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Fluoride Action Network

A summary of the Petition calling on the U.S. Environmental Protection Agency to ban the fluoridation of the public drinking water under provisions in the Toxic Substances Control Act.

THIS PETITION WAS SUBMITTED BY THE FOLLOWING ORGANIZATIONS ON NOV 22, 2016: Fluoride Action Network, Food & Water Watch, Organic Consumers Association, American Academy of Environmental Medicine, International Academy of Oral Medicine and Toxicology, Moms Against Fluoridation.

OVERVIEW

The addition of industrial-grade fluoride chemicals to public water supplies for the purpose of preventing tooth decay is hailed by public health institutions in the U.S., but has been rejected by most of continental Europe without any demonstrable adverse effect on childhood caries rates.

Fluoride is not an essential nutrient and does not need to be swallowed to prevent tooth decay. By contrast, fluoride's risks to health come from ingestion, including the spectrum of neurotoxic effects discussed in this petition.

Petitioners request that EPA exercise its authority under TSCA, to prohibit the purposeful addition of fluoridation chemicals to U.S. water supplies. Petitioners make this request on the grounds that a large body of animal, cellular, and human research shows that fluoride is neurotoxic at doses within the range now seen in fluoridated communities.

Using the EPA's preferred method for ascertaining a Safe Reference Dose (RfD) (the Benchmark Dose method BMD) for a toxic substance, it can be shown that a dose of 1.4 mg per day could lower the IQ of a child by 5 IQ points. Children drinking two liters of water at 0.7 mg/liter 0.7 ppm) would reach this dose. If a modest safety factor was applied to this number (1.4 mg/day) to account for the known wide range of vulnerability to any toxic substance in a large population, many millions of children in the USA would be exceeding the computed safe reference dose (RfD) for lowered IQ (see section IX below for details).

When considering the principles set forth in EPA's *Guidelines for Neurotoxicity Risk Assessment,* Petitioners submit that fluoridation is incompatible with a neurologically safe use of fluoride.

Petitioners further make 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 the many processed beverages and

foods made therefrom) 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.

II. THE TOXIC SUBSTANCES CONTROL ACT (TSCA).

The Toxic Substances Control Act (TSCA) invests EPA with the authority to take certain actions if it determines that "the manufacture, processing, distribution in commerce, use, or disposal of a chemical substance . . . presents an unreasonable risk of injury to health." In making this determination, TSCA commands that EPA consider not only risks to the general public, but to "susceptible subpopulation[s]" as well. Further, TSCA commands that EPA conduct the risk evaluation "without consideration of costs or other non-risk factors."

The actions that EPA may take include: (1) a complete prohibition on the manufacture, processing, and distribution of the substance or (2) a prohibition on a "particular use" of the substance.

EPA's authority to prohibit and regulate the use of chemical substances under TSCA encompasses drinking water additives.

Why TSCA, rather than SDWA?

Although EPA has certain authorities to regulate fluoride in drinking water under the Safe Drinking Water Act (SDWA), there is an important distinction between TSCA and SDWA that permits EPA to take the requested action under TSCA in a more targeted, efficient, and less expensive manner than would be the case under SDWA. Namely, TSCA permits the EPA to differentiate between fluoride that is *added* to water versus fluoride that is *naturally occurring*. As explained in the petition, prioritizing regulatory action against fluoridation *additives* is further justified on policy and scientific grounds. It is therefore in the public interest for EPA to take the requested action under TSCA, instead of SDWA.

III. REGULATORY BACKGROUND: FLUORIDE IN DRINKING WATER

In 2003, the EPA asked the National Research Council (NRC) to review the scientific merits of EPA's Maximum Contaminant Level Goal (MCLG) of 4 mg/L. for fluoride.

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. 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."

This conclusion by the NRC rested primarily on its review of animal studies, since—at the time - few human studies were available. However, since the publication of NRC report in 2006 46 human studies have been published that have found significant relationships between fluoride and adverse cognitive outcomes. This number dwarfs the 5 studies that were available to the NRC in 2006.

It has been 10 years since the NRC concluded that the MCLG for fluoride be lowered, but 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. Instead, in December 2010, EPA OW established a reference dose for fluoride based solely on severe dental fluorosis. 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. But nowhere in EPA OW's risk assessment did it account for the neurotoxicity research published subsequent to NRC's review, despite the many studies on neurotoxicity sent to them.

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* that EPA has stated it "*will* follow in evaluating data on *potential* neurotoxicity associated with exposure to environmental toxicants."

Petitioners submit 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. THE RESEARCH DEMONSTRATING FLUORIDE'S NEUROTOXICITY INCLUDES OVER 180 STUDIES PUBLISHED SINCE THE NRC'S 2006 REVIEW

In total, Petitioners 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 three post-NRC systematic reviews of the literature, including two that address the human/IQ literature, and one that addresses the animal/cognition literature.

Petitioners have attached copies of over 300 human, animal, and cell studies of fluoride's neurotoxicity which include those that have become available since NRC's review as listed above.

