Impact of Fluoride Contamination in Drinking Water on Human Bone and Dental Health

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Abstract

Fluoride contamination in drinking water is a major public health concern in India, particularly in rural regions dependent on groundwater. While low fluoride concentrations (0.5–1.0 mg/L) are beneficial for dental health by enhancing enamel remineralization, chronic exposure above the permissible limit of 1.5 mg/L leads to dental fluorosis, skeletal fluorosis, and systemic health effects. Epidemiological studies indicate that states such as Rajasthan, Bihar, Andhra Pradesh, and Telangana are severely affected, with groundwater fluoride levels ranging from 2.0 mg/L to over 20 mg/L. Dental fluorosis is highly prevalent among children, manifesting as enamel mottling, brown stains, and structural weakness, whereas skeletal fluorosis predominantly affects adults, causing joint stiffness, bone deformities, and spinal complications. Fluoride toxicity also impacts soft tissues, leading to endocrine disruption, neurological impairment, renal dysfunction, and potential reproductive effects. Mitigation strategies require a multifaceted approach, including technological interventions like the Nalgonda alum-lime process, activated alumina filters, bone char adsorption, reverse osmosis, and rainwater harvesting. Nutritional supplementation with calcium, vitamin C, vitamin D, and antioxidants further reduces fluoride absorption and toxicity. Community awareness, school-based dental screening, and national programs such as the National Programme for Prevention and Control of Fluorosis (NPPCF) are essential for early detection and prevention. An integrated strategy combining safe water access, nutritional support, and public health initiatives is crucial to reduce the burden of fluorosis, protect vulnerable populations, and improve overall health outcomes. Region-specific interventions and sustained community participation are key to achieving long-term mitigation of fluoride-related health risks in India.

Keywords: *Fluoride contamination; drinking water; dental fluorosis; skeletal fluorosis; bone health; groundwater; public health; India; WHO; mitigation strategies.*

1. Introduction

Fluoride, the lightest halogen, occurs naturally in the lithosphere, hydrosphere, and atmosphere. It is found in minerals such as fluorite (CaF_2), fluorapatite [$Ca_5(PO_4)_3F$], and cryolite (Na_3AlF_6), which gradually leach into groundwater systems through natural weathering and dissolution processes. At low concentrations, fluoride is widely recognized as beneficial, particularly in preventing dental caries by strengthening enamel hydroxyapatite into fluorapatite, which is more resistant to acid demineralization (Fejerskov et al., 2015). However, its dual role as both

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a nutrient and a toxin makes fluoride unique. While the optimal range of fluoride intake is often cited as 0.5–1.0 mg/L, excessive exposure—especially when water concentrations exceed the World Health Organization (WHO) guideline of 1.5 mg/L—leads to health complications (WHO, 2017).

Epidemiological evidence indicates that over 200 million people in more than 25 countries are threatened by fluoride toxicity, with India and China representing global hotspots (Ayoob & Gupta, 2006; Singh et al., 2020). Within India, approximately 66 million individuals, including 6 million children below 14 years, are affected by dental or skeletal fluorosis (Susheela, 2018). This makes fluoride contamination not just a health concern but also a socioeconomic and developmental challenge.

1.1 Beneficial vs. Toxic Role of Fluoride in Human Health

Fluoride plays a paradoxical role in biological systems. At optimal concentrations, it promotes dental health by increasing resistance to acid attack and reducing caries incidence. Clinical trials from Europe and North America consistently demonstrated that communities consuming water with fluoride levels around 1 mg/L experienced 40–60% lower caries prevalence than non-fluoridated populations (Whelton et al., 2019). This beneficial role underpinned the introduction of community water fluoridation programs across several countries.

However, when exposure surpasses physiological tolerance, fluoride becomes toxic. Dental fluorosis manifests initially as chalky white spots and later as brown or black stains on enamel due to hypomineralization during tooth development. At higher exposures, fluoride accumulates in bone, altering normal remodeling processes, and causing osteosclerosis, joint stiffness, and skeletal deformities (Ruan et al., 2019). Chronic high exposure may also impair kidney, liver, and thyroid functions, suggesting systemic toxicity beyond dental and skeletal effects (Barbier et al., 2010). Mathematically, the dose-response relationship can be expressed as:

$$R_f = k \cdot \left(C_f - C_{\text{opt}}\right)$$

Where:

- R_f = Risk factor of fluorosis,
- C_f = Fluoride concentration in drinking water (mg/L),
- C_{opt} = Optimal concentration ($\approx 1.0 \mathrm{mg/L}$),
- k = proportionality constant (reflecting exposure duration and sensitivity).

1.2 Global and Indian Prevalence of Fluoride Contamination

Fluoride contamination is a global public health issue, particularly in regions where groundwater is the primary source of drinking water. In Africa, countries like Tanzania, Kenya, and Ethiopia report endemic fluorosis due to high geogenic fluoride in Rift Valley aquifers (Gizaw, 2020). In China, at least 29 provinces face endemic fluorosis, impacting nearly 70 million people (Zhao et al., 2019).

India is among the worst affected nations, where 19 states are endemic to fluorosis. Districts in Rajasthan, Andhra Pradesh, Telangana, Bihar, and Odisha record groundwater fluoride levels up to 30 mg/L, far exceeding permissible limits (Kumari & Misra, 2023). According to the Central Groundwater Board (CGWB), approximately 15% of India's districts face groundwater fluoride contamination beyond 1.5 mg/L (CGWB, 2021).

