9.67 points of IQ and was significant after controlling for covariates (P=0.003). Among children carrying variant genotypes, 1 unit increase in [urinary fluoride] resulted in a decrease of 1.85 IQ points, but this was not statistically significant in this stratum.”

37 See, e.g., Schiff (2008); Ibarra-Santana et al. (2007); Torra et al. (1998); Warady et al. (1989).

38 In national surveys conducted between 2000 and 2008, “Black infants consistently had the lowest rates of breastfeeding initiation and duration across all study years.” CDC (2013b).

39 It is well established that non-Hispanic black children have higher levels of lead in their blood than non-Hispanic white children. CDC (2013a); Bernard & McGheein (2003). This has relevance to the risks of fluoride exposure, since animal studies have found that fluoride can exacerbate the toxicity of lead, and vice versa. Leite et al. (2011); Sawan et al. (2010); Mahaffey & Stone (1976).

40 Watters et al. (2007); Wooten & Price (2004). The reduced level of anti-oxidants found in the blood of African American adults, which may relate to low consumption of fresh fruits and vegetables (Zenk et al. 2005), has implications for fluoride toxicity, because oxidative stress is a key mechanism by which fluoride harms cells, (Barbier 2010), including in the brain. (E.g., Banala & Karnati 2015; Zhang K. et al. 2015; Basha et al. 2014; Nabavi et al. 2013; Nabavi et al. 2012a,b,c; Basha et al. 2011; Inkielewicz-Stepniak & Czarnowski 2011; Nabavi et al. 2011; Bharti & Srivastava 2009; Gao et al. 2009).

41 Studies dating back to the 1960s have found that African Americans suffer higher rates of dental fluorosis than Caucasians. Martinez-Mier & Soto-Rojas 2010; Beltran-Aguilar et al. (2015, tbl. 23); Kumar (2000); Williams & Zermer (1990); Butler et al. (1985); Russell (1962). Consistent with this, documents obtained through the Freedom of Information Act show a stark racial disparity in adolescent fluorosis rates in CDC’s 1999-2004 NHANES survey, with 58% of African American adolescents diagnosed as having the condition, versus 36% of white adolescents. FOIA (2011).
As recognized in EPA’s *Guidelines*, it is standard risk assessment practice to apply “uncertainty factors” (UF) of 10 when converting a LOAEL, NOAEL, or BMD into a safe “reference dose” (RfD) or “reference concentration” (RfC). (Martin et al. 2013) This is significant because application of even a single UF of 10 to the daily doses/concentrations of fluoride associated with neurotoxic harm in humans and animals produces an RfD or RfC that is less than, and thereby *incompatible with*, the levels of fluoride added to water for fluoridation (0.7 to 1.2 mg/L). This point is illustrated in the following table, which shows what the RfD and RfC would be if *merely* one UF of 10 was applied to the various fluoride exposures that have been associated with neurotoxic harm.

**TABLE 5: RfCs/RfDs for Fluoride If Just One Uncertainty Factor of 10 Is Applied**

<table>
<thead>
<tr>
<th>Fluoride Dose/Concentration Producing Harm</th>
<th>Study</th>
<th>Effect</th>
<th>RfD/Rfc After Application of one UF</th>
<th>Water Fluoridation Doses/Concentrations</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.06 mg/kg/day (Dose/Humans)</td>
<td>Das (2016)</td>
<td>Reduced IQ</td>
<td>0.006 mg/kg/day</td>
<td>0.03 to 0.09 mg/kg/day (Average Total Daily Dose in F areas) (NRC 2006, Tbl 2-13)</td>
</tr>
<tr>
<td>0.08 mg/kg/day (Dose/Humans)</td>
<td>Wang (2012)</td>
<td>Reduced IQ</td>
<td>0.008 mg/kg/day</td>
<td>0.03 to 0.09 mg/kg/day (Average Total Daily Dose in F areas) (NRC 2006, Tbl 2-13)</td>
</tr>
<tr>
<td>1 mg/L (Water/Rats)</td>
<td>Chouhan (2010); Wu (2008)</td>
<td>Behavioral alterations; Neurochemical changes</td>
<td>0.1 mg/L</td>
<td>0.7 to 1.2 mg/L (Water F Levels in F areas)</td>
</tr>
<tr>
<td>0.7 to 1.2 mg/L (Water/Humans)</td>
<td>Malin (2015); Peckham (2015)</td>
<td>Hypothyroidism; ADHD</td>
<td>0.07 to 0.12 mg/L</td>
<td>0.7 to 1.2 mg/L (Water F Levels in F areas)</td>
</tr>
<tr>
<td>0.7 to 1.2 mg/L (Water/Humans)</td>
<td>Sudhir (2009)</td>
<td>Reduced IQ</td>
<td>0.07 to 0.12 mg/L</td>
<td>0.7 to 1.2 mg/L (Water F Levels in F areas)</td>
</tr>
<tr>
<td>2.3 mg/L (Water/Rats)</td>
<td>Gao (2009); Liu (2014); Liu (2010); Sandeep (2013); Zhang K (2015)</td>
<td>Impaired learning; Behavioral alterations; Neurochemical changes</td>
<td>0.23 mg/L</td>
<td>0.7 to 1.2 mg/L (Water F Levels in F areas)</td>
</tr>
<tr>
<td>2.3 mg/L (Water/Humans)</td>
<td>The average water F concentration in the 13</td>
<td>Reduced IQ</td>
<td>0.23 mg/L</td>
<td>0.7 to 1.2 mg/L (Water F Levels in F areas)</td>
</tr>
</tbody>
</table>
studies reviewed by Choi (2012) which found effects at < 4 mg/L

<table>
<thead>
<tr>
<th>0.05 mg/L (Serum/Humans)</th>
<th>Xiang (2011) Reduced IQ</th>
<th>0.005 mg/L (Typical range of Serum F in US)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0.019 to 0.076 mg/L</td>
</tr>
</tbody>
</table>

The need to apply at least one UF to the doses/concentrations associated with fluoride neurotoxicity cannot seriously be disputed. After all, these are doses and concentrations associated with overt neurotoxic harm, and thus the safe reference dose will obviously need to be set at a lower level. Moreover, as discussed above, EPA’s Guidelines recognize that there is often a large degree of intra-species variability in the way humans respond to neurotoxins and a default factor of 10 is generally considered necessary to protect against this variability.42

Although we have only utilized one uncertainty factor in the analysis here, we do not mean to imply that only one UF is sufficient for converting these adverse effect levels into RfDs or RfCs. Indeed, it is clearly insufficient to apply only one UF when converting a LOAEL from an animal study into a safe dose for humans. We present the above Table, therefore, for the limited purpose of demonstrating that even if EPA were to apply an insufficiently protective UF, the resulting RfD or RfC would still be incompatible with water fluoridation; thus highlighting, once again, the overlap between the doses associated with a neurotoxic risk and the doses many Americans now receive.

Finally, FAN recognizes that EPA has a preference for utilizing Benchmark Dose (BMD) methodology for risk assessments where there is dose-response data that permits the analysis. In the case of fluoride neurotoxicity, the Xiang dataset is a suitable dataset for conducting a BMD analysis, as it shows a dose-related reduction in IQ spanning five dose groups ranging from 0.75 to 4.5 mg F/day without an apparent NOAEL. (Wang et al. 2012). EPA’s Guidelines recognize the probative value (and rarity) of a human dataset covering more than three dose groups.43 Further, the Xiang dataset benefits from the fact that the study controlled for most of the key confounding factors, including lead, arsenic, iodine, parental education, and socioeconomic status. (Xiang et al. 2003a,b; Xiang et al. 2013).

As with the LOAEL analyses discussed above, application of the BMD methodology to the Xiang dataset produces an RfD for fluoride that is incompatible with water fluoridation. Specifically, applying EPA’s BMDS software to Xiang’s dataset produces a BMD of just 1.4

42 According to the Guidelines, “In general, it is assumed that an uncertainty factor of 10 for intrapopulation variability will be able to accommodate differences in sensitivity among various subpopulations, including children and the elderly. However, in cases where it can be demonstrated that a factor of 10 does not afford adequate protection, another uncertainty factor may be considered in conducting the risk assessment.” (EPA 1998, at 65). As demonstrated by Martin et al. (2013), the use of a default uncertainty factor of 10 to account for intra-species variability is amply justified by empirical data on differences in human sensitivity related to genetic polymorphisms, gender, disease, old age, and toxicokinetics.

43 The Guidelines note that (1) “Human studies covering a range of exposures are rarely available” and (2) “Evidence for a dose-response relationship is an important criterion in establishing a neurotoxic effect, although this analysis may be limited when based on standard studies using three dose groups or fewer.” (EPA 1998, at 50 & 106).

mg F/day, if the Benchmark Response (BMR) is set at 5 IQ points, as displayed in the following figure. This result can be interpreted as predicting that children exposed to 1.4 mg fluoride per day will have, on average, 5 less IQ points than children exposed to no fluoride. The RfD would obviously need to be set at a lower level, since such a large loss in IQ is clearly an adverse effect, and because uncertainty factors would need to be added to account for variation in sensitivity within a population as large as the U.S.

**FIGURE 9: BMD for Loss of 5 IQ Points from Fluoride**
*(Linear Model, BMR = 5 IQ Points)*

Some commentators have incorrectly claimed that the recent study by Broadbent et al. establishes the safety of water fluoridation for neurologic development. The Broadbent study found no difference in the IQs of children and adults who spent their first 3 to 5 years of life in fluoridated (0.7 to 1.0 mg/L) vs. non-fluoridated (0 to 0.3 mg/L) areas of Dunedin, New Zealand. A glaring limitation with the Broadbent study, however, is that a substantial portion of the “non-fluoridated” control population used 0.5 mg/day fluoride tablets and fluoridated toothpaste, resulting in only a marginal difference in average total fluoride exposure between the fluoridated and non-fluoridated populations. In fact, in response to criticism on this point,

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44 If the BMR is set at 1 IQ point, the BMD is 0.28 mg/day of fluoride.
45 There are several other significant problems with the Broadbent study as well. First, the study did not collect any data on individual water intake or internal biomarkers of fluoride exposure (e.g., urine fluoride, etc.). Second, the study used a crude estimate of fluoride toothpaste usage (“always” vs “sometimes” vs “never”) that fails to account for the complexity of individual fluoride exposure. In conclusion, the Broadbent study does not establish the safety of fluoridation.
(Osmunson et al. 2016), the authors conceded that the average difference in total daily intake between the children in the fluoridated and non-fluoridated areas would be < 0.3 milligrams per day, while the average intake for all subjects was 0.9 mg/day. At most, therefore, the Broadbent study established that < 0.3 milligrams of fluoride was not a sufficiently large enough contrast in daily fluoride exposure to produce a demonstrable effect on average IQ in the study cohort. This does not mean, however, that the fluoride exposures in a fluoridated community are safe, since no truly low exposure comparison group existed in the Broadbent study, and the Broadbent team made no attempt to study vulnerable subsets of the population (e.g., those with suboptimal nutrition, genetic polymorphisms, etc).

The inherent limitation resulting from the Broadbent study’s comparison of populations with marginal contrasts in fluoride intake highlights an important strength of the endemic fluorosis/IQ studies from China, India, Iran, and Mexico. Specifically, the endemic fluorosis studies have generally compared communities with clear and stable contrasts in fluoride exposure, thus increasing the power of these studies to detect fluoride’s effect on IQ. Moreover, unlike Broadbent’s study, many of the endemic fluorosis studies have analyzed the relationship between IQ and individual measures of exposure (e.g., individual urine fluoride levels), thus overcoming the limitation imposed by Broadbent’s ecological (group level) estimates of fluoride intake. Although Broadbent and others have criticized the endemic fluorosis studies for failing to control for potential confounders, several of these studies did carefully control for confounders and the association between fluoride and cognitive impairment remained intact. (Choi et al. 2015; Rocha Amador et al. 2009; Xiang et al. 2003a,b; Xiang et al. 2013). Further, while it’s undisputed that many of the IQ studies used relatively simple study designs, the consistency of these studies, and their repeated corroboration by research showing that fluoride impairs learning in rodents under carefully controlled laboratory conditions, gives confidence to the conclusion that fluoride is a neurotoxin that impairs cognition.

For the foregoing reasons, the reference dose for protecting against fluoride neurotoxicity cannot reasonably be based on a risk assessment that treats the Broadbent study as establishing 0.7 to 1.0 mg/L as a NOAEL without application of an uncertainty factor(s) to account for intra-human variability and other issues left unanswered by Broadbent’s study. Indeed, as spelled out in the Guidelines, it is problematic to develop an NOAEL based on a single study of a single neurotoxic endpoint, particularly a study with such limited “dose spacing” between the groups.

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46 A previous study of total fluoride intake among 3-to-4 year olds in fluoridated and non-fluoridated areas of New Zealand found the daily intakes to be 0.68 ± 0.27 and 0.49 ± 0.25 mg F/day, respectively. (Guha-Chowdhury et al. 1996).

47 According to the Guidelines, “Neurotoxic effects (and most kinds of toxicity) can be observed at many different levels, so only a single endpoint needs to be found to demonstrate a hazard, but many endpoints need to be examined to demonstrate no effect. For example, to judge that a hazard for neurotoxicity could exist for a given agent, the minimum evidence sufficient would be data on a single adverse endpoint from a well-conducted study. In contrast, to judge that an agent is unlikely to pose a hazard for neurotoxicity, the minimum evidence would include data from a host of endpoints that revealed no neurotoxic effects.” (EPA 1998, at 55).

48 According to the Guidelines, “the NOAEL is also directly dependent on the dose spacing used in the study.” (EPA 1998, at 57)
XI. THE BENEFITS OF PREVENTING FLUORIDE NEUROTOXICITY DWARF THE COSTS OF RESTRICTING FLUORIDE CHEMICALS

A. Extent and Magnitude of Neurotoxic Risk from Fluoridation Chemicals

There is little question that neurotoxicity is a serious insult to health. (Grandjean & Landrigan 2014). In a nation besieged by neurological disorders of poorly understood etiology, both in young children and the elderly, minimizing exposures to known neurotoxic substances should be a public health priority. (Id.)

The reduction in IQ associated with fluoride exposure has been found to be severe enough in some children to produce mental retardation. (E.g., Lin et al. 1991). But even the loss of a single IQ point is associated with significant economic loss. As calculated by Spadaro et al. (2008), a loss of a single IQ point causes an average drop in lifetime earnings of $18,000 in 2005 U.S. dollars, which, when adjusted for inflation, amounts to $22,250 in current dollars.49 Since 200 million Americans now live in areas where water is fluoridated,50 and since virtually all Americans consume processed foods and beverages made with fluoridated water, any reduction in IQ from consumption of fluoride-treated water stands to have very large economic consequences.

While the precise extent to which fluoridation is reducing IQ in the U.S. cannot yet be calculated, the dose-response data from Wang et al. (2012) indicates that daily consumption of a liter of fluoridated water per day (=0.7 mg F/day) during childhood would cause IQ to drop by an average of 2.5 points when compared to children with no exposure to fluoride, while consumption of half a liter per day (=0.35 mg F/day) would cause IQ to drop by an average of 1.25 IQ points. (Wang’s data is consistent with a linear, no threshold, dose-response relationship between fluoride and IQ, and we have applied Wang’s data here with that assumption.)

In 2010, there were 74.2 million children under the age of 18 living in the U.S., of which we can estimate roughly 50 million were living in fluoridated areas.51 US Census Bureau (2011). If we apply Wang’s dose-response data and assume that these 50 million children consumed between 0.5 to 1 liters of fluoridated water per day during childhood, fluoridation would have caused a loss of between 62.5 to 125 million IQ points. Based on the earnings data from Spadaro et al. (2008), a loss in the range of 62.5 to 125 million IQ points represents a total loss in lifetime earnings of between $13.9 to 27.8 trillion for this generation.

Due to the sheer number of people exposed to fluoridation chemicals, even if only sentinel or susceptible populations in fluoridated areas suffer IQ loss, the economic impacts will still be substantial. For example, even if we conservatively assume that only 1 to 5% of children in

50 The CDC states that 211,393,167 Americans now drink fluoridated water; the vast majority of this population is consuming artificially fluoridated water, as CDC estimates that only 11,883,007 Americans have “naturally” fluoridated water. See: http://www.cdc.gov/fluoridation/statistics/2014stats.htm
51 According to the CDC, 66% of the U.S. population receives fluoridated tap water. See: http://www.cdc.gov/fluoridation/statistics/fsgrowth.htm.
a fluoridated area suffer any IQ loss,\textsuperscript{52} and even if this IQ loss averaged just 1 IQ point,\textsuperscript{53} this would still amount to 500,000 to 2,500,000 lost IQ points, with a total loss in lifetime earnings ranging from $11.1 billion to $55.6 billion for this generation alone.

In short, because of the \textit{massive} extent of exposure to fluoridation chemicals in the U.S., even small effects on IQ will have very substantial economic consequences.

\textbf{B. Societal Consequences of Restricting Use of Fluoridation Chemicals}

If EPA were to ban the waterborne use of fluoridation chemicals, the one and only potential societal consequence would be an increase in tooth decay. Current research, however, indicates that any increase in dental treatment costs would be small, inconsistent, and far less than the loss in earnings associated with even small drops in IQ.

First, FAN calls the Agency’s attention to the fact that there are no randomized controlled trials on the effectiveness of fluoridation, and few of the available studies adequately account for potential confounders like socioeconomic status, sealants, and dietary habits. (Iheozor-Ejiofor et al. 2015; Cheng et al. 2007). The evidence has thus been characterized by the Cochrane Collaboration as having “high risk of bias” and limited applicability to modern lifestyles. (Iheozor-Ejiofor et al. 2015).

Second, methodological limitations notwithstanding, modern studies of fluoridation and tooth decay have found that the difference in cavity rates between fluoridated and non-fluoridated areas is small, inconsistent, and often non-existent, particularly in the permanent teeth. (Chankanka et al. 2011a,b; Maupome et al. 2007; Warren et al. 2006; Shiboski et al. 2003; Colquhoun 1997; Heller et al. 1997; Diesendorf et al. 1997; Leroux et al. 1996; Brunelle & Carlos 1990; Yiamouyiannis 1990; Hildebolt et al. 1989).

Because of the small and inconsistent differences in cavities now seen between fluoridated and non-fluoridated areas, sensitive measurements of tooth decay must be utilized in order to detect \textit{any} differences in decay.\textsuperscript{54} But, even when sensitive measurements are utilized, the differences remain small in absolute terms, inconsistent, and overshadowed by the influence of other factors known to affect decay. (Chankanka et al. 2011a; Warren et al. 2006; Armfield & Spencer 2004). A large-scale study in Australia, for example, found that adolescents who consumed fluoridated water their entire life had just 0.08 less decayed tooth surfaces (1.35 vs. 1.43 DMFS) than adolescents who consumed non-fluoridated water their entire life. (Armfield & Spencer 2004, at 290 tbl.3). Consistent with these findings, studies from Canada, Cuba, Finland, Germany, and the United States did not detect \textit{any} measurable

\textsuperscript{52} We base the 1 to 5% estimate on the approximate percentage of children with serum fluoride levels in the range (~0.05 mg/L) associated with a 4-point IQ drop (n = ~1%), and the approximate percentage of children with urinary fluoride levels (~1.3 mg/L) associated with clear reductions in IQ (n = 5%). For discussion of this data, see pages 9 to 12 above. Since the serum and urine fluoride data is for the \textit{general population}, these estimates likely \textit{understate} the percentage of children in \textit{fluoridated} areas with serum and urinary fluoride levels in this range.

