



Water Fluoridation Reconsidered: Minimal Benefits, Mounting Risks, and Ethical Dilemmas

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Abstract: Public water fluoridation, once a celebrated preventive dentistry milestone, is increasingly being reevaluated in light of current scientific, ethical, and policy considerations. While initially justified based on data suggesting systemic benefits from ingestion, current evidence demonstrates fluoride's primary anticaries mechanism is topical, not systemic. Simultaneously, recent studies have linked fluoride ingestion—even at levels considered safe (0.7 mg/L) by the U.S. Department of Health and Human Services (HHS)—to neurodevelopmental harm, endocrine disruption, and musculoskeletal risks. This article presents a review of the scientific literature addressing fluoride's mechanisms of action, its systemic risks, and the ethical challenges posed by involuntary mass medication. Based on recent epidemiological findings, policy developments, and bioethical principles, the continued practice of public water fluoridation appears scientifically outdated and ethically untenable.

INTRODUCTION:

Fluoridation of public water supplies began in the United States in the 1940's as a novel approach to preventing dental caries. Spurred by early observational studies of communities with naturally fluoridated water, fluoridation was seen as an effective, low-cost intervention to reduce tooth decay in children. In 1945, Grand Rapids, Michigan became the first U.S. city to fluoridate its water. By the 1960s, the U.S. Public Health Service endorsed the practice nationwide, thereby spreading rapid adoption.

Today, approximately 63% of the U.S. population receives fluoridated water. However, the same trend has not been observed globally. Western European countries have largely rejected or discontinued water fluoridation. For instance, Austria, Belgium, Denmark,

Finland, France, Germany, Italy, the Netherlands, Norway, Sweden, and Switzerland have all opted against fluoridation, citing concerns about safety, ethics, or the lack of demonstrable benefits. Despite similar or lower rates of tooth decay in these countries, they have achieved oral health improvements through alternative strategies such as topical fluoride use, education, and dietary interventions.

The divergence in global fluoridation policy invites scrutiny and critical questions. For example, has fluoridation become an outdated relic of public health policy? Do its claimed benefits outweigh the risks, particularly when fluoridated toothpaste is nearly universal? And finally, is it ethically defensible to administer a potentially harmful chemical via public utilities, without individual consent?

Systemic vs. Topical Fluoride: Mechanism and Efficacy Reconsidered

Historically, the rationale for water fluoridation originated from the belief that fluoride has systemic benefits, namely that fluoride ingested during childhood integrates into developing enamel, rendering teeth more resistant to acid demineralization. This paradigm has dominated public health doctrine for decades. However, research over the past 25 years has decisively shifted the scientific consensus toward topical action as the principal mechanism of fluoride efficacy while highlighting significant systemic risks pertaining to ingested fluoride.

The CDC, despite calling water fluoridation “one of the ten greatest public health achievements of the twentieth century,” acknowledged as early as 1999 that “Fluoride prevents dental caries predominantly after eruption of the tooth into the mouth, and its actions primarily are topical for both adults and children” [1]. This mirrors the findings of Featherstone et al., who concluded in their seminal 2000 study, “Fluoride, the key agent in battling caries, works primarily via topical mechanisms...Fluoride incorporated during tooth development is insufficient to play a significant role in caries protection” [2].

Moreover, a 2004 review in *Caries Research* further asserted that systemic incorporation of fluoride during enamel formation provides negligible resistance to decay, compared to topical applications: “A dogma has existed for many decades, that fluoride has to be ingested and acts mainly pre-eruptively. However, recent studies concerning the systemic effect of

fluoride supplementation concluded that the caries-preventive effect of fluoride is almost exclusively posteruptive” [3].

If fluoride works best when applied topically, then ingesting it through water becomes an inefficient and potentially hazardous route of administration. Topical application via toothpaste (1000–1500 ppm fluoride) provides controlled and effective exposure with minimal systemic absorption. In contrast, fluoridated drinking water (~0.7 ppm) results in widespread systemic distribution, affecting every organ and accumulating in tissues like bone and the pineal gland.