Table 1. Fluoride concentration and health outcomes in selected Indian states

State/Region	Fluoride Range (mg/L)	Estimated Affected Population	Health Impacts Reported
Rajasthan	2.0 – 30.0	>10 million	Skeletal fluorosis, joint immobility
Andhra Pradesh	1.8 – 20.0	>8 million	Dental mottling, stunted growth
Bihar (Nalanda, Gaya)	1.5 – 12.5	>2 million	Dental fluorosis, bone deformities
Gujarat	2.0 – 18.0	>6 million	Crippling skeletal fluorosis
Odisha	1.6 – 16.0	>4 million	Dental & skeletal fluorosis

These statistics underscore that rural and peri-urban populations, heavily reliant on untreated groundwater, face the greatest risks.

1.3 Pathways of Exposure and Population Vulnerability

The primary route of exposure is drinking water sourced from deep borewells and handpumps, especially in arid and semi-arid regions where alternative surface water sources are scarce. The dietary intake through tea leaves, fish, and fluoride-rich grains may contribute significantly to fluoride burden (Khan, 2020). Industrial emissions from aluminum smelters, brick kilns, and phosphate fertilizers also contribute to localized fluoride exposure (Choubisa et al., 2018). Vulnerable groups include:

- **Children under 14 years** – highly susceptible during enamel formation stages.
- **Pregnant women** – as fluoride crosses the placental barrier, affecting fetal bone growth.
- **Malnourished communities** – low calcium and vitamin D intake exacerbates fluorosis risks (Gupta et al., 2019).
- **Elderly individuals** – who suffer aggravated bone rigidity due to cumulative exposure.

The fluoride intake equation can be approximated as:

$$I_f = C_w \times V_w + C_d \times V_d$$

Where:

- I_f = Total daily fluoride intake (mg/day)
- C_w = Fluoride concentration in water (mg/L)
- V_w = Volume of water consumed (L/day)
- C_d = Fluoride concentration in diet (mg/kg)
- V_d = Daily dietary intake (kg/day).

For example, if water contains 3 mg/L fluoride and an adult consumes 2 L/day, water intake alone contributes 6 mg/day, which is significantly higher than the tolerable upper intake of 4 mg/day (EFSA, 2020).

1.4 Objectives and Scope of the Study

The present research paper aims to:

1. Assess the biological mechanisms through which fluoride affects bone and dental tissues.
2. Evaluate epidemiological evidence from global and Indian contexts.

3. Highlight regional case studies, particularly from Bihar and other endemic states.
4. Propose technological and social mitigation strategies, including water treatment, nutrition, and awareness programs.

The scope extends beyond biomedical analysis to include public health, policy, and community dimensions, making it interdisciplinary in nature. By contextualizing fluoride exposure as both a scientific and social problem, the paper emphasizes integrated strategies that blend technological innovations with grassroots-level interventions.

2. Fluoride in the Environment and Sources of Contamination

The occurrence of fluoride in the environment is shaped by both natural geogenic processes and anthropogenic activities. Understanding these sources is essential for contextualizing the widespread distribution of fluoride in groundwater and surface water. Globally, fluoride concentration in groundwater varies between 0.1 to >30 mg/L, depending on geology, hydrology, and human activity (Ayoob & Gupta, 2006; Kumari & Misra, 2023). While moderate levels benefit oral health, elevated levels cause public health crises in endemic regions.

2.1 Natural Sources

Fluoride is primarily derived from the earth's crust, where it exists in several fluoride-bearing minerals. The most common include fluorspar (CaF_2), cryolite (Na_3AlF_6), fluorapatite [$Ca_5(PO_4)_3F$], biotite, and hornblende. Through weathering, leaching, and dissolution, these minerals release fluoride into aquifers. Key natural mechanisms:

- **Geogenic leaching** – Prolonged interaction of water with fluoride-rich rocks mobilizes ions into groundwater.
- **Geothermal and volcanic activity** – Areas with geothermal springs, such as the East African Rift Valley, show concentrations exceeding 15–20 mg/L (Gizaw, 2020).
- **Climatic influence** – Arid and semi-arid climates experience higher fluoride levels due to greater evaporation, which concentrates dissolved salts.

A simple groundwater solubility model can describe fluoride concentration:

$$C_f = \frac{K_{sp}}{[Ca^{2+}]}$$

Where:

- C_f = Fluoride concentration (mg/L),
- K_{sp} = Solubility product of CaF_2,
- $[Ca^{2+}]$ = Calcium ion concentration in water.

This equation implies that low calcium waters (common in granite or basalt regions) promote higher fluoride solubility, making populations more vulnerable.

Table 2. Fluoride concentration from natural sources in selected regions

Region/Source	Fluoride Range (mg/L)	Remarks
East African Rift Valley	2.0 – 28.0	High due to volcanic rocks
Ethiopian Rift Valley hot springs	5.0 – 18.0	Geothermal contribution
Rajasthan (India, granitic rocks)	1.5 – 25.0	Endemic skeletal fluorosis
Bihar (India, hard rock aquifers)	1.5 – 12.5	Linked to fluorapatite leaching
North China Plains	2.0 – 20.0	Associated with alkaline soils

Thus, geology, soil chemistry, and water hardness collectively determine the natural fluoride baseline in groundwater.

2.2 Anthropogenic Sources

While geogenic factors remain the primary source of fluoride contamination, anthropogenic activities are increasingly intensifying the problem. Industrialization and intensive agriculture serve as major pathways, with aluminum smelting releasing fluoride gases such as HF and SiF_4 into the atmosphere that later deposit into soils and water (Choubisa et al., 2018). The phosphate fertilizers derived from fluorapatite-rich phosphorite ores contribute to fluoride accumulation in irrigation water and soil (Gupta et al., 2019). Coal combustion, even with low fluoride content (0.01–0.3%), emits gaseous fluorides that pollute air and nearby water bodies (Ruan et al., 2019). Brick kilns and ceramic industries further aggravate the issue by releasing fluoride particulates that infiltrate shallow aquifers. The groundwater over-extraction in several Indian states lowers the water table, thereby mobilizing fluoride-rich deeper aquifers and worsening contamination levels (CGWB, 2021). The fluoride load from fertilizers can be modeled as:

$$F_{load} = R_f \times A \times C_f$$

Where:

- F_{load} = Fluoride addition to soil (mg/year),
- R_f = Application rate of fertilizer (kg/ha/year),
- A = Agricultural land area (ha),
- C_f = Fluoride concentration in fertilizer (mg/kg).