\textsuperscript{53} This is a substantially lower loss in IQ than would be predicted by existing research. As noted in footnote 54 above, the serum fluoride level (~0.05 mg/L) upon which this estimate is based was associated with a 4-point drop in IQ by Xiang et al. (2011). Further, research on susceptible populations has found dramatic losses in IQ from fluoride exposure, including an average 15-point drop among malnourished children with mild fluorosis. Das & Mondal (2016).

\textsuperscript{54} As evident by the studies of Yiamouyiannis (1990) and Brunelle and Carlos (1990), the difference in tooth decay between fluoridated and non-fluoridated populations, while detectable when calculated in terms of Decayed, Missing & Filled Surfaces (DMFS), is not large enough to be detectable when calculated in terms of Decayed, Missing and Filled Teeth (DMFT).
increase in decay following the termination of water fluoridation programs.\textsuperscript{55} (Maupome et al. 2001; Burt et al. 2000; Kunzel et al. 2000a,b; Seppa et al. 2000).

Third, one of the few \textit{empirical} investigations of \textit{actual} dental costs in fluoridated vs. non-fluoridated areas found little meaningful difference in frequency or costs of treatment. (Maupome et al. 2007). The study examined the frequency and costs (in 1995 U.S. dollars) of restorative dental procedures over a six-year time period in fluoridated and non-fluoridated areas of Oregon and Washington. Consistent with other recent research, the authors noted that the difference in frequency and costs of dental treatment was “generally small,” with several of the age groups in the fluoridated areas having a higher frequency of dental treatment procedures than their peers in the non-fluoridated areas. (Maupome et al. 2007, at 228, tbl. 3). In total, the dental treatment costs in the fluoridated areas over the six-year period averaged $355 versus $387 in the non-fluoridated areas.\textsuperscript{56} \textit{(Id. at 228, tbl. 4).} When adjusted to 2016 dollars, the average difference in dental costs was thus only $51 over the 6-year period, \textit{or just over $8 per person per year}. With an average life expectancy of 78.8 years,\textsuperscript{57} the Maupome study suggests that fluoridation saves an average of $665 in lifetime dental costs in the U.S. This amounts to less than 3 percent of the reduction in lifetime earnings that results from the loss of a single IQ point ($22,250).

Finally, the cost-effectiveness study (Griffin et al. 2001) that advocates of fluoridation generally rely upon, is based on theoretical estimates that have several major, demonstrable problems that inflate the purported savings. (Ko & Thiessen 2015). The Griffin paper provides estimates of the annual savings in dental costs from fluoridation (in 1995 U.S. dollars) based on a review of several studies of caries rates in fluoridated vs. non-fluoridated communities. The paper estimates that fluoridation provides a net savings of anywhere from $0.85 to $33.71 per year. (Griffin et al. 2001, at 82, tbl. 4). Over the course of the average lifespan, this amounts to a lifetime savings ranging from $67 to $2656 per person when expressed in 1995 U.S. dollars. Adjusting for inflation, this amounts to a lifetime savings of $106 to $4,207 in 2016 dollars, which, even at its zenith, amounts to less than 20% of the costs ($22,500) incurred from loss of a single IQ point.

As discussed by Ko and Thiessen (2015), Griffin’s cost-savings estimates suffer from several important limitations. First, and foremost, Griffin did not make any attempt to include the costs of treating dental fluorosis in the costs side of the ledger, thereby inflating the net savings. This is a particularly significant omission since Griffin elsewhere estimated, in a separate paper, that fluoridating water causes 2 percent of children to develop aesthetically objectionable fluorosis on their front teeth. (Griffin et al. 2002). With approximately 50 million children now living in fluoridated areas, this amounts to roughly 1 million children developing aesthetically objectionable fluorosis on their front teeth as a direct result of water fluoridation. But even this is an under-estimate, since Griffin based this on the NIDR’s 1986-87 national survey, and more recent national surveys show that both the rate and severity of dental fluorosis have increased considerably over the past 20 years. (NHANES 2014; Beltran 2010). In fact, as mentioned earlier, the 2011-2012 NHANES survey found that an astonishing 21% of adolescents now have \textit{moderate} fluorosis, and an additional 2% have severe fluorosis. (NHANES 2014) Since many children who have fluorosis staining on their front teeth will

\begin{itemize}
  \item \textsuperscript{55} A recent Canadian study by McLaren et al. (2016) reported an increase in decay following cessation of fluoridation in Calgary. However, as explained by Connett (2016), the entirety of this purported increase disappears when survey data omitted from the paper is considered.
  \item \textsuperscript{56} The average costs estimate is for people who had at least one restorative procedure during this time.
  \item \textsuperscript{57} See: \url{http://www.cdc.gov/nchs/fastats/life-expectancy.htm}
\end{itemize}
have it cosmetically treated, the aggregate costs of this treatment will be substantial, and any cost-effectiveness evaluations of fluoridation that fail to account for these treatment costs will artificially inflate the cost-savings of fluoridation. Griffin’s cost-savings estimates should not, therefore, be taken at face value, but even if they are, they suggest a range of lifetime savings for the current population under 18 (i.e., $5.3 to $210 billion) that is still substantially less than the range of earnings losses associated with fluoridation-related drops in IQ (i.e., $11.1 billion to $27.8 trillion).

C. Availability and Potential Hazards of Substitutes to Fluoridation Chemicals

The addition of fluoridation chemicals to drinking water began in the U.S. prior to the advent of topical fluoride products in an era when public health authorities believed fluoride’s predominant benefit to teeth comes from ingestion. Things have changed dramatically since that time.

Today, over 95% of toothpastes contain fluoride, as do many other dental products, (CDC 2013c), and dental researchers now universally acknowledge that fluoride’s predominant benefit is topical, not systemic. (E.g., Fejerskov 2004; Featherstone 2000). As explained in the Journal of the American Dental Association, “fluoride incorporated during tooth development is insufficient to play a significant role in cavity protection.” (Featherstone 2000, at 891). The Centers for Disease Control has confirmed the primacy of fluoride’s topical mechanisms, declaring that “fluoride’s predominant effect is posteruptive and topical.” (CDC 2001, at 4). The NRC has confirmed this as well, stating that “the major anticaries benefit of fluoride is topical and not systemic.” (NRC 2006, at 13).

Since fluoride’s primary benefit comes from topical contact with the teeth, there is little benefit from swallowing fluoride, in water or any other product. In fact, a recent study of the relationship between tooth decay and total daily fluoride ingestion failed to find a detectable relationship between the two. (Levy et al. 2009). Other recent studies investigating the relationship between tooth decay and individual biomarkers of fluoride intake (e.g., toenail fluoride content and dental fluorosis) have reported similar results. (Charone et al. 2012; Komarek et al. 2005).

The widespread availability of topical fluoride products highlights the lack of necessity of adding fluoridation chemicals to water, particularly since the quality of evidence for fluoride toothpastes has been recognized as vastly superior to the quality of evidence for water fluoridation. (Cheng et al. 2007, at 701). Furthermore, it is well established that western countries that do not fluoridate their water have tooth decay rates that are just as low, and often lower, as western countries that do fluoridate their water. (Cheng et al. 2007; Pizzo 2008).

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58 Research has found that teeth with dental fluorosis, including in its “mild” forms, is perceived as an objectionable condition that warrants dental treatment. (E.g., Alkhatib et al. 2004; Riordan 1993). Consistent with this, studies have repeatedly found that staining of the front teeth, including the white splotches of fluorosis, can cause children significant anxiety and distress about the appearance of their teeth. (E.g., Tellez et al. 2012; Marshman et al. 2008).

59 This is evident when comparing the Cochrane Collaboration’s systematic review of the effectiveness of fluoride toothpastes with its systematic review of water fluoridation. Compare Iheozor-Ejiofor et al. (2015) with Marinho et al. (2003).

60 For additional data demonstrating the lack of difference in tooth decay rates between countries with extensive water (and/or salt) fluoridation and those without, FAN refers EPA to the documentation available at: http://fluoridealert.org/studies/caries01/

While fluoride toothpastes and other fluoridated dental products carry their own potential hazards when ingested, these products—unlike drinking water—are not designed to be ingested. Further, unlike the addition of fluoridation chemicals to drinking water, the use of topical fluoride products does not result in the contamination of processed foods and beverages, thus making it easier to regulate the amount of fluoride ingested when topical fluoride products are the vehicle for delivering fluoride to those who want it.

D. Impacts on Industry, Employment & International Trade from Restricting Fluoridation Chemicals

Prohibiting the addition of fluoridation chemicals to drinking water will have little, if any, impact on industry, employment and international trade. The chemicals used for fluoridation are waste by-products of the U.S. phosphate industry and various Chinese fertilizer and chemical companies. The sale of fluoridation chemicals represents a very small portion of the U.S. phosphate industry’s overall sales, and thus removing this very limited market will have little impact on the profitability of the phosphate industry. Finally, while ending fluoridation will curb imports of fluoridation chemicals from China, it will not impact American exports, because—to the best of FANs knowledge—U.S. companies do not export fluoridation chemicals abroad. Accordingly, ending fluoridation will not have any disadvantageous impact on America’s balance of trade.

XII. IT IS IN THE PUBLIC INTEREST FOR EPA TO ACT

Under SDWA, the EPA can limit the legally permissible levels of chemicals in public drinking water supplies by enacting “Maximum Contaminant Levels” (MCLs). The EPA can effectively ban fluoridation under SDWA, therefore, by enacting an MCL below the so-called “optimal” concentration of fluoride used in fluoridation programs (0.7 mg/L). Since an MCL does not distinguish, however, between fluoride that is added to water and fluoride that occurs naturally therein, implementing an MCL below the level used in fluoridation would force communities with elevated levels of naturally occurring fluoride to implement filtration programs.

As with other naturally occurring toxicants, like arsenic, FAN recognizes that natural fluoride contamination of some rural water supplies is a problem that needs to be addressed. However, there is a distinct policy difference between a risk imposed on a population through the purposeful addition of a chemical to water, versus a risk that arises from a naturally occurring phenomena beyond human control.

Differential treatment is further justified by laboratory and epidemiological research linking artificial fluoridation chemicals (i.e., fluorosilicic acid and sodium fluorosilicate) with pipe corrosion and elevated blood lead levels. (Coplan et al. 2007; Maas et al. 2007; Macek et al. 2006; Masters et al. 2000). This research includes the CDC’s own study of the issue, which analyzed the blood lead levels of children from the 1988-1994 National Health and Nutrition Examination Survey. (Macek et al. 2006).

Although the CDC study is sometimes touted as refuting the link between fluoridation and lead hazards, a close look at its data reveals that it is actually consistent with the fluorosilicate/lead thesis. As can be seen in Table 4 of the study, fluorosilicic acid was associated with:

• a 20% increased risk (but not statistically significant) for high blood lead levels among children living in houses made prior to 1946;
• a 40% increased risk (but not statistically significant) for high blood lead levels among children living in houses made between 1946 and 1973;
• a 70% increased risk (but not statistically significant) for high blood lead levels among children living in houses made after 1974;
• a 530% increased risk (which was statistically significant) for high blood lead levels among children living in houses with unknown ages.

Since three of these four elevated risks were not statistically significant, the CDC dismissed them as essentially random aberrations. However, the consistency in the direction of the risk, coupled with the large and significant five-fold increased risk for children in homes of unknown age, raises a serious red flag.

Even the CDC acknowledged that this study does not refute the connection between fluoridation and lead, and that “it is possible that larger samples might have identified additional, significant differences.” (Macek et al. 2006, at 133). Indeed, when Coplan et al. re-analyzed CDC’s data by placing all children exposed to fluorosilicic acid and sodium fluorosilicate in one group (“silicofluorides”), and all other children in another, they found that the children exposed to “silicofluoridated” water had a significantly elevated risk of having high blood lead levels. (Coplan et al. 2007, at 1039-40). According to Coplan’s re-analysis, children from the silicofluoridated communities had a 20% greater risk of having blood lead levels in excess of 5 ug/dl. Coplan’s team estimated that the risk for exceeding the 10 ug/dl threshold would be even greater. (Id. at 1039 tbl.9).

The repeated association between fluoridation chemicals and elevated blood levels provides further reason why it is in public interest for EPA to prioritize a targeted ban on fluoridation additives.

XIII. CONCLUSION

When considering the principles set forth in EPA’s Guidelines for Neurotoxicity Risk Assessment, FAN submits that fluoridation is incompatible with a neurologically safe use of fluoride. FAN further makes this request on the grounds that fluoride’s predominant role in caries prevention comes from topical contact and thus there is no reasonable justification to expose hundreds of millions of Americans to the neurotoxic risks of systemic fluoride via water and processed foods when topical fluoride products are now widely available for individual use. Most western nations, including the vast majority of western Europe, have already rejected water fluoridation. The EPA is the one federal agency with the authority to make this happen here in the U.S. We urge EPA to act accordingly.

XIV. Bibliography

The bibliography is at the end of this report on pages
Note: A flash drive containing all the studies in the bibliography was sent March 11, 2017, by Express Mail to the: EPA Docket Center, WJC West Building, Room 3334, 1301 Constitution Avenue, NW, Washington, DC 20004, for delivery on March 13.
Part 2: Risk Assessment: U.S. Children Receive Unsafe Levels of Fluoride

The finding of a recent publication by Hirzy et al. (2016)\(^1\), "Developmental neurotoxicity of fluoride - A quantitative risk analysis toward establishing a safe daily dose of fluoride for children," is that there may not be a threshold for fluoride’s effect on the developing brain, and that a Maximum Contaminant Level Goal consistent with the legislative history of the U.S. Safe Drinking Water Act should be zero.

Hirzy et al. (2016) used the best available data sources on the effect of fluoride on children’s IQ along with standard risk analysis techniques used by the U.S. EPA. The data used came largely from China, where studies comparing individuals or areas of high and low fluoride exposures via drinking water are not confounded by co-exposures to fluoride via fluoridated tooth paste and food and beverages prepared with fluoridated water, as is the case in the United States.

The results obtained in this analysis include Reference Doses (RfDs) based on the LOAEL/NOAEL and Benchmark Dose (BMD) methodologies and appropriate Uncertainty Factors. The former methodology, using UFs for LOAEL to NOAEL\(^2\), intra-individual variability\(^3\), and in utero toxicity\(^4\), resulted in an RfD of 0.047 mg/day. The latter methodology, with a Benchmark Response Level of loss of 5 IQ points and the UFs for intra-individual variability and in utero toxicity as above yielded an RfD of 0.045 mg/day. With a Benchmark Response Level of loss of 1 IQ point, the RfD determined was 0.0090 mg/day.

The LOAEL/NOAEL method was applied to an “adverse effect concentration” of fluoride in drinking water of 3.0 mg/L, taken from the 10 statistically significant IQ losses occurring at that level or below as reported in the Choi et al. (2012)\(^4\) meta-analysis of fluoride’s impact on children’s IQ. Drinking water intakes and fluoride intakes from food as reported by Xiang et al. (2009)\(^5\) were used to estimate total fluoride daily doses and derive an LOAEL. The Xiang research group also accounted for iodine\(^6\), lead\(^7\), and arsenic\(^8\) co-exposures, as well as parental income and educational levels\(^6\).

The BMD method used dose-response data from Xiang et al. (2003)\(^6\).

Data from Choi et al. (2012)\(^4\) also included studies that had “adverse effect concentration” extremes of fluoride in drinking water of 0.88 mg/L in Lin et al. (1991)\(^9\) and 8.3 mg/L in Wang SX et al. (2007)\(^10\), to which we applied the LOAEL/NOAEL method. These yielded RfDs of 0.018 mg/day and 0.12 mg/day, respectively. The former has special significance in that the 0.88 mg/L fluoride concentration was in an area of low iodine exposures, and low iodine exposures also occur in the United States (Caldwell 2011)\(^11\).

A study by Broadbent et al. (2015)\(^12\) purporting to show that community war fluoridation has no effect on children’s IQ is refuted by the above study and those of Menkes et al. (2014)\(^13\) and Osmunson et al. (2016)\(^14\) which show that the Broadbent study did not have sufficient differences in fluoride exposures to detect an effect, and it did not control for confounding variables.

The Maximum Contaminant Level Goal for fluoride should be zero.
References:


Part 3: Cancer and its Cost Due to Fluoridation Chemicals

Industrial grade fluoridation chemicals used in the U.S. contain:

- trace amounts of at least two known human carcinogens: Arsenic\(^1\) and Radionuclides\(^2\);
- trace amounts of Lead which is categorized as a “reasonably anticipated human carcinogen\(^3\);”
- at least three heavy metals whose toxicity targets the central nervous system: Arsenic, Lead and Mercury\(^4\).

While the U.S. EPA’s health-based drinking water standard for Arsenic, Lead, and Radionuclides is zero, these contaminants are allowed to enter the public drinking water supply via fluoridation chemicals.\(^5\)

In 2013 an article by Hirzy et al.\(^6\) was published in *Environmental Science and Policy* comparing costs and benefits of using pharmaceutical grade (USP) Sodium fluoride (NaF) and industrial grade Hydrofluorosilicic acid (HFSA) as fluoridating agents. The authors calculated the number of cancers that would be caused by both agents and the costs associated with their use. Arsenic levels in HFSA vary substantially but are typically about 30–35 mg/kg (see Supplemental Material Appendix A). The most common form of NaF used in tooth paste in the U.S. is made by a different process and contains markedly less arsenic. In their original published paper the authors erroneously failed to account for life time costs of using the two agents, while using life time arsenic exposures to calculate the number of resulting lung and bladder cancer cases. The resulting error was corrected in a Corrigendum\(^7\) published the following year, that made these points:

1. On an annual basis of costs and typical exposures these results obtain:
   - a. HFSA: 4.6 cancer cases; treatment costs of $16.1 million. Chemical costs of $23 million; Total costs of $39.1 million.
   - b. NaF: 0.05 cancer cases with treatment costs of $0.175 million. Chemical costs of $120 million; Total costs of $120.175 million.

2. On an annual basis of costs and exposures permissible under NSF, Inc. Standard 60
   - a. HFSA: 59 cancer cases; treatment costs of $206.5 million. Chemical costs of $23 million; Total costs of $229.5 million
   - b. NaF: 0.05 cancer cases with treatment costs of $0.175 million. Chemical costs of $120 million; Total costs of $120.175 million

3. On an annual basis of costs and exposures reported by Reeves (IFIN 2001)
   - a. HFSA: 26 cancer cases; treatment costs of $91 million. Chemical costs of $23 million; Total costs of $114 million
   - b. NaF: 0.05 cancer cases with treatment costs of $0.175 million. Chemical costs of $120 million; Total costs of $120.175 million

Under Example 3, the cost of avoiding the cancers anticipated is $240,000/per case.
The authors concluded that the “U.S. could save $1 billion to more than $5 billion/year by using USP NaF in place of HFSA while simultaneously mitigating the pain and suffering of citizens that result from use of the technical grade fluoridating agents.”