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.

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

A frequent claim made by fluoridation promoters 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 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 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) or a Benchmark Dose level (BMD).

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.

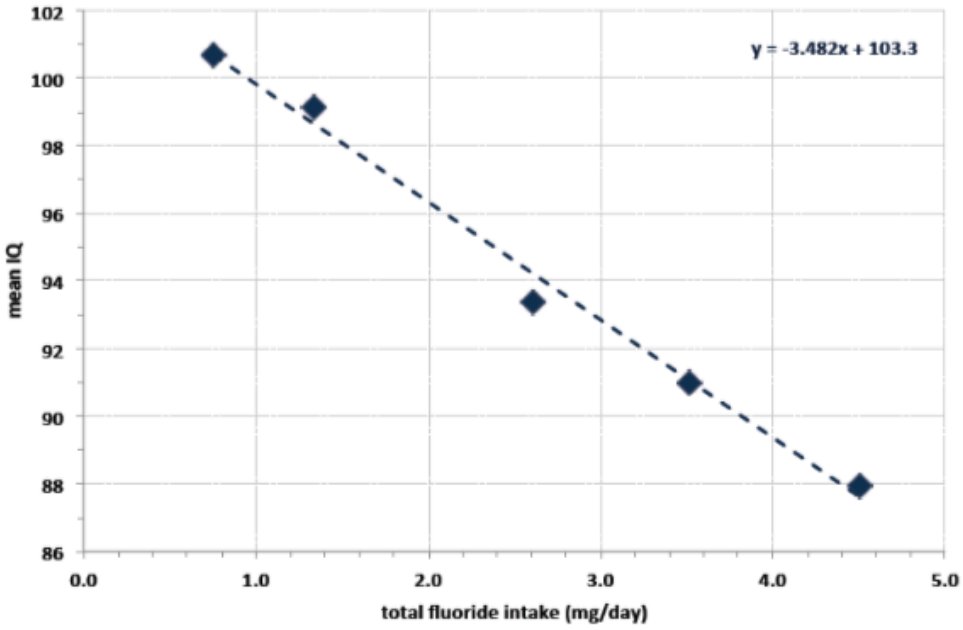
For example, in rural China (where most of the IQ studies have been conducted), fluoridated toothpaste is rarely used. By contrast, in the United States, over 95% of toothpastes are fluoridated and research shows that toothpaste can contribute more fluoride to a child's daily intake than fluoridated water.

Available evidence suggests that (i) the daily fluoride doses, (ii) urine fluoride levels, (iii) serum fluoride levels, and (iv) dental fluorosis levels are all associated with IQ reductions

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.

In the study by Wang, 2012 the authors 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* than the *average* daily intake (**0.087 mg/kg/day**) for non-nursing infants (i.e. bottle-fed) in the United States, and just two times greater than the *average* daily dose for 8-12 year old American children.

As with other forms of fluoride toxicity, the potential for fluoride neurotoxicity is magnified among children with suboptimal nutrient intake. 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, the authors confirmed a significant correlation between total fluoride intake and reduced IQ. Notably, these authors 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**.). This is a dose that many infants and children in the U.S. are estimated to exceed.

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.

VII. RECENT EPIDEMIOLOGICAL STUDIES CORROBORATE NEUROTOXIC RISK FROM FLUORIDATED WATER IN WESTERN POPULATIONS

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).

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 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." Since both clinical and subclinical hypothyroidism during pregnancy have been associated with reduced IQ in offspring 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 TO FLUORIDE NEUROTOXICITY AND NEED PROTECTION

Recent research in both humans and animals has specifically demonstrated that nutrient deficiencies (i.e., iodine and calcium) amplify fluoride's neurotoxicity.

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 (A) deficient nutrient intake (particularly 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." 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

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 (Benchmark Dose) into a safe “reference dose” (RfD) or “reference concentration” (RfC).