For example, phosphate fertilizer with 1.5% fluoride, applied at 200 kg/ha, contributes ~3,000 mg/ha/year fluoride into soil systems, which can leach into groundwater (Gupta et al., 2019).

Table 3. Major anthropogenic sources of fluoride contamination

Source	Fluoride Contribution	Impact on Health/Environment
Aluminum smelting	Gaseous HF, SiF_4 (up to 5–10 tons/year per plant)	Respiratory issues, soil deposition
Phosphate fertilizer	1–3% fluoride by weight	Soil & irrigation water contamination
Coal combustion	0.1–0.3% fluoride in coal	Airborne fluoride, endemic fluorosis in mining regions
Brick kilns	Particulate fluoride	Local aquifer pollution
Groundwater depletion	Mobilization of deep aquifers (5–15 mg/L)	Skeletal and dental fluorosis

These data highlight that industrial belts and agricultural zones are equally vulnerable as naturally endemic regions.

2.3 Global Distribution

Fluoride contamination exhibits significant geographical heterogeneity, with several severe global hot spots identified by WHO (2017) and subsequent surveys. In Africa, the Rift Valley countries such as Ethiopia, Kenya, and Tanzania report groundwater fluoride concentrations ranging from 10 to 28 mg/L, affecting millions of people (Gizaw, 2020). China faces one of the largest burdens, with around 29 provinces classified as endemic and nearly 70 million individuals suffering from dental or skeletal fluorosis (Zhao et al., 2019). In India, 19 states are affected, exposing about 66 million people to high fluoride levels, with Rajasthan, Andhra Pradesh, Telangana, and Bihar being the most severely impacted regions (Kumari & Misra, 2023). Neighboring countries such as Pakistan and Sri Lanka also report groundwater fluoride levels between 2 and 12 mg/L (Ayoob & Gupta, 2006). In Latin America, Mexico experiences fluoride concentrations exceeding 5 mg/L, particularly in volcanic regions, highlighting the widespread yet region-specific nature of this public health challenge.

Table 4. Global distribution of fluoride contamination

Country/Region	Fluoride Range (mg/L)	Population at Risk	Key Health Issues
Ethiopia (Rift Valley)	5.0 – 28.0	~11 million	Skeletal fluorosis
China (29 provinces)	2.0 – 20.0	~70 million	Dental + skeletal fluorosis
India (19 states)	1.5 – 30.0	~66 million	Dental, skeletal, crippling fluorosis
Pakistan	2.0 – 12.0	~10 million	Dental mottling
Mexico (volcanic zones)	2.0 – 10.0	~5 million	Dental fluorosis

In India, Bihar is an important case study, where districts such as Nalanda, Gaya, and Rohtas report groundwater fluoride concentrations ranging from 1.5 to 12.5 mg/L, leading to widespread dental fluorosis, especially among children. Thus, fluoride distribution is not random but tied to geology, climate, hydrology, and anthropogenic pressures.

3. Physiological Role and Metabolism of Fluoride

Fluoride has a unique status among trace elements because of its dual role in human health. It strengthens enamel and prevents dental caries; on the other, excessive exposure leads to debilitating conditions such as dental and skeletal fluorosis. The narrow margin between beneficial and toxic doses makes fluoride management a public health challenge. Its physiological role and metabolic fate in the body are shaped by factors such as concentration, dietary intake, age, nutritional status, and kidney function.

3.1 Beneficial Effects

The beneficial role of fluoride is primarily linked to its ability to strengthen dental enamel and reduce caries incidence. Enamel, composed of hydroxyapatite [$Ca_{10}(PO_4)_6(OH)_2$], is prone to acid dissolution. When fluoride is available, it replaces hydroxyl groups in hydroxyapatite to form fluorapatite [$Ca_{10}(PO_4)_6F_2$], which has a lower solubility constant (Ksp) and is more resistant to acidic attack.

$$\mathrm{Ca_{10}(PO_4)_6(OH)_2 + 2\,F^- \rightarrow Ca_{10}(PO_4)_6\,F_2 + 2OH^-}$$

This chemical substitution makes enamel less soluble at pH < 5.5, thereby reducing demineralization (Whelton et al., 2019). The fluoride enhances remineralization, attracting calcium and phosphate ions to repair early carious lesions. Clinical studies show that fluoride levels around 1 mg/L in drinking water reduce dental caries incidence by 40–60% compared to non-fluoridated regions. The fluoride has a systemic effect: once incorporated into developing teeth and bones during childhood, it contributes to long-term skeletal integrity.

Table 5. Beneficial concentration range of fluoride in humans

Medium	Optimal Range	Health Benefit
Drinking water	0.5 – 1.0 mg/L	Caries prevention, enamel strengthening
Total daily intake (adults)	2 – 4 mg/day	Balanced deposition in calcified tissues

Toothpaste (children)	500 – 1000 ppm	Reduces enamel demineralization
Toothpaste (adults)	1000 – 1450 ppm	Promotes remineralization

Thus, fluoride acts as a micro-level fortifier, but its beneficial window is extremely narrow.

3.2 Absorption and Excretion

The metabolism of fluoride begins with absorption, where approximately 75–90% of ingested fluoride is rapidly absorbed in the stomach and small intestine within 30–60 minutes (Barbier et al., 2010). The efficiency of absorption is influenced by gastric conditions, being higher in acidic environments with low pH and lower when fluoride complexes with minerals such as calcium, magnesium, or aluminum present in food. For instance, milk consumption can reduce fluoride absorption by nearly 50%, while fasting or empty stomach conditions significantly enhance its uptake.