References:


5. EPA. Table of Regulated Drinking Water Contaminants, Online at https://www.epa.gov/ground-water-and-drinking-water/table-regulated-drinking-water-contaminants


Part 4: Fluoride and Cancer, particularly Osteosarcoma

In January 2016, FAN prepared a detailed submission on the existing literature on fluoride and cancer for the National Toxicology Program literature review on fluoride carcinogenicity. The submission provides detailed discussion and review of all recent individual studies, most of which are on the relationship between fluoride and osteosarcoma. The submission includes a cover letter with overview and numerous appendices which provide details and provides a review of the literature current as of that date. These submissions include:

Cover Letter and Submission to the National Toxicology Program’s review of fluoride as a carcinogen. By Connett & Neurath. January 8, 2016. (19 pages. Scroll down to read full submission.)

The Appendices attached to submission:

Appendix 1A. Revisiting the Fluoride-Osteosarcoma connection in the context of Elise Bassin’s findings: Part I. Originally submitted to the NRC on March 2, 2005.
Appendix 1B. Revisiting the Fluoride-Osteosarcoma connection in the context of Elise Bassin’s findings: Part II. Originally submitted to the NRC on March 2, 2005.
Appendix 5. Thiessen submission to California Office of Environmental Health Hazard Assessment, July 2011.
Appendix 8-A. Review of Blakey/McNally 2014 fluoride--osteosarcoma ecological study Great Britain.
Appendix 10-B. Young 2015 correction for misclassification of fluoridation exposure to osteosarcoma risk.
Appendix 11. Review of Levy 2012 F-osteosarcoma ecological study USA.
Part 5: Food Treated with Highly Neurotoxic Fumigant

When EPA approved the use of Sulfuryl fluoride in 2004\(^1\) and 2005\(^2\) as a postharvest fumigant it approved the highest ever tolerances (approved residues) for Fluoride in and/or on food in U.S. history. At the same time, EPA also approved a second set of tolerances for Sulfuryl fluoride, a highly neurotoxic substance that breaks down rapidly to fluoride in the body. In studies performed by Dow Chemical, Sulfuryl fluoride was found to harm the brains of every animal species tested (rats, mice, rabbit, dog). Vacuolation (holes) in the brain’s neurons and White Matter were a consistent effect\(^3\).

The U.S. Department of Agriculture (USDA) published a database of fluoride levels in beverages and food in 2004\(^4\) on the basis that a national fluoride database would be a useful tool “for epidemiologists and health researchers to estimate the intakes and to investigate the relationships between intakes and human health.” USDA updated that database in 2005\(^5\) to include “a column with mean values in part per million” and data from more sources. However, USDA’s lists were generated prior to the approval of Sulfuryl fluoride as a food fumigant.

The approved fluoride tolerances (residues) on foods from both Cryolite (the pesticide approved to leave high residues of fluoride on food) and Sulfuryl fluoride can be seen here\(^6\). (Organic food is not grown with Cryolite nor fumigated with Sulfuryl fluoride. Sadly, most poor people cannot afford the extra cost for organic.)

Fluoride Action Network, together with Beyond Pesticides and the Environmental Working Group (the “Objectors”), collaborated for more than seven years in formally objecting to tolerances for Sulfuryl fluoride. In 2006, the Objectors submitted a 160-page Consolidation of the Objections to EPA\(^7\) at their request. In response to these Objections, the EPA proposed a phase-out of Sulfuryl fluoride as a food fumigant in a January 2011 Federal Register Proposed Rule\(^8\) on the basis that children were overexposed to fluoride. However, due to intense lobbying efforts by Dow AgroSciences, who had support from several corporate food groups and even an environmental group (whose comment on fluoride was “it only leaves white spots on teeth”), the U.S. Congress approved language in the 2013 Farm Bill that effectively stopped EPA’s phase out of Sulfuryl fluoride\(^9\).

In this same Proposed Rule the EPA noted it agreed with all the Objections, which included a request for a Developmental Neurotoxicity Study on Sulfuryl fluoride, which is something that should be a high priority for EPA.

As far as we know, there are only two countries that fumigates food with Sulfuryl fluoride: the U.S. and Australia. European countries allow fumigation with Sulfuryl fluoride, but only in empty warehouses and empty mills.

References:


Part 6: Fluoride and Environmental Justice

Environmental justice, as defined by the U.S. Environmental Protection Agency (EPA), is the fair treatment and meaningful involvement of all people regardless of race, color, national origin, or income, with respect to the development, implementation, and enforcement of environmental laws, regulations, and policies. According to the EPA, environmental justice will be achieved when all communities and persons across this nation are afforded the same degree of protection from environmental and health hazards, and are afforded equal access to the decision-making process to have a healthy environment in which to live, learn, and work.

In this section we ask if water fluoridation, and U.S. fluoride policy in general, is an environmental racism issue that is consistent with EPA definitions and guidelines for environmental justice. The answer in short is yes; water fluoridation is an environmental justice issue for several reasons set forth below.

Evidence is presented that artificial water fluoridation as promoted by U.S. federal agencies has been ineffective at helping fight tooth decay in the inner cities and in addition causes “disproportionately high and adverse human health effects...on minority populations and low income populations,” in violation of Presidential Executive Order 12898 of February 11, 1994.

The issue has been compounded by the failure of these same agencies to warn minority populations of their special vulnerabilities to fluoride exposure in general and the water fluoridation program in particular. The current ongoing determination by the Environmental Protection Agency’s (EPA) Office of Water of a new Maximum Contaminant Level Goal (MCLG) and the Maximum Contaminant Level (MCL) for fluoride as reported in 2011 is scientifically flawed and betray an insensitivity to Environmental Justice issues. There are more positive and creative ways of fighting tooth decay in the inner city that address other Environmental Justice issues in a more holistic fashion.

Those who promote fluoridation often do so based upon equity considerations. They correctly claim that most tooth decay is concentrated in low-income families and especially in communities of color. Yet the evidence suggests that promoters were overly optimistic when they thought that forcing everyone to swallow fluoride would even-up the playing field when it comes to dental inequalities. Water fluoridation has not evened up the playing field, as evidenced by the numerous reports of the dental crises being reported among low income and communities of color in large U.S. cities that have been fluoridated for decades. As we explain below, far from helping low-income families and communities of color, fluoridation is actually causing disproportionate harm. In fact, fluoridation is a rather graphic example of an environmental injustice.

Access to dental treatment is also an issue. With low-income families and communities of color suffering from disparate rates of tooth decay, it is tragic that 80% of dentists in the U.S. refuse to treat children on Medicaid. The poor need special and focused attention. Putting a toxic substance into everyone’s drinking water is a very poor substitute.

Environmental Justice issues go beyond the failure of the government agencies to identify disparate risks of exposure or warn communities of color about their vulnerability to fluoride. The practice of water fluoridation penalizes communities of color and families of low income in a variety of ways, including:
1) Low income families cannot afford to avoid fluoridated water if they want to do so because both removal equipment and bottled water (for drinking and cooking) is very expensive.

2) Low income families cannot afford the expensive treatments to conceal the damage that fluoride can cause to the enamel (dental fluorosis).

3) Dental fluorosis rates are higher in Black and Hispanic communities than White communities especially in the more severe forms that require treatment (Beltrán-Aguilar et al., 2005).

4) Fluoride is more toxic when exposure is accompanied by poor nutrition. Poor nutrition is more likely to occur in low-income families than those with higher incomes.

5) Lactose intolerance is more frequent among Blacks and other ethnic groups than white, and less consumption of dairy products typically means lower exposure to calcium. Calcium in the diet helps to a certain extent to protect against absorption of fluoride from the gut.

6) Minority families are less likely to breastfeed their children. Infants ingesting powdered infant formula mixed with fluoridated water can ingest over 200 times more fluoride than a breastfed child. In addition, low fluoride, ready-to-feed formula is more expensive than powdered formula (as is distilled water), representing an additional cost burden to low income families.

7) Fluoride is neurotoxic and in 45 studies it has been associated with lowered IQ in children. The last children that need their IQ lowered are children from low-income families.

8) Low income and minority groups living in the inner city are likely to have a greater exposure to lead. Fluoride appears to enhance the toxicity of lead. Lead increases the risk of dental fluorosis. Both lead and fluoride are neurotoxic.

9) Children from low-income families are more likely to get mercury amalgam fillings than families with higher income. Mercury is neurotoxic. The combined impact of mercury and fluoride on a child’s mental development may be greater than either acting alone.

10) Minority communities have a greater incidence of kidney disease. Poor kidney function increases fluoride’s uptake into the bone, which is likely to increase the rates of arthritis and hip fractures (over a lifetime).

11) Minority communities have a greater incidence of diabetes, some forms of which lead to an increased consumption of water, which in turns leads to a greater consumption of fluoride.

12) Race may be a factor in sensitivity to certain thyroid diseases, which may make communities of color more vulnerable to fluoride’s impacts on thyroid function.

13) African-Americans experience a disparate rate of pre-term births, of which there is an association with water fluoridation.

Many of these issues are discussed in more detail and documented in the text below.

**A Timeline of the Fluoridation Program with Special Emphasis on Dental Fluorosis and Environmental Justice (early 1900s to 2017)**

In the early 1900s a handful of dentists, particularly Frederick McKay (1916, 1928) and G.V. Black & McKay (1916) were interested in what was causing a condition (which was prominent in both Texas and Colorado), which led to discoloration and marking of the teeth. The condition was called “dental mottling.” McKay described dental mottling as “the most poorly constructed enamel of which there is any record in the history of dentistry.”

1925

Norman Ainsworth in a study of 4000 children in Essex County in England reported a lowered prevalence of dental caries in Maldon and Heybridge, which were areas endemic for “dental mottling” (now known as areas with high natural levels of fluoride in the water) – (see Mullen, 2005).

1928

Frederick McKay (1928) noted that while the discoloration and marking of the teeth in cases of “dental mottling” looked very bad, it did not appear to increase the child’s susceptibility to tooth decay, in fact there appeared to be less tooth decay among children with dental mottling than those without.

1931

In 1931 three separate research teams (Smith et al., 1931; Churchill et al, 1931 and Vehu, 1931) identified the cause of this condition as fluoride in the drinking water and the name was changed to “dental fluorosis,” which literally means “poisoning of the teeth by fluoride.” It was quickly recognized that dental fluorosis was a “systemic” not a “topical” effect. It can only be contracted before the permanent teeth have erupted. It is occasionally seen in the primary teeth (Warren et al., 1999) but it is most frequently observed in the secondary teeth.

1930s and 40s

Under the leadership of H. Trendley Dean the US Public Health Service (PHS) studied the occurrence of this condition throughout the USA. In addition to this mapping exercise Dean subsequently published his famous classification of the different levels of severity of this condition: very mild, mild, moderate and severe. According to Dean et al. (1934, 1935):

**Very mild** ranged from white patches on the cusp of the teeth to up to 25% of the enamel impacted.

**Mild** impacted between 25 and 50% of the enamel.

**Moderate** impacted 100% of the enamel.

**Severe** impacted 100% of enamel with pitting and chipping.

Pictures illustrating these four levels of dental fluorosis are given in Figure 1.
1942

In 1941-1942, Dean and his colleagues published his famous 21-city study, which purported to show that as the fluoride level in the water went from about 0.1 to 2.6 ppm tooth decay fell. Most of reduction occurred between 0.1 and 0.9 ppm, with only a modest further decrease occurring between 0.9 and 2.6 ppm. He further noted that there was little noticeable dental fluorosis occurring below 1 ppm. Thus was born the notion that the “optimal level” for reducing tooth decay while minimizing the risk of dental fluorosis was 1 ppm. Dean later indicated that at 1 ppm only about 10% of children would have dental fluorosis and only in the very mild category. Dean later testified in the US Congress that mild dental fluorosis would not be an acceptable trade off for lowered tooth decay. This is what he said to the Delaney Committee in 1952:

“We don’t want any ‘mild’ [fluorosis] when we are talking about fluoridation. We don’t want to go that high…I don’t want to recommend any fluoridation where you get any ‘mild’”. (Connett et al., 2010, page 110).

All the children in Dean’s 21 city study were white: there were no Blacks or Hispanics in the 7,257 children studied.

Figure 1. Pictures of the four levels of dental fluorosis. (Photographs by Dr. Hardy Limeback and Dr. Iain Pretty, et al)
1945

By 1945 Dean and others were convinced that natural levels of fluoride in the water lowered tooth decay and there were no side effects other than dental fluorosis. The question became: could one deliberately add a fluoride-containing compound to the public water supply and achieve the same result? The PHS decided to run a series of experiments to check this out. Instead of these experiments being conducted in the form of randomized control trials on individual volunteers they were launched on whole cities. In their discussions the early promoters did not want to use the word experiment because as they said, "people don’t like to be experimented upon!" They also saw them more as demonstrations – demonstrating that what they had seen with natural fluoride could be reproduced with artificial fluoride. These fluoridation experimental trials began in 1945 in Grand Rapids, MI; Newburgh, NY and Brantford, Ontario, Canada using sodium fluoride at 1 ppm (1 mg fluoride/liter of water). Most now agree that the methodology used in these experiments would not be acceptable by modern epidemiological standards but nevertheless they provided the foundation for the widely accepted belief in this practice for many decades. Dr. Philip Sutton wrote two monographs and a whole book on the inadequacies of these experimental trials, and his arguments have never been successfully rebutted by proponents (Sutton, 1959, 1960, 1996).

1950

Figure 2: Dean’s famous 21-city plot of Dental caries experience in each community versus the concentration of fluoride in the community’s water supply in ppm (Dean et al., 1941, 1942)
The trials were meant to last for 10 years, but before any of them had been completed the PHS endorsed fluoridation in 1950 and over the next two years with little science on the table it was endorsed by nearly every dental, public health and medical body in the country. Despite their lack of science these endorsements have been used heavily by promoters ever since.

1956

In 1956, Schlesinger et al. published the health findings for the Newburgh, NY (control city Kingston, NY) experiment. They reported that young men in fluoridated Newburgh had a significantly greater number of cortical bone defects than non-fluoridated Kingston (about 2 to 1). There was no follow-up on this finding, which is surprising because the cortical bone is the outside layer of the bone and protects against fracture. However, Dr. Caffey who examined the X-rays said in 1955 that the age, sex and anatomical distribution of these defects were remarkably similar to osteosarcoma. 20 years later this comment prompted the National Academy of Science (NAS) in 1977 to recommend that researchers check to see if there was an increase in osteosarcoma in young men under 30 in fluoridated communities (NAS, 1977). The other finding by Schlesinger was that young girls were menstruating on average 5 months earlier in the fluoridated community than in the non-fluoridated one. This observation was not considered important at the time but today it is intriguing in the context of Luke’s findings, a) that fluoride accumulates in the human pineal gland (Luke, 2001) and b) lowers melatonin production in animals and shortens the time to puberty (Luke, 1997).

1962

A January 10, 1962 internal memorandum, from a top PHS official, F.J. Maier, in connection with the first fluoridation trial, revealed that,

“negroes in Grand Rapids had twice as much [dental] fluorosis than others.”

Based on this, Maier asked,

“In a community with a larger number of negroes (say in Dekalb County, Georgia) would this tend to change our optimum fluoride levels?” (Maier, 1962)

1983

In 1983 the U.S. Surgeon General convened a panel to review the literature as part of the process of determining a safe drinking water standard for fluoride (the MCL, or Maximum Contaminant level). One member of the panel on reviewing pictures of dental fluorosis stated that,

“You would have to have rocks in your head to allow your child much more than two parts per million (Grossman, 1990)...” Over-exposure to fluoride damages teeth as the photos of the various stages of dental fluorosis above, also known as enamel fluorosis, clearly show.

1985

When the EPA published its rationale for both a MCL and MCLG (goal) at the very high level of 4 ppm they did not include dental fluorosis as an adverse health effect but as a “cosmetic effect.”
effect” (for which they produced a non-enforceable secondary standard of 2 ppm). Instead of dental fluorosis the EPA used skeletal fluorosis as the health effect of concern – even so, they did not use the first signs of skeletal fluorosis (which are identical to arthritis) but the terminal stages in which the patient is crippled, i.e. crippling skeletal fluorosis. Choosing the gross end point of the problem conflicts with the normal way that the EPA comes up with protective standards. Normally they determine the Lowest Observable Adverse Effect Level (LOAEL) and then apply safety factors to that. Note also that U.S. standard of 4 ppm is about three times the WHO guideline of 1.5 ppm, which is the standard adopted by Canada, Mexico and most of the rest of the world.

Professionals at the EPA who witnessed this process have stated that the level of 4 ppm was chosen for political not scientific reasons. It was chosen to accommodate concerns of states like South Carolina which did not want to spend a lot of money removing high natural fluoride levels from drinking water if a lower level were chosen (Grossman, 1990 in Appendix A; and FAN, 2007).

1985

In a Texas survey, published in 1985, Butler et al. reported that the prevalence of dental fluorosis among African-American children was greater than for Hispanic and non-Hispanic white children. The reported Odds Ratio was 2.3.

1986-1987

The National Institute of Dental Research (NIDR) conducted one of the largest surveys of tooth decay and dental fluorosis ever carried out in the USA. They looked at the teeth of approximately 39,000 children in 84 communities. The dental caries results were reported in 1990 by Brunelle and Carlos, but the dental fluorosis data was not reported until 1997 by Heller et al. The latter reported 29.9% of the children living in communities with fluoride levels between 0.7 and 1.2 ppm had some form of dental fluorosis. Of these 22.5 % had very mild, 5.8% had mild, 1.3% had moderate and none were in the severe category.

As far as dental caries was concerned Brunelle & Carlos found that for children aged 5-17, who had lived all their lives in a fluoridated versus a non-fluoridated community, the average saving in tooth decay was 0.6 of one tooth surface (see their Table 6). There are 4 and 5 surfaces for the “cutting” and “chewing” teeth respectively, and by the time all the child’s teeth have erupted there are a total of 128 tooth surfaces. Even this very modest saving of 0.6 of one tooth surface was not shown by the authors to be statistically significant, but this did not stop them declaring:

“The results show that water fluoridation has played a dominant role in the decline of caries and must continue to be a major prevention methodology.”

Brunelle and Carlos also noted that, “Contrary to some earlier observations, however, white children had lower mean DMFS scores than non—whites (blacks and all others) at most ages (Fig. 7).”