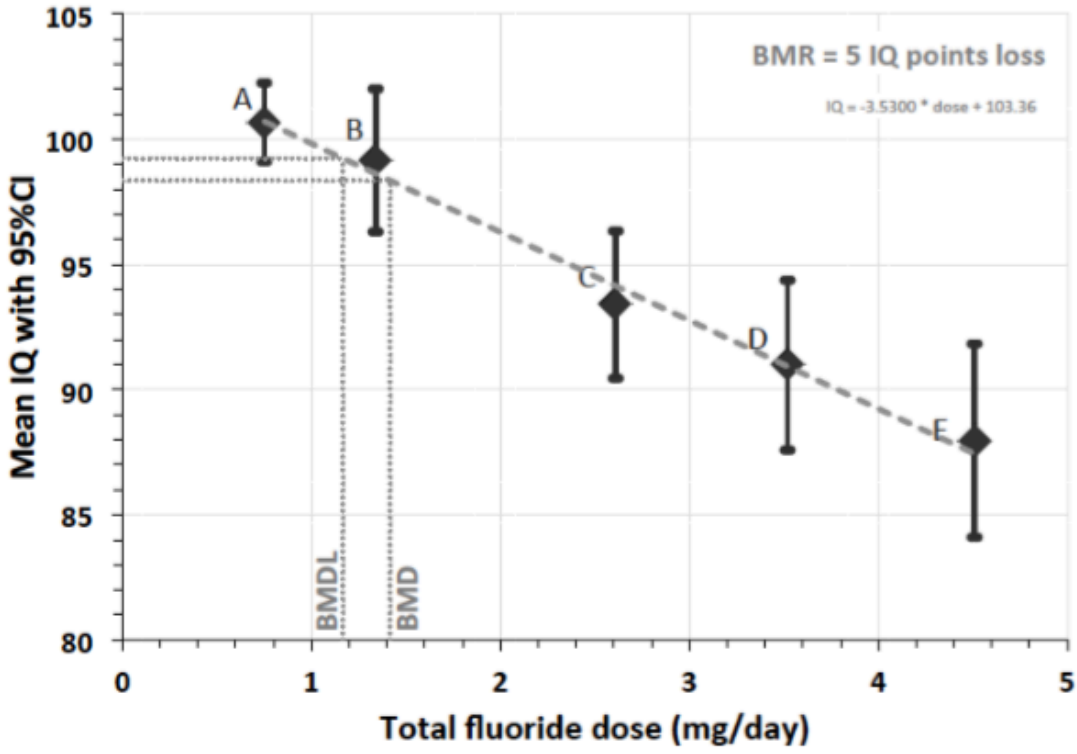
This is significant because application of even a single UF of 10 to the daily doses/concentrations of fluoride associated with neurotoxic harm in humans and animals produces an RfD or RfC that is less than, and thereby *incompatible with*, the levels of fluoride added to water for fluoridation (0.7 to 1.2 mg/L).

Petitioners recognize 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).

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).

If the BenchMark Response (BMR) is set at 5 IQ points, applying a BMD analysis to Xiang’s database results in a BMD of just **1.4 mg F/day** (see the following figure)

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



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.

X. THE BROADBENT STUDY DOES NOT ESTABLISH THE SAFETY OF FLUORIDATION

Some commentators have incorrectly claimed that the recent study by Broadbent et al. 2015 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, the authors conceded that the average difference in total daily intake between the children in the fluoridated and non-fluoridated areas would be less than 0.3 milligrams per day, while the average intake for all subjects was 0.9 mg/day. (Broadbent et al. 2016). At most, therefore, the Broadbent study established that a dose less than 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.

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.

XI. THE BENEFITS OF PREVENTING FLUORIDE NEUROTOXICITY DWARF THE COSTS OF RESTRICTING FLUORIDE CHEMICALS

EPA's authority to act under Section 6 of TSCA is premised on two distinct findings: (1) a *risk* exists and (2) the risk is *unreasonable*. Here, in evaluating the preliminary question of whether a neurotoxic risk exists from use of fluoridation chemicals, the EPA is duty bound to follow its *Guidelines*, as the Agency has stated it "*will* follow" the *Guidelines* when "evaluating data on *potential* neurotoxicity associated with exposure to environmental toxicants."

For the reasons set forth above, a good faith application of these *Guidelines* to the current research on fluoride will show that neurotoxicity is a hazard of fluoride exposure, and that the doses associated with this hazard overlap the doses—as reflected by (a) total daily intake, (b) urinary fluoride level, (c) serum fluoride level, and (d) severity of dental fluorosis—that U.S. children are exposed to in areas with fluoridated water. Neurotoxicity must thus be considered a risk from adding fluoridation chemicals to drinking water.

Petitioners now turn, therefore, to the second prong of the inquiry: whether the neurotoxic risk posed by fluoridation chemicals is an unreasonable one. As EPA has stated, the reasonableness inquiry considers the benefits of reducing the risk with the costs of doing so. In considering these respective benefits and costs of risk reduction, EPA has stated it will take into account "the extent and magnitude of risk posed; the societal consequences of removing or restricting use of products; availability and potential hazards of substitutes, and impacts on industry, employment, and international trade."

There is little question that neurotoxicity is a serious insult to health. 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.

Because of the *massive* extent of exposure to fluoridation chemicals in the U.S., even small effects on IQ will have very substantial economic consequences.

Studies have shown that even a loss of a single IQ point causes an average drop in lifetime earnings of $22,250 in current dollars. Since 200 million Americans now live in areas where water is fluoridated, 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.

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. 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, resulting in 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 a fluoridated area suffer any IQ loss, and even if this IQ loss averaged just 1 IQ point, 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.

XII. IT IS IN THE PUBLIC INTEREST FOR EPA TO TAKE ACTION UNDER TSCA

As the above discussion has indicated there are urgent health and economic reasons why the EPA should use the provisions in TSCA to ban the purposeful addition of fluoride to the public drinking water.