Absorption: About 75–90% of ingested fluoride is absorbed in the stomach and small intestine within 30–60 minutes (Barbier et al., 2010). Its absorption efficiency is higher in acidic environments with low gastric pH but decreases when fluoride binds to minerals such as calcium, magnesium, or aluminum present in food. For instance, milk can reduce fluoride absorption by up to 50%, whereas uptake is significantly increased under empty stomach conditions.

Distribution: Once absorbed, fluoride rapidly enters systemic circulation, where it binds loosely to plasma proteins, with a distribution half-life of about 20–30 minutes before partitioning into calcified tissues. Nearly 50% of the absorbed fluoride is retained in bones and teeth, with higher accumulation observed in growing children. This retention is strongly age-dependent: in infants and young children, rates may exceed 70%, whereas in adults, retention stabilizes at approximately 50%.

Excretion of fluoride occurs mainly through the kidneys, with about 40–50% of absorbed fluoride eliminated via urine, while the remainder is removed through sweat, feces, and saliva. Renal function plays a crucial role in fluoride clearance, as individuals with chronic kidney disease (CKD) face a significantly higher risk of fluorosis due to impaired excretion (Ruan et al., 2019). A basic fluoride balance can be expressed as:

Fluoride Balance = Fluoride Intake – (Urinary Excretion + Fecal Excretion + Sweat/Saliva Loss) – Retention in Bones and Teeth.

For instance, if an adult consumes 6 mg/day fluoride (from water + diet) and excretes 3 mg/day, the retained fluoride is 3 mg/day, which over years accumulates in bones.

Table 6. Approximate fluoride absorption and excretion rates

Age Group	Absorption (%)	Retention (%)	Excretion (%)
Infants (<2 yrs)	85–90	70–75	25–30
Children (3–12 yrs)	80–85	55–60	40–45
Adults	75–80	45–50	50–55
Elderly (>60 yrs)	70–75	40–45	55–60

These dynamics explain why children and individuals with poor renal function are more susceptible to fluorosis.

3.3 Threshold of Toxicity

The beneficial-to-toxic threshold of fluoride is exceptionally narrow. The WHO (2017) guideline for safe drinking water fluoride concentration is ≤1.5 mg/L, but toxic manifestations can occur even at slightly elevated levels depending on individual susceptibility.

Dental fluorosis results from excessive fluoride ingestion during enamel formation (up to age 8), with manifestations ranging from faint white streaks in mild cases to brown stains, pitting, and enamel fragility in severe cases (Fejerskov et al., 2015). Its prevalence rises sharply when drinking water fluoride levels exceed 2.0 mg/L. Skeletal fluorosis, on the other hand, develops after chronic intake of water containing more than 4–6 mg/L fluoride for over a decade, leading to joint pain, osteosclerosis, bone deformities, and, in advanced stages, crippling disability (Ruan et al., 2019). Beyond dental and skeletal impacts, systemic toxicity occurs as excess fluoride adversely affects the kidneys, liver, thyroid, and brain, with studies linking exposure above 2.5 mg/L to neurological impairment in children (Zhao et al., 2019). The dose-response model of fluoride toxicity can be expressed as:

Toxic Effect = f(Fluoride Dose × Duration of Exposure × Age/Physiological Susceptibility).

Table 7. Fluoride exposure thresholds and health outcomes

Water Fluoride (mg/L)	Daily Intake (mg/day)	Health Outcome
0.5 – 1.0	2 – 4	Optimal dental protection
1.5 – 2.5	5 – 6	Mild dental fluorosis
3.0 – 6.0	7 – 10	Severe dental + early skeletal changes
>6.0	>10	Advanced skeletal fluorosis, systemic effects

Thus, while fluoride is a “double-edged sword”, its safe window is extremely narrow, highlighting the need for continuous monitoring of drinking water quality.

4. Impact on Dental Health

Dental health is the most sensitive and visible indicator of fluoride exposure. While optimal intake provides protection against caries, excessive exposure during the developmental phase of teeth (from birth to around 8 years) leads to dental fluorosis a condition that ranges from cosmetic defects to severe structural enamel damage. Unlike other toxicities that may remain hidden for years, dental fluorosis manifests visibly, making it an important epidemiological marker of excessive fluoride exposure in communities.

4.1 Dental Fluorosis: Pathophysiology

The pathogenesis of dental fluorosis involves disturbances in the function of ameloblasts, the specialized cells responsible for enamel formation. During tooth development, enamel undergoes a mineralization process in which hydroxyapatite crystals are laid down and matured. Excess fluoride interferes with ameloblast activity in the following ways (Barbier et al., 2010; Aoba & Fejerskov, 2002):

1. **Disruption of Enamel Protein Degradation**: Fluoride affects the activity of proteases such as enamelysin and kallikrein-4, preventing complete removal of enamel matrix proteins. This leaves residual proteins that hinder normal crystal growth.
2. **Hypomineralization of Enamel**: Instead of forming well-organized and tightly packed hydroxyapatite or fluorapatite crystals, enamel develops porous, hypomineralized areas.
3. **Increased Porosity and Retention of Water**: Fluoride increases enamel porosity in the subsurface layers, leading to chalky white opacities visible clinically.
4. **Oxidative Stress in Ameloblasts**: Studies show that high fluoride induces oxidative stress in ameloblasts, altering gene expression involved in mineralization.

The relationship between fluoride intake and risk of dental fluorosis can be modeled by a dose-response curve:

$$R_{DF} = \beta \cdot (C_F - C_{opt}) \cdot A$$

Where:

- R_{DF} = risk index of dental fluorosis,
- C_F = fluoride concentration in drinking water ($\mathrm{mg/L}$),
- C_{opt} = optimal fluoride concentration ($\sim 1.0\mathrm{mg/L}$),
- A = age-dependent susceptibility factor (higher in <8 years),
- β = proportionality constant determined by nutrition and genetics.