1988

In 1988, Bette Hileman, in an important review in Chemical and Engineering News reported disagreements among dental researchers as to whether dental fluorosis rates were increasing among children in the U.S.
“Dennis Leverett, chairman of the department of community dentistry at the Eastman Dental Center in Rochester, N.Y., claims that the prevalence of dental fluorosis today in communities with fluoridated water is twice the level that H. Trendley Dean, a dental surgeon in the Public Health Service, reported in 1942 ... In contrast, William S. Driscoll, acting chief of the disease prevention and health promotion branch at the National Institute of Dental Research (NIDR), and his coworkers report that surveys in 1980 “suggest that no important changes in the prevalence and severity of fluorosis have taken place” since Dean’s studies. However, Driscoll did find eight children with either moderate or severe fluorosis in a community with a fluoride level of 1 ppm…” (Hileman, 1988)

1990

In 1990, Williams and Zwemer in a study from Georgia, reported that dental fluorosis was more severe among African-American children than white children. As the following table shows, 16.7% of black children in Augusta, Georgia had moderate/severe fluorosis versus 9.1% of white children. In Richmond County, the respective rates were 3.3% vs 0% (see Table 1)

<table>
<thead>
<tr>
<th>Residence/Race</th>
<th>No Fluorosis (TSIF Score = 0)</th>
<th>Very Mild/Mild Fluorosis (TSIF Score = 1 - 3)</th>
<th>Moderate/Severe Fluorosis (TSIF Score = 4 - 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>City/Black</td>
<td>19.6%</td>
<td>63.7%</td>
<td>16.7%</td>
</tr>
<tr>
<td>City/White</td>
<td>18.2%</td>
<td>72.7%</td>
<td>9.1%</td>
</tr>
<tr>
<td>County/Black</td>
<td>47.8%</td>
<td>48.9%</td>
<td>3.3%</td>
</tr>
<tr>
<td>County/White</td>
<td>44.9%</td>
<td>55.1%</td>
<td>0%</td>
</tr>
</tbody>
</table>

Table 1. Dental Fluorosis Rates in Augusta & Richmond County, Georgia

In 1990, the long-awaited animal cancer study (requested by Congress) was published by the National Toxicology Program (NTP, 1990). This report caused great consternation because the authors reported a statistically significant increase in a bone cancer (osteosarcoma) in the male rats, which was "equivocal" evidence that fluoride was carcinogenic.

1991

Soon after the 1990 NTP study was published a cover story was published in the *Journal of the American Dental Association* speculating that fluoridation may actually be protective against cancer (McGuire et al., 1991). It was clear from the comments in this article that the authors were more worried that a finding that fluoride caused cancer would end water fluoridation, than it might be killing a few young men each year. They wrote:

"An incorrect inference implicating fluoride carcinogenicity and its removal from our water systems would be detrimental to the oral health of most Americans...a disruption in the delivery of fluoride through municipal water systems would increase decay rates over time...Linking of fluoride ingestion and cancer initiation could result in a large-scale defluoridation of municipal water systems under the Delaney clause." (Connett et al., 2010, p. 187)

One of the authors of this report was Professor Chester Douglass, chairman of the Harvard dental department. In 1994 he received a large grant from the National Institute of Environmental Health Sciences to investigate the possible connection between fluoridation and osteosarcoma. This raises serious questions about why an investigation that had the potential to end fluoridation was given a) to a dental school and b) to a dental professor who was known to be pro-fluoridation and was simultaneously a consultant for Colgate (FAN, 2006).

Despite these doubts in 2001, Douglass’s graduate student, Elise Bassin, as part of her doctoral thesis, discovered in a carefully matched case control study that young boys exposed to fluoridated water in their 6th, 7th or 8th years had a 5-7-fold increased risk of succumbing to osteosarcoma by the age of 20. Over the next three years, Douglass – given several opportunities -- hid this finding from his peers, his funders and the National Research Council of the National Academies (NRC) review panel. Bassin’s thesis (2001) was not “found” until 2004. For the rest of this intriguing story see the Harvard/Bone Cancer files (FAN, 2006); Harvard Crimson, 2006; Connett et al., 2010, chapter 18.

1997

Heller et al. (1997) paper published (see above)

1997 also saw the publication of a controversial report from the Institute of Medicine (IOM). The title of the report included fluoride in a list of well-known nutrients needed for healthy bone growth: calcium, magnesium, phosphate and vitamin D (IOM, 1997). In response to a letter from a number of scientists complaining about this false classification of fluoride as a nutrient, Dr. Bruce Alberts, President of the National Academies, and Dr.
Kenneth Shine, President of the IOM, wrote:

First, let us reassure you with regard to one concern. Nowhere in the report is it stated that fluoride is an essential nutrient. If any speaker or panel member at the September 23rd workshop referred to fluoride as such, they misspoke. As was stated in Recommended Dietary Allowances 10th Edition, which we published in 1989: “These contradictory results do not justify a classification of fluoride as an essential element, according to accepted standards. Nonetheless, because of its valuable effects on dental health, fluoride is a beneficial element for humans.” (Alberts and Shine, 1998).

1999 - 2000

Kumar et al. (1999) reported

“African-American children studied [in Newburgh and Kingston, NY] in 1995 were at higher risk for dental fluorosis than children of other racial groups… The higher risk for dental fluorosis observed among African-American children is consistent with several other studies.”

In 2000 Kumar et al. noted,

“The results support our earlier findings that African-American children were at higher risk for dental fluorosis in the fluoridated area. Even in the nonfluoridated area, there was a suggestion that African-American children were at higher risk. Whether this higher risk for African-American children is the result of their lower threshold for fluoride or due to other unknown sources of fluoride is not known. It has been reported that African-American children in the United States drink more water and less milk compared to white children. In Newburgh, this difference in the fluid consumption may have resulted in a higher prevalence of fluorosis in African-American children… Because a race fluorosis association could have important policy implications, a large-scale study in a representative sample should be conducted to test specifically the hypothesis that African-American children are at higher risk for fluorosis.”

2003 - 2006

The US EPA Office of Water asked the National Research Council of the National Academies to review their safe water standards for fluoride. A 12-membered panel (unusually for official reviews on fluoride, the panel was balanced with 3 pro-fluoridation, 3 anti-fluoridation and 6 undeclared) was appointed by the National Research Council of the National Academies to do this. The panel reported back in 2006 with a landmark 500-page review (NRC, 2006).

The NRC panel concluded that the safe drinking water goal and standard for fluoride in water (MCLG and MCL) of 4 ppm was not protective of health and a new risk assessment needed to be performed to determine a new MCLG (maximum contaminant level goal).

The panel had this to say on dental fluorosis:

“Severe enamel fluorosis is characterized by dark yellow to brown staining and discrete and confluent pitting, which constitutes enamel loss... Severe enamel fluorosis compromises that health-protective function by causing structural damage to the tooth.

The damage to teeth caused by severe enamel fluorosis is a toxic effect that is consistent
with prevailing risk assessment definitions of adverse health effects...

“Severe enamel fluorosis occurs at an appreciable frequency, approximately 10% on average, among children in U.S. communities with water fluoride concentrations at or near the current MCLG [maximum contaminant level goal] of 4 mg/L. Thus, the MCLG is not adequately protective against this condition...

“The committee finds that it is reasonable to assume that some individuals will find moderate enamel fluorosis on front teeth to be detrimental to their appearance and that it could affect their overall sense of well-being. However, the available data are not adequate to categorize moderate enamel fluorosis as an adverse health effect on the basis of structural or psychological effects.

“Since 1993, there have been no new studies of enamel fluorosis in U.S. communities with fluoride at 2 mg/L in drinking water. Earlier studies indicated that the prevalence of moderate enamel fluorosis at that concentration could be as high as 15%...” (NRC, 2006)

However, even though the NRC panel concluded that severe dental fluorosis constituted an adverse health effect no federal or state agency has gone to any lengths to inform the public that this is the case. Nor have they warned the African-American and Mexican-American communities with a total population of 101 million people (Colby & Ortman, U.S. Census, Table 2, 2015) that they are particularly vulnerable to this condition.

2005

In 2005, the Centers for Disease Control and Prevention (Beltrán-Aguilar et al. See Table 2 below) acknowledged for the first time publicly that the black community has higher rates of dental fluorosis than the white community. It took a Freedom of Information Act request, however, to learn the full extent of this disparity. 58% of black children were diagnosed with dental fluorosis in CDC’s 1999-2004 national survey, versus 36% of white children. (Gracia, 2011; see also Stockin, 2015).

Table 2: A copy of Table 23. Enamel fluorosis* among persons aged 6-39 years, by selected characteristics United States, National Health and Nutrition Examination Survey, 1999-2002.

Source: Beltrán-Aguilar et al., 2005 (CDC, 2005)
According to attorney Michael Connett and Special Projects Director for the Fluoride Action Network (FAN):

“The epidemic of fluorosis now seen in the black community is the visible legacy of the government’s failure to act on what it knew. They knew in 1962 that ‘negroes in Grand Rapids had twice as much [dental] fluorosis than others’ (Maier, 1962).”

2010

In 2010 another report from the CDC revealed that 41% of U.S. children between the ages of 12 and 15 had some form of dental fluorosis. This total included children from both fluoridated and non-fluoridated communities. No breakdown was given for race and ethnicity. A breakdown of the 41% total showed that 28.5 % has very mild, 8.6% had mild, and 3.6 % had either moderate or severe dental fluorosis (Beltrán-Aguilar et al., 2010).
2011

On January 7 the U.S. Department for Health and Human Services and EPA held a joint press conference in Washington, DC (HHS, 2011a). The HHS announced its proposal to lower its recommended fluoride level in water to fight tooth decay from a range of 0.7 – 1.2 ppm to 0.7 ppm, largely because of the escalating prevalence of dental fluorosis among US children.

At this same press conference EPA’s Office of Water announced that it had begun its determination of a new safe drinking water standard for fluoride (recommended by the NRC panel in March of 2006). While stating that they wanted to find a safe level for fluoride in drinking water (their federal responsibility), they also stated that they were interested in protecting children’s teeth (not their federal responsibility). According to EPA Assistant Administrator for the Office of Water Peter Silva.

“‘EPA’s new analysis will help us make sure that people benefit from tooth decay prevention while at the same time avoiding the unwanted health effects from too much fluoride (HHS, 2011a).’” (our emphasis)

EPA at this juncture threw away its objectivity in the setting of a “safe” Maximum Contaminant

Level goal (MCLG) for fluoride in drinking water. In other words they were indicating that they were going to select the safe level for fluoride as a contaminant that would not conflict with the HHS recommended level for fluoride in the fluoridation program. Clearly that is a political judgment. However, from a legal point of view no consideration of any perceived benefit of a contaminant should be allowed to interfere with the EPA’s obligation to determine a safe Maximum Contaminant Level Goal (MCLG). According to the Safe Drinking Water Act the MCLG should be determined based on a known or reasonably anticipated harmful effect, with appropriate safety factors applied to protect everyone in society, including vulnerable subsets. Such calculations should be scientifically determined and should not be compromised by accommodating some perceived benefit.

2015

The HHS formally announced its new recommended level of 0.7 ppm fluoride in water claiming that it would lower tooth decay, while minimizing the prevalence of the more objectionable stages of dental fluorosis (HHS, 2015). In so doing they continued their 60-year plus denial of any other potential health effect other than dental fluorosis at the doses experienced by any American, including the most vulnerable, drinking fluoridated water and getting fluoride from other common sources such as dental products (see section 25 for our response to this).

SUMMARY: DENTAL FLUOROSIS IN THE U.S. 1945--2017

In 1945, the “father of fluoridation” H. Trendly Dean estimated that about 10% of children would develop dental fluorosis in communities fluoridated at 1 ppm. Since then children are being exposed to fluoride not only in fluoridated water but also from all the beverages and processed foods made with fluoridated water, and from many other sources including dental products, and pesticide residues on food, including EPA permitted fluoride residues – from the fumigant sulfuryl fluoride – of 900 ppm fluoride in powdered eggs, 130 ppm fluoride in wheat flour, and 70 ppm fluoride in 99.99% of all processed food (FAN, 2005). As a result the rates of dental fluorosis are getting significantly worse across the U.S. However, the CDC’s Division of Oral Health continues to promote artificial water fluoridation despite its disproportionate impact on communities of color and low-income groups. Studies sponsored by this CDC division in 2005 and 2007 confirm the growing epidemic of dental fluorosis in minority populations. It is an open question as to whether reducing the fluoride levels from a range of 0.7 to 1.2 ppm across the country to a single value of 0.7 ppm, will have a major effect on decreasing the prevalence of this condition in general or in minority communities in particular. A larger question is whether the level of 0.7 ppm will cause other health problems, but for the CDC’s Division of Oral Health that is a mute question since they adamantly deny that any other tissue is harmed by water fluoridation or from all sources combined.

Meanwhile, at no time have federal government officials ever taken steps to warn Black communities of their heightened fluorosis risk.
Figure 4: Dental fluorosis rates in the United States: 1950 through 2004 (FAN).

* Graph by Fluoride Action Network

Figure 5: What was predicted in 1950 (top) vs. What has actually occurred (bottom).


Fluoride and Dental Fluorosis, an Environmental Justice Issue

Data have consistently shown that dental fluorosis is more prevalent in minority (African-American and Mexican-American) communities and in low-income communities. Certain minority groups and people of lower SES have statistically greater intakes of fluoride, and increased rates of dental fluorosis (including the more severe forms) than do Whites or people of higher SES (Beltrán-Aguilar et al., 2005).

According to H. Trendley Dean, moderate dental fluorosis discolors and disfigures 100% of the tooth enamel. Moderate and severe dental fluorosis combined currently impacts 3.6% of all American children aged 12-15 (Beltrán Aguilar et al., 2010). NRC (2006) states “the committee finds that it is reasonable to assume that some individuals will find moderate enamel fluorosis on front teeth to be detrimental to their appearance and that it could affect their overall sense of well-being.” (p. 5). According to NRC (2006), “only 24.2% of parents were satisfied with the color of their children’s teeth when the TSIF score was 4 or greater (moderate or severe dental fluorosis), versus. 73.9% satisfaction with not dental fluorosis.” An ad-hoc panel of behavioral scientists convened by the U.S. EPA and the National Institute of Mental Health in 1984 to evaluate the psychological impacts of fluorosis concluded that “individuals who have suffered impaired dental appearance as a result of moderate and severe fluorosis are probably at increased risk for psychological and behavioral problems or difficulties” (Kleck RE, cited in 50 FR 20164, EPA, 1985; NRC, 2006, p. 119).

Racial data on disparate rates of fluorosis have been remarkably consistent.

- In 1962, F.J. Maier, a top PHS official involved with the first fluoridation trial, reported, “negroes in Grand Rapids had twice as much [dental] fluorosis than others.”

- In 1985, Butler et al revealed that the prevalence of dental fluorosis among African-American children was greater than for Hispanic and non-Hispanic white children in Texas. The reported Odds Ratio was 2.3. In 1990, Zwemer reported that dental fluorosis was more severe among African-American children than white children in Georgia.

- In 1990 Williams and Zwemer reported that dental fluorosis was more severe among African-American children than white children in Georgia.

- In 1997, Heller study (on the 86-87 NIDR data) showed that African-Americans had roughly 20% more fluorosis than whites.

- In 1999, Kumar reported “African-American children studied [in Newburgh and Kingston, NY] in 1995 were at higher risk for dental fluorosis than children of other racial groups... The higher risk for dental fluorosis observed among African-American children is consistent with several other studies”.

- In 2005, CDC acknowledged for the first time publicly that the black community has higher rates of dental fluorosis than the white community (Beltran-Aguilar, 2005).

- In 2010, the CDC/NHANES reported that 41% of U.S. adolescents have dental fluorosis (Beltran-Aguilar, 2010)

- In 2010, Martinez-Mier found differences in the rates and severity of fluorosis between whites and African-Americans, with African-Americans experiencing higher rates of...
fluorosis.

- In 2012, CDC/NHANES data (unpublished) revealed that 58% of U.S. adolescents had dental fluorosis, with African-Americans still experiencing fluorosis at a higher rate than whites.

However, in all this time neither the CDC nor any other federal agency that promotes water fluoridation has sought to warn communities of color of their particular vulnerability with respect to this permanent visually objectionable injury from systemic exposure to fluoride. Nor have they indicated what this means: their children have been over-exposed to fluoride before their permanent teeth have erupted and this over-exposure might indicate they have been damaged in other ways. This failure to warn communities of color of this problem is a clear example of environmental injustice.

Fluoride and Nutrition, an Environmental Justice Issue

Poor nutrition frequently occurs among low-income families. As inadequate nutrition increases the toxicity of fluoride, low-income children and adults are more susceptible to the detrimental effects of fluoride exposure. As with African-Americans, low-income children have been found to consume significantly more total fluids and plain water, and thus receive more fluoride from drinking water, than higher-income children (Sohn et al., 2001). Low-income families also consume substantially less fresh fruits and vegetables and thus more processed foods than other groups. Most vegetables have a relatively low (<0.5 ppm) fluoride concentration (EPA, 2010b, p. 21), while processed foods including mechanically deboned chicken, contain higher levels of fluoride.

The inadequate diet often common in low-income families includes reduced calcium and iodine intakes, which are known to increase the toxicity of fluoride. For example, participants in the Food Stamp Program consumed a significantly smaller percentage of the AI for calcium than did higher-income non-participants (73% versus 83% of AI) (Fox and Cole, 2004). This is what was said about this issue in a 1952 article that appeared in the Journal of the American Dental Association:

“The data from this and other investigations suggest that malnourished infants and children, especially if deficient in calcium intake, may suffer from the effects of water containing fluorine while healthy children would remain unaffected…Thus low levels of fluoride ingestion which are generally considered to be safe for the general population may not be safe for malnourished infants and children. Therefore, the nutritional status must be carefully assessed and guarded in areas with endemic fluorosis. Nutritional studies should be included in any comprehensive program of fluoridation of water with special attention to chronically ailing infants and children”. (Massler & Schour, 1952).

Also of concern is the inability of low-income families living in fluoridated communities to provide low-fluoride or fluoride-free water to reconstitute infant formula. Low-income families are likely not able to afford expensive filtration systems to remove fluoride from tap water, nor are they likely able to afford bottled water containing low or no fluoride.

While breast-feeding is encouraged as the best source of nutrition for infants, a substantial proportion of American children receive their nutrition solely from milk or soy-based infant formulas. In fact, at 6 months of age, less than 15% of infants in the U.S. are exclusively

breast-fed (CDC, 2010). When concentrated formula, which already contains up to 0.3 mg F/L (NRC, 2006) is reconstituted with fluoridated water containing up to 1.2 mg F/L, these babies could potentially receive more than 200 times more fluoride than a breast-fed infant, whose mother’s milk contains only 0.007 (average in fluoridated and non-fluoridated communities). If a 3 kg newborn consumes ~0.5 L formula per day, the amount of formula recommended by the American Academy of Pediatrics (2010; 75 ml per 0.45 kg body weight), the water used to make that formula could contain no more than 0.2 mg F/L to ensure that the RfD of 0.08 is not exceeded. Presently, a 3 kg newborn receiving 0.5 L soy-based formula (containing up to 0.3 mg F/L) reconstituted with “optimally” fluoridated water (containing 1.2 mg F/L) could receive up to 0.25 mg F/kg/day. This is 25 times the AI set forth by IOM (1997), nearly 4 times the upper limit set forth by IOM (1997), and more than 3 times the EPA RfD for fluoride.