The topical benefits of fluoride from drinking water are minor compared to those from toothpaste. Drinking water only briefly contacts enamel during consumption. The Cochrane Collaboration’s 2015 systematic review found that most studies demonstrating caries reduction were conducted before 1975, when fluoridated toothpaste use was far less common [4]. In modern populations, the evidence of significant additional benefit from water fluoridation is weak, especially when controlling for confounding variables such as socioeconomic status, diet, and access to dental care.

Moreover, the 2024 Cochrane update concluded that among studies published after 1975, the mean reduction in decayed, missing, or filled primary teeth (DMFT) was only 0.24 teeth per child—a negligible effect [5]. In terms of public health return on investment, this minor benefit does not justify widespread and indiscriminate exposure to ingested fluoride and its many health risks.

In the next sections, these risks are explored in detail, beginning with perhaps the most concerning: neurotoxicity and the implications for childhood cognitive development and intelligence quotient (IQ).

Neurodevelopmental Risk: Fluoride and Brain Health

Among the most consequential findings in fluoride research are those regarding its effects on the developing brain. Mounting evidence points to fluoride being a potential neurotoxin, particularly when exposure occurs during prenatal and early postnatal development. Concerning evidence linking fluoride to reductions in intelligence quotient (IQ) and altered neurobehavioral outcomes also merits close examination.

Numerous observational studies from varied

geographic regions have associated higher fluoride exposure with lower IQ scores in children. A 2012 meta-analysis by Choi et al., published in *Environmental Health Perspectives*, analyzed 27 studies and found a consistent association between elevated fluoride levels in drinking water and an average IQ reduction of 6.9 points in children [6]. While many of these studies were conducted in regions with naturally high fluoride concentrations (e.g., China, Iran, and India), the implication of fluoride as a neurotoxin is biologically plausible and consistent across cohorts.

Crucially, more recent studies have focused on populations with fluoride exposures comparable to those found in fluoridated North American water systems (~0.7 mg/L). The ELEMENT study in Mexico, for example, followed mother-child pairs and found that a 1 mg/L increase in maternal urinary fluoride during pregnancy was associated with a 5–6 point reduction in child IQ by ages 4–12 [7]. This was not a small or isolated effect—it remained significant after controlling for multiple confounders including socioeconomic status, maternal education, and lead exposure.

The Canadian MIREC study, a government-funded cohort of over 500 mother-child pairs, similarly found that a 1 mg/L increase in maternal urinary fluoride corresponded to a 4.5-point IQ decrease in male offspring at ages 3–4, with a smaller, non-significant effect in girls [8]. Another analysis from the same cohort reported that infants who were formula-fed with fluoridated water scored significantly lower in performance IQ than those fed with non-fluoridated water, particularly boys [9].

Finally, a 2023 dose-response meta-analysis by Grandjean and Veneri published in *Environmental Research* provided a more granular understanding of the relationship. This team calculated a benchmark dose level (BMDL) at which fluoride exposure would lead to a 1-point IQ loss. The BMDL was approximately 0.2 mg/L in maternal urine fluoride—far below the average levels observed in fluoridated populations [10]. These findings suggest that even current “optimal” fluoridation levels may exceed reasonable thresholds for neurotoxicity in vulnerable populations.

The biological plausibility of fluoride as a neurotoxin is also supported by animal studies. Fluoride readily crosses the placenta and accumulates in fetal tissue, including the brain. In rodents, prenatal fluoride exposure has been linked to oxidative stress, altered neurotransmitter levels, and histological changes in the hippocampus—a region critical for memory and

learning [11].

In response to mounting concerning evidence, the U.S. National Toxicology Program (NTP) completed a systematic review in 2020, concluding that fluoride is “presumed to be a cognitive developmental hazard to humans” [12]. This classification was based on moderate to high confidence in the human evidence and some supporting animal studies. Notably, the NTP found consistent associations between fluoride exposure and lower IQ in children, even in studies of water fluoride levels below 1.5 mg/L.

Opponents of these findings argue that ecological studies are subject to bias and confounding. However, the strongest studies—such as ELEMENT and MIREC—are prospective birth cohorts with individual-level fluoride measurements and extensive adjustment for potential confounders. Their findings cannot be easily dismissed or blamed on poor design.