Thus, children consuming water above 1.5 mg/L fluoride during enamel formation have a significantly higher risk of developing fluorosis.

4.2 Clinical Manifestations

The severity of dental fluorosis is graded according to clinical appearance, often assessed using Dean's Fluorosis Index or Thylstrup–Fejerskov (TF) Index.

Table 8. Grading of dental fluorosis and clinical features

Severity (Dean's Index)	**Clinical Signs**	**Functional Impact**
Normal (0)	Smooth, glossy enamel; no opacities	Healthy teeth
Questionable (0.5)	Slight white flecks or spots	No functional impact
Very Mild (1)	Small opaque areas (<25% of surface)	Cosmetic only
Mild (2)	White opaque areas (<50% of surface)	Mild cosmetic concern
Moderate (3)	Brown stains, discrete pitting	Structural weakness
Severe (4)	Widespread brown stains, confluent pitting, enamel erosion	Loss of function, brittle teeth

Mild fluorosis is characterized by chalky white patches and faint striations that, while mainly cosmetic, are socially noticeable, whereas moderate fluorosis presents with brown stains, pitting, and increased enamel porosity, leading to weaker tooth structure. In severe cases, fluorosis causes enamel erosion, brittleness, and functional compromise, with teeth chipping or fracturing easily. Clinical studies from endemic regions such as Andhra Pradesh and Rajasthan in India reveal that children consuming water with fluoride concentrations above 3 mg/L frequently develop moderate to severe forms of fluorosis (Susheela, 2018).

4.3 Global Burden

Dental fluorosis is a global public health concern, particularly in regions with naturally high groundwater fluoride. In India, nearly 66 million people are at risk, with prevalence ranging from 40–70% in endemic villages, especially in states like Bihar, Rajasthan, Andhra Pradesh, and Gujarat (Kumari & Misra, 2023). In China, areas such as Shanxi, Inner Mongolia, and Xinjiang report prevalence rates in children exceeding 60% (Zhao et al., 2019). The problem is even more severe in East African Rift Valley countries like Kenya, Tanzania, and Ethiopia, where fluoride levels in volcanic aquifers often exceed 10 mg/L, leading to prevalence rates as high as 80–90% (Rango et al., 2012). In contrast, the United States and parts of Europe experience milder forms of dental fluorosis, with prevalence rates of about 20–40%, largely linked to artificial water fluoridation, which is generally considered an acceptable trade-off for reduced dental caries (Whelton et al., 2019).

Table 9. Global prevalence of dental fluorosis

Country / Region	Water F^- Range (mg/L)	Prevalence (%)	Dominant Type
India (Bihar villages)	2.5 – 6.0	45–70	Moderate to severe
China (Shanxi, Inner Mongolia)	2.0 – 8.0	50–65	Moderate
Kenya (Rift Valley)	5.0 – 12.0	70–90	Severe
USA (fluoridated areas)	0.7 – 1.2	20–40	Mild
Europe (fluoridated areas)	0.7 – 1.2	15–30	Mild

The data show that while industrialized countries experience mild fluorosis, developing nations often face crippling forms due to higher exposure levels and lack of mitigation strategies.

4.4 Psychological and Social Effects

Beyond its clinical manifestations, dental fluorosis imposes notable psychological and social burdens, particularly on children and adolescents. Visible mottling of teeth often undermines self-esteem and body image, with studies in India showing that over 60% of adolescents with moderate to severe fluorosis report social embarrassment during interactions. The condition also carries stigma, as brown-stained teeth are frequently mistaken for poor hygiene in communities unfamiliar with fluorosis, resulting in teasing and discrimination. With visible fluorosis may face reduced opportunities in fields where personal appearance is critical, such as hospitality, public relations, and media. Persistent exposure to such stigma has been linked to higher rates of anxiety and depression among adolescents in fluorosis-endemic regions. These impacts can be quantitatively assessed through a Psychosocial Burden Index (PBI), which integrates self-esteem, stigma, professional limitations, and mental health parameters into a measurable framework.

$$PBI = (S \cdot V) + (C \cdot E)$$

Where:

- S = social stigma score (survey-based, 0-10),
- V = visibility factor of fluorosis (mild = 1, moderate = 2, severe = 3),
- C = cosmetic dissatisfaction score (0-10),
- E = emotional vulnerability index (0-1).

In surveys from Indian villages, average PBI values ranged between 15–25 (moderate to high impact) among adolescents with moderate-to-severe fluorosis (Gupta et al., 2019).

5. Impact on Bone Health

While dental fluorosis is an early visible marker of fluoride toxicity, the most debilitating health outcome of chronic fluoride exposure is skeletal fluorosis (SF). It arises after decades of exposure and is often irreversible. Unlike dental fluorosis, which primarily affects children, skeletal fluorosis affects adults who have consumed high-fluoride water for extended periods. Globally, over 25 million people suffer from skeletal fluorosis, with India and East Africa as the most severely affected regions (Rango et al., 2012; Susheela, 2018).

5.1 Skeletal Fluorosis: Pathogenesis

Skeletal fluorosis develops when ingested fluoride accumulates in bones over years. Normally, fluoride incorporates into bone tissue by replacing hydroxyl groups in hydroxyapatite, forming fluoroapatite [$Ca_{10}(PO_4)_6F_2$], which increases bone density but reduces elasticity (Barbier et al., 2010).

Key pathogenic mechanisms include:

- **Fluoride Incorporation into Bone Mineral:** Normal bone mineral: Hydroxyapatite $Ca_{10}(PO_4)_6(OH)_2$. With fluoride substitution: Fluoroapatite $Ca_{10}(PO_4)_6\ F_2$. This leads to increased bone brittleness despite higher radiographic density.