In general, the EPA has failed to consider the disproportionate harm to people with inadequate nutrition in its risk assessment of fluoride. EPA has ignored that fluoride toxicity may be exacerbated by poor nutrition, including deficiencies in iodine, calcium, magnesium, vitamin C (ATSDR, 1993, p.112), selenium, and vitamin D (e.g. ATSDR, 1993, p.112; NRC, 2006). Poor nutrition has been found to increase the incidence and severity of dental fluorosis (Pandit et al., 1940; Murray et al., 1948; Littleton et al., 1999) and skeletal fluorosis (Pandit et al., 1940; Marier et al., 1963; Fisher et al., 1989; Teotia et al., 1984; Littleton et al., 1999).

The dose of fluoride at which disturbed endocrine function occurs is reduced in situations of iodine deficiency (NRC, 2006). Lin et al. (1991), in a UNICEF-sponsored study, found that even modest levels of fluoride in the water (0.88 mg/L vs. 0.34 mg/L) resulted in reduced IQ and increased frequency of hypothyroidism when combined with low iodine, even more so than with iodine deficiency alone. Moreover, the increasing dietary intake of fats in the U.S. may have negative repercussions in terms of fluoride metabolism, as “Diets high in fat have been reported to increase deposition of fluoride in bone and, thus, to enhance toxicity” (HHS, 1991).

Yet the EPA has consistently ignored this susceptible subpopulation in its assessment of fluoride harm. This is another example of failing to comply with EPA’s stated goal of achieving Environmental Justice “for all communities and persons across this Nation” (EPA, 2011a).

Fluoride and Lead, an Environmental Justice Issue

Elevated blood lead levels are linked to developmental delays in children under age six and fetuses. Lead can adversely affect almost every organ and system in the body. The most sensitive is the central nervous system, particularly in children. Lead also damages kidneys and the reproductive system. The effects are the same whether it is breathed or swallowed.

Lead exposure and lead poisoning have been concerns for decades in African-American communities. The Huffington Post cites a CDC report that says that lead poisoning is a disease that primarily impacts African-Americans. According to the CDC (Jones et al.), children of color whose families are poor and who live in housing built before 1950 have the highest lead poisoning risk:

On average, between 1999 and 2004, **black children were 1.6 times** more likely to test positive for lead in their blood than white children. And among children who tested
positive for extremely high lead levels (≥10 micrograms per deciliter), the disparity was even more stark. Black children were nearly three times more likely than white children to have highly elevated blood---lead levels, the type of lead poisoning where the most damaging health outcomes occur. (Jones et al., 2009).

African-Americans in the inner city have had more exposure to lead than white children. In 1995 Stevens reported, “Of impoverished black children aged three to five living in American inner cities, 90% have elevated blood-lead levels.” CDC in 2003 stated, “Of the children reported with confirmed elevated [blood lead levels] between 1997 and 2001, approximately 17% were non-Hispanic whites, 60% were non-Hispanic blacks, 16% were Hispanic, and 7% were of other races or ethnicities. As reported by the MMWR in 2013:

“This report summarizes the results of that analysis, which indicated that the percentage of children aged 1–5 years with BLLs at or above the upper reference interval value of 5 µg/dL calculated using the 2007–2010 NHANES cycle was 2.6%. Thus, an estimated 535,000 U.S. children aged 1–5 years had BLLs ≥5 µg/dL based on the U.S. Census Bureau 2010 count of the number of children in this age group.” (MMWR, 2013)

**Combined Lead and fluoride exposure**

In a study by Leite et al. (2011), the authors found that rats treated with both lead and fluoride had worse dental fluorosis than rats treated with fluoride alone. Thus it is possible that children with lead exposure will be more susceptible to developing dental fluorosis.

There are other experiments that have exposed animals to a combination of lead and fluoride. These have reported the following:

- Liu et al. (2008) reported that co-exposing rat pups to lead and fluoride resulted in “alterations in testis morphology and sperm quality, including low viability and high abnormality, thereby suggesting that disturbance of energy metabolism may be one of the mechanisms by which F or Pb affects the male reproductive system.”

- In the animal study cited above by Leite et al. (2011), rats treated with both lead and fluoride had worse dental fluorosis than rats treated with fluoride alone.

- Niu et al. (2009) rat study: “Results showed that the learning abilities and hippocampus glutamate levels were significantly decreased by F and Pb individually and the combined interaction of F and Pb. The activities of AST and ALT (markers of lead toxicity) in treatment groups were significantly inhibited, while the activities of GAD were increased, especially in rats exposed to both F and Pb together. These findings suggested that alteration of hippocampus glutamate by F and/or Pb may in part reduce learning ability in rats.”

- Niu et al. (2008) study with adult rats: “From results of the Y---maze test, we can see a significant decrease in learning ability of animals in the HiF+HiPb (High fluoride with high lead) group.”

- Sawan et al. (2010) study with rats: “These findings show that fluoride consistently increases BPb and calcified tissues Pb concentrations in animals exposed to low levels of lead and suggest that a biological effect not yet recognized may underlie the epidemiological association between increased BPb lead levels in children living in water-fluoridated communities.”

- Panov et al. (2015) reported the following from a study where rats were exposed to both fluoride and lead:

- Comparison of the values obtained for the groups of separate and combined exposure shows that, for the majority of the toxicodynamic indices, the combined effect is more marked than the effect of fluoride alone or lead alone.

- With a combined exposure of lead and fluoride (but not alone) significant reduction in the thyrotropin level was observed. Thyrotropin is a hormone secreted by the pituitary gland that regulates the production of thyroid hormones.

- Neither fluoride nor lead produced a reduction in triiodothyronine level, but it was reduced under the combined effect (i.e. overt synergism took place). On the contrary, at exposure to lead alone or in combination with fluoride the level of thyroxine was raised.

In addition to the interaction between lead and fluoride is the additional problem that the chemicals used to fluoridate water appear to interact with chloramine (a common disinfection agent) to increase the dissolution of lead from brass fittings. Some of the first indications that fluoride chemicals could leach lead into water came in the early 1990s when water departments in Maryland (Robb, 1994) and Washington (TPU, 1992) noticed significant drops in water lead levels immediately after terminating their fluoridation programs.

The first two studies to investigate this issue analyzed the blood lead levels of almost 400,000 children living in areas with and without fluoridated water in New York and Massachusetts (Masters 1999, 2000). These studies found that children living in areas with fluoride-treated water were at increased risk of having markedly elevated blood levels (>10 ug/dl). These peer-reviewed studies, conducted by Dartmouth professor Roger Masters and chemical engineer Myron Coplan, sent shock waves through the public health community. As Masters noted,

“If further research confirms our findings, this may well be the worst environmental poison since leaded gasoline.”

The CDC has conceded that, if research confirms the link between fluoridation and elevated lead exposure, fluoridation would need to end, noting that “efforts to prevent dental caries via the use of fluoridated drinking water should continue unless a causal impact of certain fluoridation methods on PbB [blood lead] concentration is demonstrated by additional research.” (Macek 2006)

After criticizing the methodology of Masters and Coplan’s studies, the CDC published an analysis of a smaller sample of 10,000 children from across the country, whose blood was measured for lead during the 1988-1994 National Health and Nutrition Examination Survey. (Macek 2006). The CDC study - which controlled for the key factors known to influence blood lead levels, including race/ethnicity, poverty status, and urbanicity - is sometimes touted as refuting the link between fluoridation and lead hazards, but a close look at its data shows that the study does little to dampen concern.
According to the CDC’s data, flurosilicic acid was associated with an elevated risk for high blood lead (> 5 ug/dl) in every single category of children identified by the CDC, even after controlling for the other key risk factors. Fluoride was associated with:

- **A 20% increased risk** (but not statistically significant) for high blood lead levels among children living in houses made prior to 1946;
- **A 40% increased risk** (but not statistically significant) for high blood lead levels among children living in houses made between 1946 and 1973;
- **A 70% increased risk** (but not statistically significant) for high blood lead levels among children living in houses made after 1974;
- **A 530% increased risk** (which was statistically significant) for high blood lead levels among children living in houses with unknown ages.

Since most of these elevated risks were not statistically significant, the CDC dismissed them as essentially a random fluke. However, the consistency in the direction of the risk, coupled with the statistically significant 530% increased risk for children in homes of unknown age, raises a serious red flag. Even the CDC has acknowledged that the study does not refute the connection between fluoridation and lead, and that “it is possible that larger samples might have identified additional, significant differences.” (Macek 2006)

In 2007, Coplan, along with Richard P. Maas, PhD and Steven C. Patch PhD, co-directors of the Environmental Quality Institute at the University of North Carolina, re-analyzed CDC’s data by placing all children exposed to flurosilicic acid and sodium fluorosilicate in one group (“silicofluorides”) and all other children in another group. The authors again found that the children exposed to silicofluoridated water had a significantly elevated risk of having high blood lead levels (Maas et al., 2007). According to the re-analysis, children from the silicofluoridated communities had a 20% greater risk of having blood lead levels in excess of 5 ug/dl. Maas’ team estimated that the risk for exceeding the 10 ug/dl threshold would be even greater.

Importantly, the authors reported that a combination of chloramines and flurosilicic acid, especially with extra amounts of ammonia, leaches lead from meters, solder and plumbing systems (Maas et al., 2007), stating:

“Tests showed lead levels three and four times higher in water with that combination of chemicals ... About 500 systems, across the country, have switched to chloramine treatment since 2001...and most also use fluorosilicic acid.”

Maas et al. explained that flusilicic acid “does not leach lead simply because it is an acid”. It may also leach lead due to its “unique affinity for lead”. In carefully controlled laboratory experiments, the authors found that flusilicic acid can increase the leaching of lead in non-acidic waters (pH = 8), even from common brass fixtures, like faucets, that contain small quantities of lead (Maas et al., 2007).

The North Carolina study’s findings demonstrate that - despite theoretical arguments to the contrary (Urbansky-Schock, 2000) - highly diluted levels of FSA can leach lead from pipes and common brass fixtures, *even in pH-adjusted water*, and this effect can be unpredictably amplified in the presence of other common water treatment chemicals.

In spite of the examples above, as far we know, no federal agency has yet published anything on the synergistic effects of exposure to fluoride and lead. The Agency for Toxic
Substances and Disease Registry (ATSDR, 2004) produced an “interaction profile” to exposures of the mixture containing uranium, fluoride, cyanide and nitrate. However, no information was available on any interaction.

Given the potential synergistic effect of combined fluoride and lead, and given African-American’s susceptibility to lead exposure, water fluoridation is tantamount to an environmental injustice in the Black community.

**Fluoride and Mercury, an Environmental Justice Issue**

According to Kaste et al. (1996), national data indicate that 80% of tooth decay in children is concentrated in 25% of the child population, with low-income children and racial/ethnic minority groups having more untreated decay on average than the U.S. population as a whole. This means that they also have greater exposure to mercury via mercury amalgam fillings.

According to the Food & Drug Administration,

Dental amalgam is a mixture of metals, consisting of liquid (elemental) mercury and a powdered alloy composed of silver, tin, and copper. **Approximately 50% of dental amalgam is elemental mercury by weight.** The chemical properties of elemental mercury allow it to react with and bind together the silver/copper/tin alloy particles to form an amalgam.

Dental amalgam fillings are also known as “silver fillings” because of their silver-like appearance. Despite the name, “silver fillings” do contain elemental mercury (FDA, 2015).

According to Counter & Buchanan (2011), “Children are particularly vulnerable to Hg intoxication, which may lead to impairment of the developing central nervous system, as well as pulmonary and nephrotic damage...” Exposures from dental amalgams “release Hg vapors, and Hg2+ in tissues... [and] fetal/neonatal Hg exposure from maternal dental amalgam fillings.” The authors state:

It has been known for sometime that dental amalgam is a major source of Hg\(^0\) (elementary mercury) exposure in humans because Hg is the principal metal in most dental fillings (approximately 50% Hg by weight) (Nadarajah et al., 1996). The health effects of dental amalgam Hg have been a subject of considerable debate for years, with no scientific consensus on an association between amalgam Hg exposure and adverse health consequences, either in adults or children (Clarkson, 2002; Ratcliffe et al., 1996). However, questions have been raised regarding a possible association between maternal Hg dental fillings and the health of the developing fetus, neonate, and infant. Significant levels of Hg have been measured in oral vapor, blood, and in organs of animals and humans with Hg containing dental amalgam restorations (Abraham et al., 1984; Snapp et al., 1989; Vimy et al., 1990, 1997). In the oral cavity, Hg\(^0\) vapor is rapidly oxidized to inorganic divalent Hg (Hg2+) in vivo after release from dental amalgam and absorbed through inhalation.

As shown above, children from low-income families are more likely to get mercury amalgam fillings than families with higher income. Mercury is neurotoxic. The combined impact of mercury and fluoride on a child’s mental development may be greater than either acting alone.
Fluoride and Kidneys, an Environmental Justice Issue

The kidneys are responsible for excreting most of the fluoride from the body (NRC, 2006), which is about 50% of ingested fluoride in a healthy person. Kidneys may be exposed to fluoride concentrations five times greater than other soft tissues (Whitford, 1996), as these organs concentrate fluoride as much as 50-fold from plasma to urine (NRC, 2006). Indeed, with the exception of the pineal gland, the kidneys accumulate more fluoride than any other soft tissue in the body (Hongslo et al., 1980; Ekstrand, 1996; Whitford, 1996). As such, the potential for fluoride-induced damage to the kidneys is likely greater than it is for most other soft tissues (NRC, 2006).

The National Kidney Foundation (NKF) provides sobering statistics on the prevalence of kidney disease in African-Americans. NKF reports that:

- Due to high rates of diabetes, high blood pressure and heart disease, Blacks and African-Americans have an increased risk of developing kidney failure.

- Blacks and African-Americans suffer from kidney failure at a significantly higher rate than Caucasians - more than 3 times higher.

- African-Americans constitute more than 35% of all patients in the U.S. receiving dialysis for kidney failure, but only represent 13.2% of the overall U.S. population.

- Diabetes is the leading cause of kidney failure in African Americans. African Americans are twice as likely to be diagnosed with diabetes as Caucasians. Approximately 4.9 million African Americans over 20 years of age are living with either diagnosed or undiagnosed diabetes.

Indications of kidney damage have been associated with even relatively low fluoride exposures. Morphological changes were observed in the kidneys of rats drinking water with 1 mg F/L (McKay et al., 1957; Varner et al, 1998), a concentration that produces plasma fluoride concentrations equivalent to a human consuming <1.2 mg F/day (Teotia et al., 1978), or 0.017 mg F/kg/day for a 70 kg adult. Plasma fluoride levels in rats as low as 2 umol/L (equivalent to a human intake of perhaps 2.2 mg F/day; Teotia et al., 1978; NRC, 2006, p. 70) significantly decreased the amount of plasma membrane Ca++-pump protein in kidney membranes (Borke and Whitford, 1999). Among children, kidney damage has been revealed in a dose-dependent manner, with effects associated with water containing only 2.6 mg F/L (Liu et al., 2005). Adding to this weight of evidence is that kidney disease is often found to be co-morbid with skeletal fluorosis in humans (Jolly et al., 1980; Reggabi et al., 1984; Lantz et al., 1987; Ando et al., 2001).

Parameters of kidney function have been found to be altered at levels within the range of that proposed by EPA OW as the new fluoride RfD. For example, the amount of plasma membrane and endoplasmic reticulum Ca++-pump protein in kidney membranes of rats showed a significant reduction associated with a plasma concentration of only 2 umol F/L (Borke and Whitford, 1999). This level is equivalent to that achieved in humans with an intake of perhaps 2.2 mg F/day (Teotia et al., 1978; NRC, 2006, p. 70), or 0.03 mg/kg/day for a 70 kg adult.

For people whose kidney function is already impaired, fluoride toxicity can be exacerbated (ATSDR, 1993). Renal clearance of fluoride is dependent on pH and glomerular filtration rate.
(NRC, 2006, p.91). Those with impaired renal function are unable to excrete fluoride efficiently, and thus accumulate fluoride more quickly than a healthy individual (Johnson et al., 1979; Bober, 2006; NRC, 2006).

Although often overlooked, a paper by Mayo Clinic scientists Johnson, Jowsey and Taves (Fluoridation and bone disease in renal patients) is extremely important (Johnson et al., 1979). They showed that patients with long-term renal failure were getting bone damage when drinking water with relatively low levels of fluoride in their water. They conclude that:

“The available evidence suggests that some patients with long-term renal failure are being affected by drinking water with as little as 2 ppm fluoride. All of the patients showed increased bone density and two patients showed calcification of interosseous ligaments which is thought to be diagnostic of skeletal fluorosis. The average concentration of fluoride in bone of 4.4 moles of fluoride per 100 moles of calcium is equivalent to 9,000 ppm of fluoride on an ash weight basis and is in the middle range of the values that have been reported for advanced fluorosis. The excessive osteoid formation seen in these patients is probably accentuated by fluoride.

... The meaning of these findings for community fluoridation will depend on whether or not further work will clearly show adverse effects in patients with renal failure drinking water with a concentration of 1 ppm of fluoride and whether these effects can be easily avoided. The finding of adverse effects in patients drinking water with 2 ppm of fluoride suggests that a few similar cases may be found in patients imbibing 1 ppm, especially if large volumes are consumed, or in heavy tea drinkers and if fluoride is indeed the cause.”

For both infant and adult patients with end-stage renal disease (ESRD) undergoing long-term dialysis treatments, plasma fluoride levels were found to be elevated (Armstrong et al., 1980; Warady et al., 1989; al-Wakeel et al., 1997), a condition that when persistent could lead to renal osteodystrophy and other bone damage (Gerster et al., 1983; Pettifor et al., 1989). Fluoride can affect the bone in renal failure patients, for example, by interfering with bone mineralization and increasing osteoid content, and may interact with aluminum to exacerbate osteomalacic lesions (Ng et al., 2004). As mentioned above, African-Americans constitute more than 35% of all patients in the U.S. receiving dialysis for kidney failure.

For these reasons, The National Kidney Foundation recommends that “individuals with CKD (chronic kidney disease) should be notified of the potential risk of fluoride exposure” (NKF, 2008). The same year, The National Kidney Foundation withdrew its official support for community water fluoridation.

That fluoride’s effect on the kidneys can lead to a greater harm to African-Americans constitutes an environmental injustice. As such, EPA would be wise to consider the potential harm to those with kidney dysfunction, and especially African-Americans, in its fluoride risk assessments.

**Fluoride and Diabetes, an Environmental Justice Issue**

Diabetes mellitus is a potentially life-threatening disease, in which the body fails to properly regulate blood sugar levels. Diabetes mellitus affects nearly 26 million people in the U.S. alone—with 7 million of these remaining undiagnosed, and therefore untreated.
Fluoride has been shown to increase blood glucose levels and impair glucose tolerance, likely by inhibiting insulin production or secretion. Impaired glucose tolerance, often a precursor to type 2 diabetes, has been found to occur in humans with fluoride intakes of only 0.07-0.4 mg/kg/day—a dose that can be reached in areas of “optimally” fluoridated water. Current fluoride intake, therefore, may contribute or exacerbate some types of diabetes. According to the National Research Council (2006), “any role of fluoride exposure in the development of impaired glucose metabolism or diabetes is potentially significant.”