The implications of fluoride-induced neurotoxicity are far-reaching. Even a modest average IQ reduction across a population can shift the curve of intellectual ability, reducing the number of high-performers and increasing the number of individuals requiring special educational services. According to Grandjean, a population-wide IQ loss of 5 points equates to a profound economic and societal burden, including reduced productivity and increased social support needs [13].

Given that fluoride’s primary dental benefits are topical, not systemic, and that systemic exposure offers only very minor cavity prevention, the tradeoff between significant cognitive damage and negligible dental health benefits becomes medically and ethically indefensible.

Endocrine Disruption: Fluoride’s Effects on Thyroid and Pineal Gland Function

Fluoride’s interaction with the endocrine system, particularly the thyroid and pineal glands, is well documented in toxicological and epidemiological research. The thyroid gland plays a critical role in regulating metabolism, brain development, and mood. Fluoride, as a halogen in the same chemical family as iodine, competes with iodine uptake in the thyroid. This competition can reduce the synthesis of thyroid hormones—especially in iodine-deficient individuals.

Historically, sodium fluoride was used pharmacologically to treat hyperthyroidism due to its

known suppressive effects on thyroid activity. The mechanism involves inhibition of thyroid peroxidase (TPO), an enzyme essential for the iodination of tyrosine residues and the subsequent synthesis of T3 and T4 hormones.

Modern epidemiological studies have reignited concern about fluoride's impact on the thyroid. A 2015 study by Peckham et al., conducted in England, used a large dataset from primary care providers to compare hypothyroidism prevalence in fluoridated versus non-fluoridated areas. The results revealed that those in areas with fluoride levels ≥ 0.7 mg/L were nearly twice as likely to report high rates of hypothyroidism, even after adjusting for age and sex [14].

Further evidence comes from cross-sectional analyses conducted in the United States and Canada. Malin AJ, et al. found that adolescents living in fluoridated regions had statistically lower free T4 concentrations than those in non-fluoridated regions, despite no difference in TSH levels, suggesting subtle suppression of thyroid hormone production [15]. This pattern may be particularly consequential during periods of rapid development, such as adolescence or pregnancy.

In animal models, fluoride exposure has consistently resulted in thyroidal changes. Rats exposed to 1–5 mg/L fluoride in drinking water exhibited decreased serum T3 and T4 levels and increased TSH, mirroring hypothyroidism in humans. These endocrine effects have downstream implications for cognitive development, growth, and energy metabolism.

The pineal gland is another endocrine organ profoundly affected by fluoride. Located near the center of the brain, the pineal gland secretes melatonin—a hormone that regulates circadian rhythm and the sleep-wake cycle. Melatonin is also implicated in antioxidant activity and immune modulation.

In 2001, University of Surrey researcher Dr. Jennifer Luke demonstrated that fluoride accumulates in the pineal gland in quantities similar or greater than fluoride accumulation in the bones and teeth [16]. Her autopsy-based study found pineal fluoride concentrations averaging over 300 mg/kg, with the degree of calcification correlated with decreased melatonin production in adolescents. Luke proposed that this could partly explain the earlier onset of puberty observed in fluoridated populations, as

melatonin suppresses reproductive hormone secretion during childhood.

Other studies corroborate Luke's conclusions. A 2019 ecological study in the U.S. found that adolescents in fluoridated communities reported more sleep disturbances and delayed bedtimes than those in non-fluoridated regions [17]. Melatonin assays in small cohorts have shown reductions in overnight levels among individuals with high cumulative fluoride exposure, though larger controlled studies are still needed.

Given the widespread nature of thyroid disease and the critical function of melatonin in regulating mood, sleep, and immune response, fluoride's potential role as an endocrine disruptor raises serious public health questions. While subtle at the individual level, the population-level effects—particularly on vulnerable groups—appears to be substantial.

Musculoskeletal Effects: Bone Quality, Fractures, and Joint Health

Fluoride's impact on the musculoskeletal system is multifaceted, with negative effects accumulating over time. Unlike most environmental toxins, which are excreted relatively quickly, the body retains approximately 50% of ingested fluoride, primarily in the bones and teeth. This cumulative effect raises concerns about skeletal fluorosis, impaired bone quality, increased risk of fractures, and arthritic symptoms.