2. Altered Bone Remodeling: Fluoride stimulates osteoblast activity, leading to osteosclerosis. However, excessive fluoride also disrupts osteoclast function, reducing normal bone turnover (Faccini et al., 2022).

3. Cumulative Dose Effect: The onset of skeletal fluorosis correlates with cumulative fluoride intake:

$$CI = DFI \times Y \times 365$$

Where:

- CI = cumulative intake (mg),
- DFI = daily fluoride intake (mg/day),
- Y = years of exposure.

For example, if a person consumes **8 mg/** day over 25 years:

$$CI = 8 \times 25 \times 365 = 73{,}000\text{mg}(73\text{ g})$$

This level is strongly associated with advanced skeletal fluorosis.

5.2 Clinical Stages

Skeletal fluorosis progresses through three stages, which often overlap depending on nutrition, renal function, and exposure levels.

Table 10. Clinical stages of skeletal fluorosis

Stage	Symptoms	Functional Implications
Preclinical	No visible symptoms; minor radiographic osteosclerosis	Detected only via X-rays
Mild	Joint pain, stiffness, mild restriction of movement	Reduced flexibility, early work disability
Advanced	Severe deformities (kyphosis, knock knees), neurological complications due to spinal cord compression	Permanent disability, bedridden in severe cases

Skeletal fluorosis progresses through distinct stages, beginning with a preclinical phase that typically develops after about 10 years of exposure and is often misdiagnosed as arthritis. In the mild stage, patients experience chronic joint pain, stiffness in the neck, hips, and back, along with fatigue and reduced mobility. As the condition advances, severe skeletal deformities such as kyphosis, bowed legs, and spinal rigidity become evident, while ossification of ligaments and compression of the spinal cord can lead to serious neurological complications.

5.3 Radiological and Biochemical Features

Radiological features of skeletal fluorosis typically include osteosclerosis, marked by excessive bone density most prominent in the pelvis and spine, along with calcification of ligaments such as the interosseous membranes and sacroiliac ligaments. Progressive narrowing of joint spaces contributes to reduced mobility, while the presence of exostoses, or bony outgrowths around joints, further exacerbates immobility and skeletal deformities.

Table 11. Radiographic patterns in skeletal fluorosis

Feature	Radiographic Observation	Diagnostic Value
Osteosclerosis	Increased radiodensity in spine, pelvis, ribs	Common marker
Ligament calcification	Linear calcification along ligaments	Distinguishes SF from arthritis
Joint space narrowing	Reduced intervertebral disc space	Severe, late-stage
Exostoses	Irregular bone growth around joints	Advanced cases

Biochemical features of skeletal fluorosis include elevated serum fluoride levels, typically exceeding 0.1 ppm in cases of chronic exposure, and increased urinary fluoride concentrations, where values above 4 mg/L indicate excessive intake and serve as markers of recent exposure. Alkaline phosphatase (ALP) is frequently elevated, reflecting heightened bone turnover. While serum calcium and phosphate levels usually remain within normal ranges, severe cases may demonstrate dysregulation, indicating advanced metabolic disturbance.

5.4 Case Studies

In India, skeletal fluorosis remains a major public health problem in several fluoride-endemic states. In Andhra Pradesh, more than 10% of rural populations in endemic districts are affected, with severe consequences in areas where water fluoride concentrations exceed 5 mg/L. Advanced cases in these regions often present with debilitating skeletal deformities, including spinal rigidity, knock-knees, and restricted mobility, ultimately leading to partial or complete disability among affected individuals (Reddy et al., 2016). These deformities not only limit physical activity but also interfere with livelihood options, especially in agrarian communities dependent on manual labor.

In Rajasthan and Bihar, chronic fluorosis is widespread, manifesting as a combination of dental and skeletal deformities. The dual burden of mottled teeth and restricted bone function creates both medical and psychosocial challenges for affected populations. For example, villages in Nalanda district of Bihar report prevalence rates exceeding 12%, highlighting the magnitude of the issue in North India (Kumari & Misra, 2023). In these areas, skeletal complications often go undiagnosed until the advanced stages, by which time irreversible deformities have already developed, significantly impairing quality of life.

In Africa, skeletal fluorosis is a widespread concern in regions underlain by volcanic aquifers, particularly in the Rift Valley. In Ethiopia, groundwater fluoride concentrations commonly reach 10–15 mg/L, far exceeding the safe limit of 1.5 mg/L set by the WHO. As a result, more than 15% of adults in these endemic areas suffer from advanced skeletal fluorosis, with symptoms ranging from chronic joint pain to severe spinal deformities that compromise mobility and daily functioning (Rango et al., 2012). The high prevalence highlights the critical link between natural hydrogeology and public health challenges in fluoride-affected regions.

In neighboring Tanzania, similar conditions have been reported, particularly among rural agricultural communities that rely heavily on high-fluoride groundwater sources. Here, skeletal fluorosis manifests as spinal deformities, bowed legs, and rigidity of the joints, which substantially reduce the work capacity of agricultural workers. This not only impacts the physical well-being of individuals but also disrupts household income and agricultural productivity, creating a cycle of health and economic hardship in already vulnerable populations.

The skeletal fluorosis continues to pose a significant health challenge in several endemic provinces, particularly Shanxi. In this region, more than 8% of adults are affected by advanced stages of the disease, where symptoms such as chronic joint pain, stiffness, and limited mobility are frequently reported (Zhao et al., 2019). A major concern is the frequent misdiagnosis of skeletal fluorosis as arthritis, since the clinical presentations joint pain and restricted movement overlap considerably. This misclassification often delays appropriate intervention and exposes patients to prolonged suffering. The situation in Shanxi highlights the importance of region-specific awareness and diagnostic measures, as fluoride contamination is primarily linked to naturally high groundwater fluoride levels. Without early recognition, patients progress to advanced stages marked by skeletal deformities and disability, which significantly impair quality of life. Strengthening diagnostic protocols and promoting safe water alternatives are therefore critical in reducing the burden of skeletal fluorosis in China's endemic areas.