In response to elevated blood sugar and increased frequency of urination, diabetics (especially those with untreated or poorly controlled diabetes) drink significantly more water than nondiabetics, and may consume more fluoride on a daily basis from water and other beverages. Furthermore, research has found that diabetics have a reduced capacity to clear fluoride from the body, (Hanhijarvi 1975), which may be a result of the kidney damage (nephropathy) that can accompanies diabetes. As noted in one review, “subjects with nephropathic diabetes can exhibit a polydipsia-polyurea syndrome that results in increased intake of fluoride, along with greater-than-normal retention of a given fluoride dosage.” (Marier 1977)

The increased exposure and retention of fluoride places diabetics at increased risk for fluoride-related toxicity. In animals with type-1 diabetes, for example, fluoride has been found to increase vascular contractions (Hattori et al, 2000), possibly contributing to an already elevated risk for cardiovascular disease in diabetics. This renders diabetics as a sensitive subpopulation with regard to fluoride toxicity.

According to The National Kidney Foundation, diabetes is the leading cause of kidney failure in African-Americans. African-Americans are twice as likely to be diagnosed with diabetes as Caucasians. Approximately 4.9 million African Americans over 20 years of age are living with either diagnosed or undiagnosed diabetes.

The most common type of diabetes in African Americans is type-2 diabetes. The risk factors for this type of diabetes include: family history, impaired glucose tolerance, diabetes during pregnancy, hyperinsulinemia and insulin resistance, obesity and physical inactivity. African Americans with diabetes are more likely to develop complications of diabetes and to have greater disability from these complications than Caucasians. African Americans are also more likely to develop serious complications such as heart disease and strokes.

Given that minority communities have a greater incidence of diabetes, some forms of which lead to an increased consumption of water, which in turns leads to a greater consumption of fluoride, water fluoridation would be yet another example of an environmental injustice.

Fluoride and Thyroid, an Environmental Justice Issue

Thyroid impairment is increasingly prevalent in the U.S. According to the American Thyroid Association (ATA, 2003), 2-3% of Americans have pronounced hypothyroidism, and as many as 10-15% have subclinical hypothyroidism. More recently, the American Association of Clinical Endocrinologists reported in 2010 that approximately 27 million Americans are experiencing thyroid disorder, making thyroid disease the leading endocrine disorder in the United States. Prescription drug data reflect this reality. WebMD reports in 2015 that Synthroid (levothyroxine), which treats hypothyroidism, has been the top-prescribed medicine in U.S. for several years.
Fluoride’s classification as an endocrine disruptor has been well established. The 2006 NRC panel labeled fluoride an endocrine disruptor. The authors state:

“The chief endocrine effects of fluoride exposures in experimental animals and in humans include decreased thyroid function, increased calcitonin activity, increased parathyroid hormone activity, secondary hyperparathyroidism, impaired glucose intolerance, and possible effects on the timing of sexual maturity. Some of these effects are associated with fluoride intake that is achievable at fluoride concentrations in drinking water of 4 mg/L or less, especially for young children or for individuals with high water intake. (p. 8, NRC 2006)

“In summary, evidence of several types indicates that fluoride affects normal endocrine function or response; the effects of the fluoride--induced changes vary in degree and kind in different individuals. Fluoride is therefore an endocrine disruptor in the broad sense of altering normal endocrine function or response, although probably not in the sense of mimicking a normal hormone.” (p. 266, NRC 2006)


On thyroid function, the NRC panel reported:

“Fluoride exposure in humans is associated with elevated TSH concentrations, increased goiter prevalence, and altered T4 and T3 concentrations; similar effects in T4 and T3 are reported in experimental animals, but TSH has not been measured in most studies.” (p. 262)

The panel also indicated that affects on the thyroid have been observed at very low levels. They state that,

“In humans, effects on thyroid function were associated with fluoride exposures of 0.05-0.13 mg/kg/day when iodine intake was adequate and 0.01-0.03 mg/kg/day when iodine intake was inadequate (Table 8-2).” (p. 263, NRC 2006).

For example, several endocrine effects have been observed at fluoride doses at or below that being proposed by EPA OW as the new RfD. These include altered thyroid function (T4 and T3 concentrations) and elevated TSH concentrations at 0.05-0.1 mg/kg/day (0.03 mg/kg/day with iodine deficiency); elevated calcitonin concentrations at 0.06-0.87 mg/kg/day; goiter prevalence >20% at 0.07-0.13 mg/kg/day (>0.01 mg/kg/day with iodine deficiency); and impaired glucose tolerance at 0.07-0.4 mg/kg/day (NRC, 2006). To reach these dosages (which depend on bodyweight) it takes remarkably little fluoride. For those with borderline iodine deficiency it would only take the consumption of 0.1 to 0.3 mg of fluoride per day for a 10 kg infant and 0.7 to 2.1 mg/day for a 70 kg adult. These are easily exceeded in a fluoridated community. For someone whose iodine levels are adequate for a 10 kg infant it would take between 0.5 and 1.3 mg /day and for a 70 kg adult it would take 3.5 mg to 9.1 mg/day. The lower end of these ranges would be reached by some people in a fluoridated community.

Concerns about fluoride’s effect on the thyroid have been recently buttressed by new research conducted in the UK. Peckham et al., 2015 found that higher levels of fluoride in...
drinking water are a useful predictor of the prevalence of hypothyroidism. The authors noted that:

“Approximately, six million people (10%) in England live in areas where drinking water contains natural fluoride or which has been artificially fluoridated at a target concentration of 1 ppm (1 mg/L). Using prevalence data from the UK QOF, an analysis was undertaken to determine whether prevalence was affected by practice populations being situated in fluoridated areas at >0.7 mg/L and areas with lower levels of fluoride. While there are other sources of fluoride in people’s diet (e.g., tea), drinking water is the most significant source of ingested fluorides in the UK.” (Peckham et al, 2015)

They concluded:

“In many areas of the world, hypothyroidism is a major health concern and in addition to other factors—such as iodine deficiency—fluoride exposure should be considered as a contributing factor. The findings of the study raise particular concerns about the validity of community fluoridation as a safe public health measure.” (Peckham et al, 2015)

Peckham’s findings are not totally unexpected. Scientific and medical research stretching back to the 1920s has shown that fluoride can affect the thyroid. In fact from the 1930s to the 1950s doctors in Argentina, France and Germany used fluoride to lower thyroid function in hyperactive thyroid patients. The levels of fluoride used overlap with the levels of exposure known to occur in some people drinking artificially fluoridated water today (Galletti & Joyet, 1958).

Importantly, race may be a factor in sensitivity to certain thyroid diseases, which may make communities of color more vulnerable to fluoride’s impacts on thyroid function. As the Journal of the American Medical Association reports, race appears to be a factor in determining a person’s risk of developing autoimmune thyroid conditions (JAMA 2014).

Additionally, reduced thyroid function in pregnant women is linked to reduced IQ in their children and there is accumulating evidence that fluoride, at levels within the range to which fluoridated populations are exposed, is associated with lowered IQ. It is possible that fluoride’s effect on thyroid function might be the mechanism by which it lowers IQ, though more study is needed.

Fluoride and Pre-term Births, an Environmental Justice Issue

African-Americans experience a disparate rate of pre-term births, of which there is an association with water fluoridation. According to the CDC:

In 2012, preterm birth affected more than 450,000 babies—that’s 1 of every 9 infants born in the United States. Preterm birth is the birth of an infant before 37 weeks of pregnancy. Preterm--related causes of death together accounted for 35% of all infant deaths in 2010, more than any other single cause. Preterm birth is also a leading cause of long--term neurological disabilities in children. Preterm birth costs the U.S. health care system more than $26 billion in 2005.  
http://www.cdc.gov/reproductivehealth/MaternalInfantHealth/PretermBirth.htm

In November 2009, Hart et al. presented an abstract at the American Public Health Association on the “Relationship between municipal water fluoridation and preterm birth in Upstate New York.” In part, the authors stated:

The annual incidence of preterm birth (PTB) (<37 weeks gestation) in the United States is approximately 10% and is associated with considerable morbidity and mortality. Current literature suggests an association between periodontal disease and PTB. Domestic water fluoridation is thought to have lessened the burden of dental disease. Theoretically, one would expect water fluoridation to be protective against PTB. The aim of our study was to examine the relationship between municipal water fluoridation and PTB.

Domestic water fluoridation was associated with an increased risk of PTB (9545 (6.34%) PTB among women exposed to domestic water fluoridation versus 25278 (5.52%) PTB among those unexposed, p < 0.0001). This relationship was most pronounced among women in the lowest SES groups (>10% poverty) and those of non-white racial origin. Domestic water fluoridation was independently associated with an increased risk of PTB in logistic regression, after controlling for age, race/ethnicity, neighborhood poverty level, hypertension, and diabetes (Hart et al., 2009).

In 2013, the Henry J. Kaiser Family Foundation reported that non-Hispanic blacks had the highest rate for "Preterm Births as a Percent of All Births by Race/Ethnicity." [http://kff.org/other/state-indicator/preterm-births-by-raceethnicity/](http://kff.org/other/state-indicator/preterm-births-by-raceethnicity/)

Why Are African-Americans More Sensitive To Fluoride’s Toxicity?

Officials in the US Public Health Service knew as early as 1962 that African-Americans had a higher prevalence of dental fluorosis than whites. Dental researchers have continued to report this over many decades. The National Research Council 1993 Review (NRC, 1993) reported four earlier studies showing that ethnicity plays a role in the effects of fluoride:

- Russell (1962), in the Grand Rapids fluoridation study, noted that fluorosis was twice as prevalent among African-American children as white children.
- In the Texas surveys in the 1980s, the odds ratio for African-American children having dental fluorosis, compared with Hispanic and non-Hispanic white children, was 2.3 (Butler et al., 1985).
- Dental fluorosis also tended to be more severe among African-American children than white children in the Georgia study (Williams and Zwemer, 1990), although the difference was not statistically significant.
- In Kenya, prevalence and number of severe cases were unexpectedly high when related to fluoride concentrations in drinking water (Manji et al., 1986), although nutritional factors could have confounded these results. The reasons for these findings are unknown and do not seem to have been explored further.

Data published in CDC's Morbidity and Mortality Weekly Report in 2005 (Beltrán-Aguilar et al., 2005) show that Black and Mexican-Americans significantly higher rates of dental fluorosis particularly in its most disfiguring categories (moderate and severe) than do Whites, as shown in Table 4.
As discussed above African Americans and Hispanics have been shown to be at an increased risk of developing dental fluorosis, and have a higher risk of suffering from the more severe forms of this condition (Russell, 1962; Butler et al., 1985; Williams & Zwemer, 1990; Beltrán-Aguilar et al., 2005, 2010; Martinez-Mier & Soto-Rojas, 2010).

It is not yet known why blacks suffer higher rates of dental fluorosis. According to the CDC, it may be a result of "biologic susceptibility or greater fluoride intake." (Beltrán-Aguilar et al., 2005). Whatever the explanation, it is clear that the black community is being disproportionately harmed by current fluoride policies in the United States.

Here are a few possible explanations:

1) African Americans consume significantly more total fluids and plain water, and thus receive more fluoride from drinking water, than white children (Sohn et al., 2009).

2) According to CDC, African-Americans are less likely to breastfeed than most other racial groups: "non---Hispanic blacks had a lower prevalence of breastfeeding initiation than non---Hispanic whites in all but two states..." (CDC, 2010). As human milk contains very low levels of fluoride (Ekstrand et al., 1981, 1984; Sener et al., 2007), babies fed formula made with fluoridated water at 0.7 - 1.2 mg/L will receive 100 to 200 times more fluoride than a human-fed baby simply through consumption of the water. If the parent reduces the amount of formula in a fluoridated community to save money as many poor parents do (Stein 2008; Egemen et al., 2002; Parraga et al., 1988), and adds more water than recommended, these children will receive even higher levels of fluoride.

3) It is possible that children with lead exposure will be more susceptible to developing dental fluorosis. African-Americans in the inner city have had more exposure to lead than white children. On average, between 1999 and 2004, black children were 1.6 times more likely to test positive for lead in their blood than white children.

4) Fluoride’s toxicity is exacerbated by inadequate nutrition, including lower intakes of iodine and calcium (see studies at FAN, 2012). African-American communities suffer from a greater

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<table>
<thead>
<tr>
<th>Characteristic</th>
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<th>Mild</th>
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<td>67.40</td>
<td>2.65</td>
<td>9.91</td>
<td>1.35</td>
<td>15.55</td>
</tr>
</tbody>
</table>

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71

prevalence of inadequate nutrition.

5) Certain racial groups are more likely to be lactose intolerant than others. Included among these are Central and East Asians (80-100% lactose intolerant; de Vrese et al., 2001), Native Americans (80-00% lactose intolerant; National Institute of Child Health and Human Development, 2006), African Americans (75% lactose intolerant), and Southern Indians (70% lactose intolerant; de Vrese et al., 2001). The elevated incidence of lactose intolerance may indicate lower rates of milk consumption, and higher consumption rates of water or other beverages, than Whites (21% lactose intolerant; Scrimshaw, 1988). Sohn et al. (2001) states “The effect of race or ethnicity and socioeconomic status (SES) on fluid consumption were particularly noticeable,” with African American children consuming significantly more plain water and less milk than other racial or ethnic groups. Thus these groups may be more heavily exposed to fluoride in water and other beverages than are Caucasian Americans, and their calcium and Vitamin D intakes may be compromised. Calcium in the diet is partially protective of fluoride because it lowers uptake of fluoride from the gut.

6) Dark pigmentation reduces the synthesis of Vitamin D in the skin at a given level of sunlight, and reduction of sunlight by inner-city pollution may be a further factor.

7) African-Americans tend to use more fluoridated toothpaste and this could contribute to differences in the rates and severity of fluorosis between whites and African-Americans (Martinez-Mier, 2010).

Has Fluoridation Helped Reduce Tooth Decay In The Inner City?

While it is clear that the fluoridation program has failed to limit the prevalence of dental fluorosis to levels anticipated in 1945, what about the other half of the program? Has it reduced tooth decay? And in the context of this discussion has it reduced tooth decay in low-income families and minority communities especially in the inner city?

Despite the laudable aim to reduce the inequalities in dental care, putting fluoride in everyone’s water to reduce tooth decay among inner city children has not been the magic bullet it was expected to be. Story after story in the media of major fluoridated cities in the US tell the same story: we still have a dental crisis among America’s inner city children especially among poor and minority families. In Table 3 we summarize these reports from New Haven CT; Washington DC; Detroit MI; Boston MA; Concord NH; Manhattan and the Bronx in NY; Cincinnati OH; Pittsburgh PA; and San Antonio TX.

TABLE 3: Communities with water fluoridation and high dental decay

<table>
<thead>
<tr>
<th>Fluoridation Status</th>
<th>Detail</th>
</tr>
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<tbody>
<tr>
<td>CONNECTICUT</td>
<td>… Peters [director of New Haven Public Schools school health centers] said this past June New Haven Public schools screened 484 Troup students, from kindergarten on up to grade 8, and found that 35 percent had moderate to severe dental needs. “The need for dental care is very clear in Connecticut and New</td>
</tr>
</tbody>
</table>

90.3% of the population receive fluoridated water as of 2012

Haven,” Peters said at Troup Wednesday. “Tooth decay is the most common childhood disease. It is five times more common than asthma and its the leading reason for missed school across the state.” …


**CONNECTICUT**

See above

“Dental decay remains the most common chronic disease among Connecticut’s children. Poor oral health causes Connecticut children to lose hundreds of thousands of school days each year. One in four Connecticut children is on Medicaid, but two of three Connecticut children receive no dental care. And DSS continues to exploit the seriously stretched public health providers and the few remaining private providers. There is an oral health crisis in Connecticut.”


**DISTRICT OF COLUMBIA**

Fluoridated since 1952

Washington DC has “one of the highest decay rates in children in the country.” The “typical new patient, age 6, has five or six teeth with cavities — a ‘staggering’ number” at the Children’s National Medical Center.


**DISTRICT OF COLUMBIA**

Fluoridated since 1952

- Low-income Children in Washington, DC are at High Risk for Poor Oral Health and Consequently Inadequate School Readiness
- a large proportion (44 percent) of the 144 students examined had a history of dental caries,
- Examined students are primarily from some of the most impoverished Wards (5, 6, 7, & 8) and exhibit high caries incidence


**ILLINOIS**

Fluoridation is mandatory

98.5% of the state’s population receive

Thousands of low-income children and adults in Illinois suffer from untreated dental disease. They can’t eat or sleep properly, do their best at school or work or smile and are at risk for other serious health problems…

… Illinois has among the lowest rates in the nation for government funded dental care. As a result we face an oral health care crisis…

fluoridated water (as of 2012)  & Illinois currently has just one clinic per 8,400 children who rely on government insurance…  


INDIANA  
94.8% of the state’s population receive fluoridated water (as of 2012)  

Results from the 2006 BRFSS also indicated that 47 percent of Hoosiers ages 18 and older have had permanent teeth extracted—a percentage that was significantly higher than the national median of 44 percent (see Figure 2). Groups with the highest prevalence of tooth extractions included blacks; individuals with an annual household income of less than $35,000; and individuals with lower educational attainment. Prevalence of extractions was highly associated with age—as age increased so did the percentage of Hoosiers who reported having had any permanent teeth extracted. … The elderly, minorities, and low income citizens often face the unfortunate need to have some or all of their teeth extracted.  


MASSACHUSETTS  
70.4% of the state residents receive fluoridated water  

Children from low-income families and children from certain racial/ethnic groups not only have a much higher prevalence of oral disease but are also less likely to have had their dental caries treated. (Page 4) Significant racial, ethnic and socioeconomic disparities exist within all oral health indicators, at each grade level, and among the state’s 14 counties. (page 5)  

**Kindergarten**  
- 39.4% of non-Hispanic Black kindergarten children have been affected by dental caries, 1.7 times higher than non-Hispanic white kindergarten children;  
- 40.9% of Hispanic kindergarten children have been affected by dental caries, 1.8 times higher than non-Hispanic white kindergarten children; and  
- 41.5% of kindergarten children from low-income families have been
affected by dental caries, 1.9 times higher than kindergarten children from families with higher incomes.


**MASSACHUSETTS**

70.4% of the state residents receive fluoridated water

…”Children are going to school with cavities, gum infections, rotting teeth. I don’t think people know how serious a problem it is,” said Ms. Cepeda, who has served as coordinator of the volunteer committee. The problem is one that a special state legislative commission last year called an oral health crisis in Massachusetts: Not enough dentists are available for people on MassHealth, the state’s health plan that includes Medicaid and the Children’s Health Insurance Program...


**MICHIGAN**

Detroit
Fluoridated since 1967

Excerpt from abstract: To describe the epidemiology of dental caries among low-income African American children 5 years old and younger in the City of Detroit. **Conclusion:** Dental Caries in primary teeth in children 5 years of age and younger in Detroit is a major dental public health problem. 2006. Severity of Dental Caries Among African American Children in Detroit. By Ismail AI, Tellez M, Sohn W. Presented at the 35th Annual Meeting & Exhibition of the American Assoc. for Dental Research in Orlando, Florida. March.