Skeletal fluorosis is a chronic metabolic bone disease caused by prolonged ingestion of high levels of fluoride. It is characterized by increased bone density (osteosclerosis), joint pain, stiffness, and in severe cases, crippling deformities. While endemic skeletal fluorosis is rare in fluoridated countries, research indicates that subtler skeletal effects may occur even at exposure levels typical of municipal fluoridation (0.7–1.5 mg/L).

Clinical trials and observational studies have produced mixed results on fluoride's effects on bone mineral density (BMD) and fracture risk. For example, randomized controlled trials using high-dose fluoride (20–40 mg/day) for osteoporosis showed increases in spinal BMD but paradoxically led to an increased rate of non-spinal fractures, suggesting that fluoride-incorporated bone may be denser but structurally inferior [18].

In population-based studies, similar concerns have

emerged. A cohort study from Sweden followed 4,306 postmenopausal women for over a decade and found that those with the highest fluoride exposure (~2.5 mg/day from water and food) had a 1.59-fold increased risk of hip fracture compared to those with the lowest exposure [19]. Importantly, the fluoride levels in this study were within the range considered safe by U.S. standards.

Additional data from the U.S. suggest that water fluoridation may correlate with higher rates of bone fracture in children and adolescents. A 2021 ecological study comparing pediatric fracture rates across the country found that those with water fluoride levels at or above 0.7 mg/L had statistically higher incidences of forearm, elbow, and lower-limb fractures [20]. While the ecological design limits causal interpretation, the findings are consistent with animal studies showing compromised bone microarchitecture from chronic fluoride exposure.

Fluoride also affects joint health. In regions with moderate to high fluoride in drinking water (~1.5–3.0 mg/L), several case-control studies have found a strong association between urinary fluoride levels and the prevalence of radiographically confirmed osteoarthritis. A 2020 Chinese study reported that each 1 mg/L increase in urinary fluoride was associated with a 27% increased occurrence of knee osteoarthritis [21]. Subjects in the highest exposure quartile had more than double the risk compared to those in the lowest.

Furthermore, skeletal fluoride burden is known to increase with age, water intake, and renal insufficiency. Individuals with impaired kidney function—such as the elderly or those with chronic kidney disease—are especially vulnerable, as their ability to excrete fluoride is reduced. Over decades, this may predispose to earlier onset of osteoporosis, degenerative joint disease, and even spinal stenosis.

Taken together, the evidence suggests that fluoride's skeletal effects are dose- and duration-dependent. Even at current fluoridation levels, long-term accumulation may subtly alter bone quality and joint function. When juxtaposed against the marginal benefits of water fluoridation for dental health, these musculoskeletal risks merit serious public health consideration.

Ethical Considerations of Involuntary Fluoridation

The practice of fluoridating public water supplies raises pressing ethical questions regarding autonomy,

consent, risk distribution, and public health governance. Central to the debate is whether it is appropriate for governments to administer a biologically active compound to entire populations without individual consent—particularly when the compound's primary benefits are topical and when solid evidence suggests ingestion has substantial health risks.

Informed Consent and Autonomy

Modern bioethics emphasizes the principle of informed consent, especially in the context of medical or quasi-medical interventions. By fluoridating municipal water, public health agencies bypass the individual's right to choose regarding consuming a pharmacologically active substance. Unlike vaccines or medications, which require informed consent and often involve targeted application based on health status, fluoridation treats all individuals identically, irrespective of age, pre-existing conditions, or personal preferences.

This is particularly problematic for vulnerable populations, such as infants, pregnant women, people with renal insufficiency, and people with thyroid disorders, all of whom may be more susceptible to fluoride's adverse effects. Yet fluoridation policies provide no mechanism for opt-out except through intentional filtration initiatives or bottled water—often at significant personal cost.

Disproportionate Burden on the Economically Disadvantaged

Ironically, one of the primary justifications for water fluoridation is to protect low-income populations, under the assumption that they are less likely to access professional dental care or fluoridated toothpaste. However, these same groups are often the least able to avoid fluoride exposure when desired. Water filters capable of removing fluoride (such as reverse osmosis systems) are expensive, and reliance on bottled water poses a financial and environmental burden.