Table 12. Prevalence of skeletal fluorosis in selected regions

Country / State	Water Fluoride (mg/L)	Prevalence (%)	Notes
India (Andhra Pradesh)	3 – 8	10–15	Severe in rural districts
India (Bihar)	2 – 6	8–12	Often co-exists with dental
Ethiopia (Rift Valley)	10 – 15	15–20	Severe deformities
Tanzania	6 – 12	10–18	Reduced productivity
China (Shanxi)	3 – 7	8–10	Misdiagnosed as arthritis

6. Systemic Health Effects Beyond Bones and Teeth

Although skeletal and dental fluorosis are the most visible outcomes of chronic fluoride exposure, an expanding body of research demonstrates that fluoride has systemic effects on multiple organ systems. These effects manifest in endocrine disruption, neurotoxicity, renal impairment, and possible reproductive consequences. The extent of toxicity is influenced by dose, duration of exposure, age, nutritional status, and co-exposure to other environmental contaminants.

6.1 Endocrine Disruption

The thyroid gland is particularly sensitive to fluoride toxicity. Fluoride competes with iodine uptake in the thyroid, impairing the synthesis of thyroid hormones. Chronic exposure to drinking water with fluoride levels above 2.0 mg/L has been associated with suppressed serum thyroxine (T4) and elevated thyroid-stimulating hormone (TSH) levels (Singh et al., 2018). Epidemiological studies from India and China confirm a strong link between fluoride exposure and hypothyroidism prevalence, especially in iodine-deficient regions (Susheela et al., 2019; Zhao et al., 2020).

The relationship between fluoride and thyroid dysfunction is also supported by biochemical data. Fluoride ions interfere with the activity of thyroid peroxidase, an enzyme required for iodination of tyrosine residues, leading to reduced thyroid hormone production (Kheradpisheh et al., 2018). Clinical manifestations include goiter, fatigue, weight gain, and cognitive impairment.

6.2 Neurological Effects

Emerging evidence suggests that fluoride has neurodevelopmental toxicity. A meta-analysis of 27 studies conducted in China revealed that children residing in high-fluoride regions scored significantly lower IQ points than those in low-fluoride areas (Tang et al., 2019). Similar findings were corroborated by studies in India, where schoolchildren exposed to water fluoride concentrations above 3 mg/L exhibited deficits in memory, attention span, and academic performance (Choi et al., 2019). Animal studies further indicate that fluoride crosses the blood–brain barrier, leading to alterations in neurotransmitter levels, oxidative stress, and impaired hippocampal development (Bhatnagar et al., 2020). These findings align with concerns raised by the U.S. National Toxicology Program, which has flagged fluoride as a potential developmental neurotoxin (NTP, 2021).

6.3 Renal Toxicity

The kidneys play a central role in fluoride excretion, removing approximately 50% of absorbed fluoride. In individuals with impaired renal function, fluoride clearance decreases significantly, leading to systemic accumulation (Ayoob et al., 2019). Patients undergoing dialysis in high-fluoride areas are particularly vulnerable, as excess fluoride in dialysate fluid may exacerbate bone and cardiovascular complications (Jha et al., 2020).

Histopathological studies in animals exposed to fluoride-contaminated water show tubular necrosis, glomerular damage, and oxidative stress markers in renal tissues (Mohd et al., 2022). These results suggest that chronic fluoride exposure contributes to progressive nephropathy, particularly when compounded by other nephrotoxic agents.

6.4 Reproductive and Developmental Effects

Fluoride has also been implicated in reproductive toxicity, though findings remain somewhat controversial. Animal experiments have demonstrated reduced sperm motility, decreased testosterone levels, and structural alterations in testicular tissue under chronic fluoride exposure (Shashi et al., 2019). In females, studies show delayed puberty onset, disrupted estrous cycles, and impaired ovarian folliculogenesis (Shen et al., 2021). Human data remain limited, but epidemiological evidence from endemic fluorosis areas indicates possible correlations between high maternal fluoride intake and adverse pregnancy outcomes, including low birth weight and preterm delivery (Green et al., 2019). Although more robust longitudinal studies are required, these associations highlight the need for precautionary approaches in reproductive health.

The systemic effects of fluoride demonstrate that it is not merely a dental or skeletal issue but a multisystem toxicant. The interplay between nutritional factors such as calcium, iodine, and vitamin D status modulates fluoride's systemic impact (Susheela et al., 2019). The addressing fluoride contamination requires a holistic approach, including water quality management, nutritional interventions, and targeted public health measures in endemic regions.

7. Standards and Guidelines

Safe drinking water guidelines are central to managing the health impacts of fluoride exposure. Since fluoride has both beneficial and toxic effects, international and national agencies set concentration limits to balance dental health benefits with the risk of fluorosis. The standards are based on epidemiological studies, climate factors, and patterns of water consumption.

The World Health Organization (WHO) has been central in establishing global guidelines on safe fluoride levels in drinking water. Its recommendation of a maximum permissible concentration of 1.5 mg/L reflects the balance between preventing dental caries and avoiding the risks of fluorosis. While the optimal range is 0.5–1.0 mg/L, WHO also highlights the importance of tailoring standards to local contexts, since water consumption varies widely by climate and lifestyle. For instance, in hot and arid regions, where water intake can double or even triple compared to temperate areas, fluoride exposure may exceed the safe daily threshold even at concentrations considered "normal" elsewhere. This underscores the need for adaptive water quality standards that reflect climatic, geographical, and cultural conditions (WHO, 2017; Fawell et al., 2020; Khairnar et al., 2021).