**MICHIGAN**

Detroit
Fluoridated since 1967

From abstract: The aim of this study was to examine the relationship between dietary patterns and caries experience in a representative group of low-income African-American adults. Participants were residents of Detroit, Michigan, with household incomes below 250% of the federally-established poverty level (n = 1,021)… This population had severe caries, poor oral hygiene, and diets that are high in sugars and fats and low in fruits and vegetables. Apart from tap water, the most frequently consumed food item by adults of all ages was soft drinks; 19% of all energy from sugar came from soft drinks alone.

“It’s overwhelming,” said Deb Bergschneider, dental clinic coordinator at the Concord center. “Because we serve the uninsured, we see the lower level of the community and the need is just astronomical. … By the time they get to us, their mouths are bombed out. They are all emergency situations. It’s a severe, severe, problem. It’s sad.”


The level of untreated decay, %d/ dft, was 91%, significantly higher than the US national population which is 76% overall, and 76% for African Americans and Mexican Americans within the US national population.

CONCLUSIONS: The children in this population have higher caries prevalence and a higher level of untreated caries than the national means as reported in NHANES III. The high level of untreated decay found in this particularly disadvantaged community suggests that enhanced dental services targeting the very young are needed in these communities.


http://fluoridealert.org/studytracker/19188/

“Bleeding gums, impacted teeth and rotting teeth are routine matters for the children I have interviewed in the South Bronx. Children get used to feeling constant pain. They go to sleep with it. They go to school with it. Sometimes their teachers are alarmed and try to get them to a clinic. But it’s all so slow and heavily encumbered with red tape and waiting lists and missing, lost or canceled welfare cards, that dental care is often long delayed. Children live for months with pain that grown-ups would find unendurable. The gradual attrition of accepted pain erodes their energy and aspiration. I have seen children in New York with teeth that look like brownish, broken sticks. I have also seen teen-agers who were missing half their teeth. But, to me, most shocking is to see a child with an abscess that has been inflamed for weeks and that he has simply lived with and accepts as part of the routine of life. Many teachers in the urban schools have seen this. It is almost commonplace.”

OHIO
Cincinnati
Fluoridated since 1969-1970

“We cannot meet the demand,” says Dr. Larry Hill, Cincinnati Health Department dental director.

“It’s absolutely heartbreaking and a travesty. We have kids in this community with severe untreated dental infections. We have kids with self-esteem problems, and we have kids in severe pain and we have no place to send them in Cincinnati. People would be shocked to learn how bad the problem has become.”

… An estimated 43 percent of the city’s 8-year-olds living in low-income homes have significant teeth decay. The rate of infection stood at 37 percent in 1996.


PENNSYLVANIA
Pittsburgh
Fluoridated since 1952

“Nearly half of children in Pittsburgh between 6 and 8 have had cavities, according to a 2002 state Department of Health report. More than 70 percent of 15-year-olds in the city have had cavities, the highest percentage in the state. Close to 30 percent of the city’s children have untreated cavities. That’s more than double the state average of 14 percent.”


TEXAS
San Antonio
Fluoridated since 2002

“After 9 years and $3 million of adding fluoride, research shows tooth decay hasn’t dropped among the poorest of Bexar County’s children it has only increased—up 13 percent this year. One out of two children in the Head Start program who were checked for cavities had some last year.”

| Fluoridation is mandatory | school or work or smile and are at risk for other serious health problems...  
98.5% of the state's population receive fluoridated water (as of 2012) | ... Illinois has among the lowest rates in the nation for government funded dental care. As a result we face an oral health care crisis... Illinois currently has just one clinic per 8,400 children who rely on government insurance...  
|---|---|---|
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... The elderly, minorities, and low income citizens often face the unfortunate need to have some or all of their teeth extracted.  
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<table>
<thead>
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<th>Detroit</th>
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<tr>
<td>Fluoridated since 1967</td>
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</tbody>
</table>

From abstract: The aim of this study was to examine the relationship between dietary patterns and caries experience in a representative group of low-income African-American adults. Participants were residents of Detroit, Michigan, with household incomes below 250% of the federally-established poverty level (n = 1,021)… This population had severe caries, poor oral hygiene, and diets that are high in sugars and fats and low in fruits and vegetables. Apart from tap water, the most frequently consumed food item by adults of all ages was soft drinks; 19% of all energy from sugarcane from soft drinks alone.

<table>
<thead>
<tr>
<th>State</th>
<th>Description</th>
<th>Relevant Information</th>
</tr>
</thead>
</table>
| Massachusetts | 70.4% of the state residents receive fluoridated water                                                                                              | Children from low-income families and children from certain racial/ethnic groups not only have a much higher prevalence of oral disease but are also less likely to have had their dental caries treated.  
   (Page 4)  
   Significant racial, ethnic and socioeconomic disparities exist within all oral health indicators, at each grade level, and among the state’s 14 counties.  
   (Page 5)  
   Kindergarten  
   - 39.4% of non-Hispanic Black kindergarten children have been affected by dental caries, 1.7 times higher than non-Hispanic white kindergarten children;  
   - 40.9% of Hispanic kindergarten children have been affected by dental caries, 1.8 times higher than non-Hispanic white kindergarten children; and  
   - 41.5% of kindergarten children from low-income families have been affected by dental caries, 1.9 times higher than kindergarten children from families with higher incomes.  
| Massachusetts | 70.4% of the state residents receive fluoridated water                                                                                              | …“Children are going to school with cavities, gum infections, rotting teeth. I don’t think people know how serious a problem it is,” said Ms. Cepeda, who has served as coordinator of the volunteer committee.  
   The problem is one that a special state legislative commission last year called an oral health crisis in Massachusetts: Not enough dentists are available for people on MassHealth, the state’s health plan that includes Medicaid and the Children’s Health Insurance Program...  
| New Hampshire | Concord Fluoridated since 1978                                                                                                                       | “It’s overwhelming,” said Deb Bergschneider, dental clinic coordinator at the Concord center. “Because we serve the uninsured, we see the lower level of the community and the need is just astronomical. … By the time they get to us, their mouths are bombed out. They are all emergency situations. It’s a severe, severe, problem. It’s sad.”  
<p>| <strong>NEW YORK</strong>&lt;br&gt;<strong>Manhattan</strong>&lt;br&gt;Fluoridated since 1965 | The level of untreated decay, %d/ dft, was 91%, significantly higher than the US national population which is 76% overall, and 76% for African Americans and Mexican Americans within the US national population. <strong>CONCLUSIONS:</strong> The children in this population have higher caries prevalence and a higher level of untreated caries than the national means as reported in NHANES III. The high level of untreated decay found in this particularly disadvantaged community suggests that enhanced dental services targeting the very young are needed in these communities. |</p>
<table>
<thead>
<tr>
<th>NEW YORK</th>
<th>Bronx</th>
<th>Fluoridated since 1965</th>
</tr>
</thead>
<tbody>
<tr>
<td>“Bleeding gums, impacted teeth and rotting teeth are routine matters for the children I have interviewed in the South Bronx. Children get used to feeling constant pain. They go to sleep with it. They go to school with it. Sometimes their teachers are alarmed and try to get them to a clinic. But it’s all so slow and heavily encumbered with red tape and waiting lists and missing, lost or canceled welfare cards, that dental care is often long delayed. Children live for months with pain that grown-ups would find unendurable. The gradual attrition of accepted pain erodes their energy and aspiration. I have seen children in New York with teeth that look like brownish, broken sticks. I have also seen teen-agers who were missing half their teeth. But, to me, most shocking is to see a child with an abscess that has been inflamed for weeks and that he has simply lived with and accepts as part of the routine of life. Many teachers in the urban schools have seen this. It is almost commonplace.”</td>
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</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>OHIO</th>
<th>Cincinnati</th>
<th>Fluoridated since 1969-1970</th>
</tr>
</thead>
<tbody>
<tr>
<td>“We cannot meet the demand,” says Dr. Larry Hill, Cincinnati Health Department dental director. “It’s absolutely heartbreaking and a travesty. We have kids in this community with severe untreated dental infections. We have kids with self esteem problems, and we have kids in severe pain and we have no place to send them in Cincinnati. People would be shocked to learn how bad the problem has become.”</td>
<td></td>
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<tr>
<td>An estimated 43 percent of the city’s 8-year-olds living in low-income homes have significant teeth decay. The rate of infection stood at 37 percent in 1996.</td>
<td></td>
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</tr>
<tr>
<td>2002. Solvig E. Special Report: Cincinnati’s Dental Crisis. The Enquirer (Cincinnati, Ohio), October 6</td>
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</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>PENNSYLVANIA</th>
<th>Pittsburgh</th>
<th>Fluoridated since 1952</th>
</tr>
</thead>
<tbody>
<tr>
<td>“Nearly half of children in Pittsburgh between 6 and 8 have had cavities according to a 2002 state Department of Health report. More than 70 percent of 15-year-olds in the city have had cavities, the highest percentage in the state. Close to 30 percent of the city’s children have untreated cavities. That’s more than double the state average of 14 percent.”</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2005. Law V. Sink your teeth into health care. Pittsburgh Tribune-Review</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| TEXAS | “After 9 years and $3 million of adding fluoride, research shows tooth decay hasn’t dropped among the poorest of Bexar County’s children it
Despite being fluoride overdosed, it’s not working for poor families and communities of color in the U.S. as they still suffer from higher rates of tooth decay in fluoridated communities (see Table 3 and also FAN, 2013a). Many poor and minority communities suffer from what health officials have called a “silent epidemic” of untreated tooth decay.

According to Kaste et al. (1996), national data indicate that 80% of tooth decay in children is concentrated in 25% of the child population, with low-income children and racial/ethnic minority groups having more untreated decay on average than the U.S. population as a whole.

Little has changed since 1996. According to Dye et al. (2015):

“Untreated tooth decay was higher for Hispanic (36%) and non-Hispanic black (42%) adults compared with non-Hispanic white (22%) and non-Hispanic Asian (17%) adults aged 20–64.”

This is not just the opinion of handful of dental researchers it is also the view of the number one promoter of fluoridation in the country: the CDC’s Division of Oral Health. In 2012, according to the CDC, the total population on fluoridated drinking water systems was 210,655,401 Americans or 67.1% of the population (CDC, 2012). Even with this astounding number, dental health disparities continue to thrive for communities of color and society’s poorest – the very groups that fluoridation was meant to serve. In the words of the CDC (2015):

- Oral health disparities are profound in the United States. Despite major improvements in oral health for the population as a whole, oral health disparities exist for many racial and ethnic groups, by socioeconomic status, gender, age and geographic location.

- Overall. Non-Hispanic blacks, Hispanics, and American Indians and Alaska Natives generally have the poorest oral health of any racial and ethnic groups in the United States.

- Children and Tooth Decay. The greatest racial and ethnic disparity among children aged 2–4 years and aged 6–8 years is seen in Mexican American and black, non-Hispanic children.

- Adults and Untreated Tooth Decay. Blacks, non-Hispanics, and Mexican-Americans aged 35–44 years experience untreated tooth decay nearly twice as much as white, non-Hispanics.

Fluoridation is not working for poor families and communities of color in the U.S. Why is this the case? The simple truth is that tooth decay is not caused by not enough ingested fluoride but by poor diet and too much sugar as well as too little intervention from dental professionals. This is
what the Senate Subcommittee on Primary Health and Aging said about the lack of access to dental care in 2012. Millions of Americans are “unable to get even the basic dental care they need.” (Sanders, 2012)

Poor nutrition and lack of access to professional dental care goes hand in hand with poverty. **Sadly 80% of dentists in the US will not treat children on Medicaid because the financial returns are so low** (FAN, 2013b).

Some of the children that need the most care get the least.

Fluoridation simply cannot compensate for poor diet, lack of early professional interventions and poor practices like baby’s sucking on bottles of sugared water, juice, milk and even fizzy drinks for hours on end leading to baby bottle tooth decay (BBTD) which ravages the infant’s first teeth. Such abuse of the primary teeth cannot be prevented with fluoridation but the prevalence can be reduced with better education.

Even though fluoridation promoters know that BBTD cannot be prevented by fluoridation – or should know – that doesn’t stop them using pictures of BBTD as a scare tactic to persuade communities to start or to continue fluoridation. In Figure 9, a Medical Office of Health from Canada holds up a picture of BBTD falsely implying that fluoridation will address this problem. It won’t. Such propaganda exercises are bad enough in the hands of rabid fluoridation promoters; they are even worse when practiced by civil servants whose salaries are paid for by the taxpayer.

![Figure 6: In Canada, Medical Officer of Health Dr. Hazel Lynn holds up a picture of Baby Bottle Tooth decay (BBTD). Lynn claimed in Owen Sound’s Sun Times (Jan 31, 2014) that water fluoridation prevents tooth decay and is a safe practice. The implication is fluoridation will mitigate against BBTD. It won’t! Photo: James Masters/QMI Agency (Langlois, 2014)](image-url)
Conclusion

When the US Public Health Service endorsed fluoridation in 1950 (before any trial had been completed or any meaningful health study had been published) it quickly fossilized into a policy that was considered beyond debate. In contrast, the FDA has never approved any fluoride containing substance intended to be ingested for the purpose of reducing tooth decay and in fact has rejected fluoride-containing vitamins, stating that:

“There is no substantial evidence of drug effectiveness as prescribed, recommended, or suggested in its labeling.” (Drug therapy, 1975).

Current data shows that water fluoridation disproportionately harms low-income and minority communities. In response to this injustice a growing chorus of civil rights advocates, community leaders, and environmental justice organizations have begun calling for a moratorium on fluoridation programs. This includes L.U.L.A.C. (the largest Hispanic civil rights organization), Andrew Young (the former Mayor of Atlanta and Ambassador to the United Nations), and Reverend Bernice King (the daughter of Dr. Martin Luther King). Water fluoridation has, in short, become an issue of environmental justice.

To date, EPA has ignored racial, ethnic, and socioeconomic differences when determining the level of fluoride considered “safe” for all Americans to consume in drinking water— on a daily basis and over a lifetime—and is therefore ignoring its own stated goals of achieving Environmental Justice for all.

Two strategic goals in the Interagency Working Group on environmental justice (EJ IWG) action agenda for fiscal years 2016---2018, create a very positive framework within which we can move forward on this issue. These strategic goals are:

I. Enhance communication and coordination to improve the health, quality-of-life, and economic opportunities in overburdened communities;

II. Enhance multi-agency support of holistic community-based solutions to solve environmental justice issues;

These goals challenge us to find a plan not just to fight tooth decay in children but also to improve their “health, quality of life and economic opportunities” and to do so with “community---based solutions,” which will involve “multi---agency support.”

We have taken up this challenge in our 5 step alternative plan to water fluoridation.

1) End water fluoridation. The EPA’s Office of Water could do this swiftly if they were instructed to determine a safe level of fluoride to protect all children from lowered IQ. This would not only remove a threat to children’s intellectual development and future economic potential, but it would also end a number of extra and unnecessary health threats for communities of color, especially for people with poor kidney function; borderline iodine deficiency and diabetes. Never has turning off a tap promised so much.

2) Establish the equivalent of both Scotland’s very successful Childsmile program and the Danish program for pre-schoolers, in all pre-school programs, kindergarten and primary schools (and possibly churches) and WIC programs in low-income areas.
3) Set up dental clinics either in schools or stand-alone facilities in the inner city and other low-income areas. In these we should use trained dental nurses to restore decay-damaged teeth and to remove infected ones.

4) Expand these dental clinics into community centers aimed at improving the child’s overall health. They could support better nutrition, physical fitness and cultural activities. Ideally these community centers would be linked to local community gardens and farms close to the city.

5) Further expand these community centers into job-creating operations and a foundation for local business opportunities. One concrete way of doing this is to integrate a "reuse and repair" operation into the Zero Waste approach for handling discarded materials.

Our positive, creative and holistic plan aims to fight tooth decay in low-income children but also find ways to improve their health, their fitness, their quality of life, their intellectual development and possibly even their employment within the community. We would like to go further. Our plan also works on other aspects of community development, including its food supply, its discarded resources, its local employment and business opportunities and the need to lower its carbon footprint.

More than anything else a scientifically balanced approach allows the transition from the politics of “no” to the politics of “yes.” Once we get off the shortsighted notion that we can battle tooth decay by putting a neurotoxic chemical into the public drinking water, we can unleash not only the full potential of the children from low-income communities, but also of the communities themselves. The three key words are education, nutrition and justice. We need education (not fluoridation) to fight tooth decay and obesity. We need better nutrition to keep our children and ourselves as healthy as possible and we need Environmental Justice for all.

References for this section at the end of the report on pages 220-231.
Part 6: Millions of Pounds of Legally Emitted Fluoride Compounds

The Toxic Release Inventory (TRI) for Hydrogen fluoride, Sulfuryl fluoride, Fluorine, and other fluoride compounds such as the fluorinated ozone-depleting gases.

Each year the TRI, managed by the EPA, reports on the millions of pounds of fluoride compounds that are released legally each year in the U.S. There is no mention in the Proposed Rule whether or not EPA calculated the fate of these emissions on citizens living downwind of fluoride or fluorine emitting industries. We know that fluoride bio-accumulates in the human body, particularly in calcifying tissues (such as bone and pineal gland\(^1\)\(^2\)) and is estimated to have a significantly long half-life of twenty years in bone\(^3\).

What actions has EPA taken to determine if citizens who live downwind of fluoride/fluorine emitting industry are adversely affected by the permits it issues? Were these citizens taken into account in EPA's assessment of the NPDWR for fluoride?

It is known that during times of deposition (rain, sleet, snow) that pollutants from high stacks fall close to the emitting industry. One concrete example of this comes from phytotoxicology tests for fluoride in soil and foliage at a site in Cornwall’s industrial Park in Ontario, Canada, in 1998. These tests were performed for the purpose of getting baseline numbers of various contaminants before a proposed incinerator was built at this site to allow comparison with future (post operational) sampling results. The industrial park is located approximately 5 miles downwind of two aluminum smelters operated in Massena, New York. The tests, taken by the Ontario Ministry of the Environment (MOE), revealed:

- **Soil fluoride:** “ALL 28 samples tested significantly exceeded the MOE’s Guidelines for Use at Contaminated Sites in Ontario”;
- **Foliage fluoride:** “27 of the 30 samples exceeded the MOE’s Upper Limits of Normal Contaminant Guidelines.”\(^4\)

A source of exposure for young children is their tendency to put non-food objects into their mouth. When issuing permits to fluoride emitting industry does EPA request that caregivers be warned about leaving children’s toys outside? How does EPA protect the vulnerable populations that live downwind of fluoride/fluorine emitting industries?

Below are two Tables on the fluoride releases as reported by TRI. Note: Several of the fluorinated gasses in Table 2 are suspected neurotoxins.