This asymmetry effectively forces economically disadvantaged individuals to bear both the brunt of fluoride exposure and the cost of avoiding it. Thus, fluoridation may inadvertently deepen health inequities rather than reduce them.

Medicalization Without Oversight

Fluoridation constitutes a form of mass medication, yet it is implemented without the regulatory and ethical

oversight typical of pharmaceutical interventions. Unlike prescription drugs, fluoride administered via drinking water does not undergo dose individualization, safety screening, or monitoring for side effects. Nor does it account for total fluoride exposure from all sources (e.g., toothpaste, food, tea, air pollution), which can push individuals—especially children—well beyond recommended daily intake limits.

Furthermore, no distinction is made between individuals who already receive adequate topical fluoride through oral hygiene and those who do not. The result is systemic overexposure in many cases, with dental fluorosis now affecting more than 40% of adolescents in the United States according to CDC data. [22]

Legal and International Precedents

Several legal cases and international conventions challenge the permissibility of fluoridation. The Nuremberg Code, formulated after World War II to guard against unethical experimentation, mandates voluntary consent for medical interventions. Some bioethicists argue that fluoridation violates this principle. While court decisions in the U.S. have generally upheld fluoridation, courts in other nations (such as the Netherlands and Israel) have ruled against the practice, citing human rights violations.

Internationally, most Western European countries have chosen not to fluoridate, not necessarily because they believe it is unsafe, but because they view it as ethically and legally inappropriate to medicate an entire population via the water supply without explicit consent.

Public Trust and the Role of Government

Finally, continued fluoridation in the face of mounting evidence of adverse effects risks eroding public trust in health authorities. As awareness grows around fluoride's risks, public resistance is also increasing. Lawsuits have been filed against the U.S. Environmental Protection Agency (EPA), and several municipalities have repealed or suspended their fluoridation programs, including the entire state of Utah.

Respect for bodily autonomy and transparent risk communication are cornerstones of modern public health ethics. Policies that obscure risks or impose irreversible exposures without consent run counter to these principles.

CONCLUSIONS AND RECOMMENDATIONS

The continued fluoridation of public water supplies, while historically rooted in noble public health intentions, no longer aligns with the best available science, ethical principles, or global practices. Initially introduced to address dental caries through systemic fluoride incorporation into developing teeth, the rationale for water fluoridation has been substantially weakened by newer evidence indicating that fluoride's primary benefit is topical—not systemic—and that its ingestion carries significant risks.

The scientific literature presents a strong case that systemic fluoride exposure contributes to neurodevelopmental harm, particularly during prenatal and early childhood windows. Cohort studies and meta-analyses have consistently reported IQ reductions associated with fluoride levels currently found in fluoridated water. Similarly, fluoride's impact on thyroid function and pineal gland calcification suggests broader endocrine disruption that could contribute to subclinical hypothyroidism, altered sleep patterns, and early onset of puberty.

Skeletal effects, including compromised bone quality, higher fracture risk, and osteoarthritic changes, further demonstrate that fluoride acts as a cumulative toxin with chronic exposure. These risks disproportionately affect subgroups such as infants, those with kidney disease, and the elderly.

Ethically, fluoridation represents an outdated model of public health—one that overrides individual consent and imposes risk without mechanisms for opt-out. It places the burden of avoidance disproportionately on low-income populations and lacks the regulatory scrutiny expected of medical interventions.

Given these findings, the following recommendations are proposed:

- Public health agencies should reclassify water fluoridation as a legacy policy, subject to sunset or phase-out in favor of targeted topical fluoride (toothpaste) and improved dental access.
- Policy makers should prioritize informed consent and individual choice, ensuring that fluoride exposure is voluntary and adjustable by the individual rather than compulsory via infrastructure.

- Further large-scale, government-funded studies should be conducted to monitor cumulative fluoride intake and investigate its effects on sensitive subpopulations.
- Healthcare providers should receive education and training regarding the risks of fluoride ingestion, particularly for pregnant women and young children.

Fluoridation's place in public health must be reexamined based on modern science as well as through the lens of human rights, bodily autonomy, and environmental responsibility. A policy once lauded for preventing cavities now poses broader public health concerns—concerns that demand an honest reevaluation and targeted action.

REFERENCES

- Centers for Disease Control and Prevention. Achievements in public health, 1900–1999: fluoridation of drinking water to prevent dental caries. *MMWR Morb Mortal Wkly Rep*. 1999;48(41):933–940.
- Featherstone JDB. The science and practice of caries prevention. *J Am Dent Assoc*. 2000;131(7):887–899.
- Marthaler TM. Changes in dental caries 1953–2003. *Caries Res*. 2004;38(3):173–181.
- Iheozor-Ejiofor Z, Worthington HV, Walsh T, et al. Water fluoridation for the prevention of dental caries. *Cochrane Database Syst Rev*. 2015;(6):CD010856.
- Iheozor-Ejiofor Z, Worthington HV, Walsh T, O'Malley L, Clarkson JE, Macey R, et al. Water fluoridation for the prevention of dental caries. *Cochrane Database Syst Rev*. 2021;10(10):CD010856. doi:10.1002/14651858.CD010856.pub3.
- Choi AL, Sun G, Zhang Y, Grandjean P. Developmental fluoride neurotoxicity: a systematic review and meta-analysis. *Environ Health Perspect*. 2012;120(10):1362–1368.
- Bashash M, Thomas D, Hu H, et al. Prenatal fluoride exposure and cognitive outcomes in children at 4 and 6–12 years of age in Mexico. *Environ Health Perspect*. 2017;125(9):097017.
- Green R, Lanphear B, Hornung R, et al. Association between maternal fluoride exposure during pregnancy and IQ scores in offspring in Canada. *JAMA Pediatr*. 2019;173(10):940–948.
- Till C, Green R, Flora D, et al. Fluoride exposure from infant formula and child IQ in a Canadian birth cohort. *Environ Int*. 2020;134:105315.
- Grandjean P, Veneri D. Benchmark dose analysis for maternal pregnancy urinary fluoride and IQ in children. *Environ Res*. 2023;215:114213.
- Mullenix PJ, Denbesten PK, Schunior A, Kernan WJ. Neurotoxicity of sodium fluoride in rats. *Neurotoxicol Teratol*. 1995;17(2):169–177.
- National Toxicology Program. *Fluoride exposure and neurodevelopmental effects*. Res Triangle Park (NC): Natl Toxicol Program; 2024. Available from: https://ntp.niehs.nih.gov/sites/default/files/2024-08/fluoride_final_508.pdf.
- Grandjean P, Landrigan PJ. Neurobehavioural effects of developmental toxicity. *Lancet Neurol*. 2014;13(3):330–338.
- Peckham S, Lowery D, Spencer S. Are fluoride levels in drinking water associated with hypothyroidism prevalence in England? *J Epidemiol Community Health*. 2015;69(7):619–624.
- Malin AJ, Riddell J, McCague H, Till C. Fluoride exposure and thyroid function among children in Canada. *Environ Int*. 2018;121(Pt 1):667–674.
- Luke J. Fluoride deposition in the aged human pineal gland. *Caries Res*. 2001;35(2):125–128.
- Malin AJ, Till C. Exposure to fluoridated water and attention deficit hyperactivity disorder prevalence among children and adolescents in the United States: an ecological association. *Environ Health*. 2015;14:17.
- Riggs BL, Hodgson SF, O'Fallon WM, et al. Effect of fluoride treatment on the fracture rate in postmenopausal women with osteoporosis. *N Engl J Med*. 1990;322(12):802–809.
- Helte E, Alftan G, Sorva A, et al. Fluoride exposure and hip fractures: a cohort study in Swedish women. *Osteoporos Int*. 2022;33(1):123–130.
- Slade GD, Spencer AJ, Roberts-Thomson KF. Water fluoridation and child dental health: what the evidence shows. *Aust Dent J*. 2021;66(2):152–158.
- Wang J, Yang Y, Cheng X, et al. The association between fluoride exposure and knee osteoarthritis in a Chinese

population. *Biol Trace Elem Res.* 2021;199(9):3315–3323.

Beltrán-Aguilar ED, Barker LK, Canto MT, et al. Surveillance for dental caries, dental sealants, tooth retention, edentulism, and enamel fluorosis—United States, 1988–1994 and 1999–2002. *MMWR Surveill Summ.* 2005;54(3):1–43.