**About these Tables:**
- **On- and Off-site releases:** This amount does not include quantities of the toxic chemical that were the result of a catastrophic event, remedial action or other, one-time event not associated with production.
- **Wastes:** This includes the On- and Off-site Releases (above) and the sum of recycled on-site, recycled off-site, energy recovery on-site, energy recovery off-site, treated on-site, treated off-site, and quantities disposed of or otherwise released on- and off-site as reported on TRI’s forms.
# TABLE 1:
Releases by Year for Hydrogen Fluoride, Fluorine, Sulfuryl Fluoride: For the Years 1995-2015

http://fluoridealert.org/researchers/overview-tri/annual-releases-tri-fluorides/

<table>
<thead>
<tr>
<th>Years</th>
<th>Hydrogen Fluoride</th>
<th>Fluorine</th>
<th>Sulfuryl Fluoride</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>On-site and Off-site Releases in pounds</td>
<td>Waste Managed in pounds</td>
<td>On-site and Off-site Releases in pounds</td>
</tr>
<tr>
<td>2015</td>
<td>24,403,880</td>
<td>234,964,442</td>
<td>62,400</td>
</tr>
<tr>
<td>2014</td>
<td>27,287,778</td>
<td>247,746,195</td>
<td>59,925</td>
</tr>
<tr>
<td>2013</td>
<td>34,143,130</td>
<td>174,790,908</td>
<td>88,197</td>
</tr>
<tr>
<td>2012</td>
<td>33,439,710</td>
<td>234,896,132</td>
<td>43,308</td>
</tr>
<tr>
<td>2011</td>
<td>38,658,877</td>
<td>244,323,957</td>
<td>91,874</td>
</tr>
<tr>
<td>2010</td>
<td>45,002,564</td>
<td>246,775,513</td>
<td>111,220</td>
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<td>2009</td>
<td>48,458,934</td>
<td>237,085,959</td>
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<tr>
<td>2008</td>
<td>65,592,917</td>
<td>299,083,601</td>
<td>91,874</td>
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<tr>
<td>2007</td>
<td>74,829,907</td>
<td>308,933,440</td>
<td>173,364</td>
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<td>2006</td>
<td>74,675,025</td>
<td>295,878,581</td>
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<tr>
<td>2005</td>
<td>77,227,609</td>
<td>316,132,301</td>
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<td>2004</td>
<td>77,360,098</td>
<td>304,340,518</td>
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<td>2003</td>
<td>75,132,332</td>
<td>331,835,962</td>
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<td>2002</td>
<td>76,496,294</td>
<td>341,277,201</td>
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</tr>
<tr>
<td>2001</td>
<td>72,696,238</td>
<td>326,070,444</td>
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</tr>
<tr>
<td>2000</td>
<td>76,689,852</td>
<td>409,062,613</td>
<td>224,003</td>
</tr>
<tr>
<td>1999</td>
<td>77,893,290</td>
<td>348,665,519</td>
<td>246,557</td>
</tr>
<tr>
<td>1998</td>
<td>76,551,752</td>
<td>369,598,342</td>
<td>261,655</td>
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<td>1997</td>
<td>13,703,681</td>
<td>240,732,202</td>
<td>84,291</td>
</tr>
<tr>
<td>1996</td>
<td>13,938,528</td>
<td>240,831,307</td>
<td>48,300</td>
</tr>
<tr>
<td>1995</td>
<td>11,947,525</td>
<td>211,881,145</td>
<td>33,319</td>
</tr>
</tbody>
</table>
TABLE 2:  
Fluorinated Ozone-Depleting Gases Tracked by TRI: 
For the Years 2000, 2005, and 2010 to 2015

http://fluoridealert.org/researchers/overview-tri/other-fluoride-compounds-in-tri/

<table>
<thead>
<tr>
<th>Name</th>
<th>Year</th>
<th>On-site &amp; Off-Site Releases</th>
<th>Wastes In pounds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorodifluoromethane</td>
<td>2015</td>
<td>1,574,957</td>
<td>3,864,590</td>
</tr>
<tr>
<td></td>
<td>2014</td>
<td>1,598,964</td>
<td>2,757,591</td>
</tr>
<tr>
<td>[C-H-Cl-F2] Suspected Neurotoxicant</td>
<td>2013</td>
<td>1,723,306</td>
<td>3,204,577</td>
</tr>
<tr>
<td></td>
<td>2012</td>
<td>2,323,582</td>
<td>3,593,354</td>
</tr>
<tr>
<td></td>
<td>2011</td>
<td>3,215,315</td>
<td>4,808,164</td>
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<td></td>
<td>2010</td>
<td>3,693,165</td>
<td>5,333,264</td>
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<td></td>
<td>2005</td>
<td>6,825,549</td>
<td>10,269,801</td>
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<td></td>
<td>2000</td>
<td>8,202,449</td>
<td>11,006,911</td>
</tr>
<tr>
<td>Freon 113 (Trichlorotrifluoroethane)</td>
<td>2015</td>
<td>507,934</td>
<td>339,923,699</td>
</tr>
<tr>
<td>[C2-Cl3-F3] Suspected Neurotoxicant</td>
<td>2014</td>
<td>381,808</td>
<td>342,371,415</td>
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<td></td>
<td>2013</td>
<td>564,057</td>
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<td>2012</td>
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<td></td>
<td>2000</td>
<td>746,159</td>
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<td>2-Chloro-1,1,2-tetrafluoroethane</td>
<td>2015</td>
<td>184,671</td>
<td>806,590</td>
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<td>[C-CI3-F]</td>
<td>2014</td>
<td>242,715</td>
<td>706,106</td>
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<td></td>
<td>2013</td>
<td>307,364</td>
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<tr>
<td>2,2-Dichloro-1,1,1-trifluoroethane</td>
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<td>2012</td>
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1,1-Dichloro-1-fluoroethane

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Chlorotrifluoromethane (CFC 13)

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Dichlorofluoromethane

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3-Chloro-1,1,1-trifluoropropane

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1,1,1,2-Tetrachloro-2-Fluoroethane

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References:


Against Forgetting

The assassination of Allende quickly covered over the memory of the Russian invasion of Bohemia, the bloody massacre in Bangladesh caused Allende to be forgotten, the din of war in the Sinai desert drowned out the groans of Bangladesh, the massacres in Cambodia caused the Sinai to be forgotten, and so on, and on and on, until everyone has completely forgotten everything.

Milan Kundera
The Book of Laughter and Forgetting (1979)

Published Fluoride Studies
2010 – February 2017

The following are studies on fluoride published after FAN’s two submission to the EPA in April 2011. This list was generated by FAN’s StudyTracker at http://fluoridealert.org/studytracker/. Some studies are cited in more than one category. Below is the number of studies in each category.

104 Bones/Joints; 82 Reproductive; 81 Brain: Animal; 75 Dental Fluorosis; 75 Total Body Burden; 48 Kidney; 46 Liver; 42 Brain: Human; 42: Chemical Co-Exposures; 35 Thyroid; 31 Cancer; 31 Heart; 29 Immune System; 20 Diabetes; 14 Brain: Cell; 12 Gastrointestinal; 11 Lung; 8 Pineal Gland; 5 Parathyroid; 4 Fetal Toxicity & Teratology.

Bones/Joints: 2017 (6)


Bones/Joints: 2016 (14)


Bones/Joints: 2015 (9)


Bones/Joints: 2014 (21)


Bones/Joints: 2013 (21)


Bones/Joints: 2012 (20)


Namkaew M, Wiwatanadate P. 2012. Association of fluoride in water for consumption and chronic pain of body parts in residents of San Kamphaeng district, Chiang Mai, Thailand. Tropical Medicine & International Health Sep;17(9):1171-6.


Ranjan S, Yasmin S. 2012. Health problems in fluoride endemic areas of Gaya District. The Ecoscan 1:237-42. FULL STUDY.


**Bones/Joints: 2011 (13)**


BRAIN: Animal Studies 2016 (8)


**BRAIN: Animal Studies 2015 (22)**


**BRAIN: Animal Studies 2014 (15)**


Lou DD, Guan ZZ, Pei JJ. 2014. Alterations of apoptosis and expressions of Bax and Bcl-2 in the cerebral cortices of rats with chronic fluorosis. Fluoride 47(3):199-207. FULL STUDY.


BRAIN: Animal Studies 2013 (13)


Basha PM, Saumya SM. 2013. Suppression of mitochondrial oxidative phosphorylation and TCA enzymes in discrete brain regions of mice exposed to high fluoride: amelioration by Panax ginseng (Ginseng) and Lagerstroemia speciosa (Banaba) extracts. Cellular and Molecular Neurobiology 33(3):453-64.


BRAIN: Animal Studies 2012 (9)


**BRAIN: Animal Studies 2011 (14)**


BRAIN: Cell Studies 2016 (3)


BRAIN: Cell Studies 2015 (1)


BRAIN: Cell Studies 2014 (1)


BRAIN: Cell Studies 2013 (3)


**BRAIN: Cell Studies 2012 (4)**

Inkielewicz-Stepniak I, Radomski MW, Wozniak M. 2012. Fisetin prevents fluoride- and dexamethasone-induced oxidative damage in osteoblast and hippocampal cells. *Food and Chemical Toxicology* Mar;50(3-4):583-9.


**BRAIN: Cell Studies 2011 (2)**


**Brain: Human 2017 (1)**


**Brain: Human 2016 (6)**


Brain: Human 2015 (7)


Brain: Human 2014 (7)


and


Brain: Human 2013 (2)


Brain: Human 2012 (12)


Brain: Human 2011 (7)


Cancer: 2017 (1)


Cancer: 2016 (1)


Cancer: 2015 (4)


Peluso ME, Munnia A, Giese RW, et al. 2015. Exocyclic DNA adducts in sheep with skeletal fluorosis resident in the proximity to the Portoscuso-Portovesme industrial estate on Sardinia Island, Italy. Toxicology Research Apr 9;4:986-993


Cancer: 2014 (7)


Cancer: 2013 (7)


Rao MV, Thakur SB. 2013. Effects of melatonin and amla antioxidants on fluoride-induced genotoxicity in human peripheral blood lymphocyte cells. *Fluoride* 46(3)128–134. **FULL STUDY.**


**Cancer: 2012 (6)**


**Cancer: 2011 (5)**


Chemical Co-Exposures: 2016 (9)


Chemical Co-Exposures: 2015 (5)


Chemical Co-Exposures: 2014 (7)

Baba NA, Raina R, Verma PK, Sultana M. 2014. Alterations in plasma and tissue acetylcholinesterase activity following repeated oral exposure of chlorpyrifos alone and in conjunction with fluoride in Wistar Rats. Proceedings of the National Academy of Sciences, India Section B: Biological Sciences Dec;84(4):969-972.


Chemical Co-Exposures: 2013 (8)


**Chemical Co-Exposures: 2012 (6)**


Inkielewicz-Stepniak I, Radomski MW, Wozniak M. 2012. Fisetin prevents fluoride- and dexamethasone-induced oxidative damage in osteoblast and hippocampal cells. *Food and Chemical Toxicology* Mar;50(3-4):583-9.


**Chemical Co-Exposures: 2011 (8)**


**Dental Fluorosis: 2017 (2)**


**Dental Fluorosis: 2016 (11)**


Dental Fluorosis: 2015 (9)


Dental Fluorosis: 2014 (23)


Dental Fluorosis: 2013 (14)


Dental Fluorosis: 2012 (10)


**Dental Fluorosis: 2011 (8)**


Opydo-Szymaczek J, Opydo J. 2011. Dietary fluoride intake from infant and toddler formulas in Poland. Food and Chemical Toxicology Aug;49(8):1759-63.

**Diabetes: 2017 (1)**

Diabetes: 2016 (3)


Diabetes: 2015 (3)


Diabetes: 2014 (1)


Diabetes: 2013 (4)


Diabetes: 2012 (5)


**Diabetes: 2011 (3)**


**Fetal Toxicity & Teratology: 2014 (2)**


**Fetal Toxicity & Teratology: 2012 (1)**


**Fetal Toxicity & Teratology: 2011 (1)**

Gastrointestinal 2016 (1)


Gastrointestinal 2014 (3)


Gastrointestinal 2013 (7)


Gastrointestinal 2012 (1)

Ranjan S, Yasmin S. 2012. Health problems in fluoride endemic areas of Gaya District. The Ecoscan 1:237-42. FULL STUDY.
Heart: 2017 (1)


Heart: 2015 (5)


Heart: 2014 (5)

Baba NA, Raina R, Verma PK, Sultana M. 2014. Alterations in plasma and tissue acetylcholinesterase activity following repeated oral exposure of chlorpyrifos alone and in conjunction with fluoride in Wistar Rats. *Proceedings of the National Academy of Sciences, India Section B: Biological Sciences* Dec;84(4):969-972.


Heart: 2013 (8)


**Heart: 2012 (6)**


Heart: 2011 (6)


Immune System: 2016 (6)


Immune System: 2015 (1)

Immune System: 2014 (7)


Immune System: 2013 (5)


Immune System: 2012 (6)


Immune System: 2011 (4)


Kidney: 2017 (1)

Kidney: 2016 (4)


Kidney: 2015 (7)


Kidney: 2014 (14)


Thangapandiyan S, Miltonprabu S. 2014. Epigallocatechin gallate supplementation protects against renal injury induced by fluoride intoxication in rats: Role of Nrf2/HO-1 signaling. Toxicology Reports Dec;1:12-30. FULL STUDY.


Kidney: 2013 (13)


Kidney: 2012 (3)


Kidney: 2011 (6)


Liver: 2017 (1)


Liver: 2016 (5)


**Liver: 2015 (7)**


Liver: 2014 (12)


Liver: 2013 (12)


Liver: 2012 (5)


detect genotoxicity and oxidative stress in mice exposed to sodium fluoride. *Mutation
Research* Nov; 18;751(1):59-65.

Vasant RA, Narasimhacharya AV. 2012. Ameliorative effect of tamarind leaf on fluoride-
induced metabolic alterations. *Environmental Health and Preventive Medicine* Nov;
17(6):484-93.

**Liver: 2011 (4)**

histopathology and synthesis of stress protein in liver and kidney of mice. *Archives of
Toxicology* Apr; 85(4):327-35.

glomerulonephritis and induces liver damage in ICR-derived glomerulonephritis mice.
*Toxicological & Environmental Chemistry* Dec;93(10):2072–2084.

Lech T. 2011. Fatal cases of acute suicidal sodium and accidental zinc fluorosilicate
poisoning. Review of acute intoxications due to fluoride compounds. *Forensic Science

Shashi A, Bhardwaj M. 2011. Study on blood biochemical diagnostic indices for hepatic
function biomarkers in endemic skeletal fluorosis. *Biological Trace Element Research*
Nov; 143(2):803-14.

**Lung: 2015 (1)**


**Lung: 2014 (1)**

Abdel-Gawad FA, Ashmawy MH, Zaki SM, Abdel-Fatah GH. 2014. Lung damage after long-
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**Lung: 2013 (4)**

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Lung: 2012 (4)


Lung: 2011 (1)


Parathyroid: 2015 (2)


Parathyroid: 2013 (1)


Parathyroid: 2011 (2)


Pineal Gland: 2015 (1)

Pineal Gland: 2014 (2)


Pineal Gland: 2013 (1)


Pineal Gland: 2012 (2)


Pineal Gland: 2011 (2)


Reproductive: 2017 (2)


Reproductive: 2016 (15)


Reproductive: 2015 (35)


Reproductive: 2014 (9)


Reproductive: 2013 (10)


Reproductive: 2012 (6)


Reproductive: 2011 (5)


Thyroid: 2017 (1)

Thyroid: 2016 (4)


Thyroid: 2015 (7)


Thyroid: 2014 (6)


**Thyroid: 2013 (10)**


**Thyroid: 2012 (3)**


**Thyroid: 2011 (4)**


**Total Body Burden: 2017 (3)**


Total Body Burden: 2016 (6)


Total Body Burden: 2015 (6)


Total Body Burden: 2014 (12)


**Total Body Burden: 2013 (19)**


Arshad M, Shanavasb P. 2013. Comparison of serum and urinary fluoride levels among fertilizer and wood industry workers in Mangalore City, India. *Fluoride* 46(2):80–82. **FULL STUDY**.


**Total Body Burden: 2012 (12)**


Total Body Burden: 2011 (17)


The Neurotoxicity of Fluoride: Bibliography

NOTE 1: A flash drive containing all the studies in this bibliography was sent on March 11, 2017, by Express Mail for delivery on March 13 to the: EPA Docket Center, WJC West Building, Room 3334, 1301 Constitution Avenue, NW, Washington, DC 20004

NOTE 2: For studies that were originally published in Chinese and later published in English, we use the date of English publication. For studies that were originally published in Chinese that have been translated into English but not yet republished, we use the date of the original study, not the date of translation.


Basha PM, Saumya SM. 2013. Suppression of mitochondrial oxidative phosphorylation and TCA enzymes in discrete brain regions of mice exposed to high fluoride: amelioration by Panax ginseng (Ginseng) and Lagerstroemia speciosa (Banaba) extracts. Cellular and Molecular Neurobiology 33(3):453-64.


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<th>Reference</th>
<th>Page</th>
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**APPENDIX A:**

**Post-NRC Human Studies Investigating Fluoride’s Impact on Cognition**

<table>
<thead>
<tr>
<th>Exhibit No.</th>
<th>Study Description</th>
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**APPENDIX B:**
Post-NRC Human Studies Investigating Fluoride’s Impact on Fetal Brain

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APPENDIX C:
Post-NRC Human Studies Investigating Fluoride’s Impact on Other Parameters of Neurotoxicity

<table>
<thead>
<tr>
<th>Exhibit No.</th>
<th>Study Details</th>
</tr>
</thead>
</table>
APPENDIX D:
Post-NRC Animal Studies Investigating Fluoride’s Neuroanatomical & Neurochemical Effects


15. Basha PM, Saumya SM. 2013. Suppression of mitochondrial oxidative phosphorylation and TCA enzymes in discrete brain regions of mice exposed to high fluoride: amelioration by Panax ginseng (Ginseng) and Lagerstroemia speciosa (Banaba) extracts. *Cellular and Molecular Neurobiology* 33(3): 453-64.


**APPENDIX E:**

Post-NRC Animal Studies Investigating Fluoride’s Effect on Learning/Memory
<table>
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<tr>
<th>Exhibit No.</th>
<th>Reference</th>
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APPENDIX F:
Post-NRC Animal Studies Investigating Fluoride’s Effect on Other Behavioral Parameters Beyond Learning/Memory


**APPENDIX G:**

**Post-NRC In Vitro Studies Investigating Fluoride’s Effect on Brain Cells**

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<th>Exhibit No.</th>
<th>Study Description</th>
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<tbody>
<tr>
<td>3.</td>
<td>Inkielewicz-Stepniak I, Radomski MW, Wozniak M. 2012. Fisetin prevents fluoride- and dexamethasone-induced oxidative damage in osteoblast and hippocampal cells. <em>Food and Chemical Toxicology</em> 50(3-4):583-589.</td>
</tr>
</tbody>
</table>


References for Part 5: Fluoride and Environmental Justice


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(This was a sidebar article in a larger article titled: Fluoridation of Water: Questions about health risks and benefits remain after more than 40 years, http://fluoridealert.org/articles/hileman/)


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