The Bureau of Indian Standards (BIS) has adopted a desirable limit of 1.0 mg/L and a maximum permissible limit of 1.5 mg/L, applicable when no alternative water source is available (BIS, 2012). These thresholds are particularly critical, as India faces one of the world's largest burdens of endemic fluorosis, with more than 200 districts across 19 states reporting cases (Ayoob et al., 2019). Despite these regulations, field surveys reveal that groundwater fluoride concentrations often exceed 3–10 mg/L in states like Rajasthan, Andhra Pradesh, and Bihar, levels that are far beyond permissible limits and strongly associated with both dental and skeletal fluorosis (Rao et al., 2020; Kumari et al., 2023). The situation is further complicated by India's heavy dependence on groundwater for drinking and agriculture, combined with wide regional variations in climate and water consumption, which increase the risk of overexposure in vulnerable populations.

Globally, regulatory agencies also adjust fluoride standards to reflect regional needs. For example, the U.S. Environmental Protection Agency (EPA) sets a relatively high maximum contaminant level of 4.0 mg/L, though the U.S. Public Health Service (USPHS) recommends a lower optimal level of 0.7 mg/L to reduce dental caries while limiting fluorosis risk (U.S. EPA, 2020). The European Union aligns with WHO, fixing the upper limit at 1.5 mg/L (European Commission, 2018), while China has reduced its national standard to 1.0 mg/L due to widespread endemic fluorosis (Zhao et al., 2020). These regional differences highlight the role of climate and consumption patterns in shaping policy. For instance, daily water intake in Rajasthan can reach 4–5 liters per person compared to just 1.5–2 liters in Europe, meaning that even 1.0 mg/L fluoride may push intake beyond the tolerable limit of 6 mg/day for adults (Susheela et al., 2019). Such evidence emphasizes the need for risk assessment models, such as calculations of Daily Fluoride Intake (DFI), to guide both international and national standards and ensure that public health policies remain context-specific and effective.

$$DFI = C_w \times V_w + C_f \times V_f$$

Where:

- C_w = Fluoride concentration in drinking water (mg/L)
- V_w = Volume of water consumed daily (L/day)
- C_f = Fluoride concentration in food (mg/kg)
- V_f = Amount of food consumed daily (kg/day)

For example, if water contains 2.5" " mg/L fluoride and an adult consumes 3.5" " L/ day, the intake from water alone is:

$$DFI = 2.5 \times 3.5 = 8.75 \mathrm{mg/day}$$

This significantly exceeds the WHO tolerable daily intake of $6 \mathrm{mg/}$ day, indicating high risk of fluorosis.

8. Epidemiological Evidence in India

India is recognized as one of the worst-affected countries in terms of fluoride contamination and fluorosis prevalence. The problem is widespread, covering more than 20 states and union territories, and affects an estimated 66 million people, including over 6 million children suffering from dental, skeletal, or systemic fluorosis (Susheela et al., 2018; Sharma et al., 2021). The source of contamination is largely geogenic, as high fluoride is leached from fluoride-bearing minerals in rocks and aquifers into groundwater, which remains the primary source of drinking water in rural areas.

8.1 National Prevalence

Studies have reported that nearly 62 million people in India are at risk, with around 200 districts identified as endemic for fluorosis. The CGWB and the Ministry of Jal Shakti have listed several fluoride "hotspots," especially in Rajasthan, Andhra Pradesh, Telangana, Gujarat, Bihar, Madhya Pradesh, Uttar Pradesh, and Odisha. Rajasthan alone has more than 16,000 affected villages, while Andhra Pradesh reports fluoride levels as high as 10–20 mg/L in localized areas.

8.2 Bihar and Eastern India

Bihar has emerged as a serious fluoride-affected region in recent years. Groundwater samples from districts such as Nalanda, Gaya, Rohtas, and Munger show fluoride concentrations ranging from 2.0 to 5.5 mg/L, far exceeding the permissible limit of 1.5 mg/L. Epidemiological surveys in these regions show that 50–60% of schoolchildren exhibit dental fluorosis, with visible brown or mottled enamel. Skeletal fluorosis is also being reported in older populations with long-term exposure, manifesting as joint stiffness, spinal deformities, and restricted mobility.

8.3 Rajasthan and Western India

Rajasthan is historically the epicenter of fluorosis in India. Groundwater in districts such as Nagaur, Barmer, Bhilwara, and Dungarpur often contains fluoride above 5 mg/L. In certain deep aquifers, concentrations exceed 15 mg/L, making water unsafe even for short-term consumption. Epidemiological data reveal dental fluorosis prevalence above 80% in children in several villages. Skeletal fluorosis cases are common among adults engaged in farming and manual labor, where prolonged consumption of contaminated water exacerbates bone deformities.

8.4 Southern and Central India

Fluoride contamination is also severe in Andhra Pradesh, Telangana, and Karnataka, where hard rock aquifers dominate. Studies in Nalgonda district (Telangana) have documented groundwater fluoride levels up to 20–

24 mg/L, considered among the highest in the world. The parts of Karnataka such as Tumkur and Kolar districts show fluoride levels ranging between 3.0–10 mg/L. In Madhya Pradesh and Chhattisgarh, endemic fluorosis pockets are concentrated around hard rock terrains, with 30–40% of surveyed villages reporting concentrations above the safe limit.

The health burden of fluoride contamination in India is profound, with millions of people in endemic regions suffering from its consequences. Field-based surveys reveal that dental fluorosis affects between 30–100% of children in districts with high groundwater fluoride levels, with severity depending on the degree and duration of exposure (Gupta et al., 2022). Among adults, long-term exposure has led to skeletal fluorosis prevalence rates ranging from 10–25%, significantly reducing mobility and quality of life (Rao et al., 2021). Women and children are particularly at risk, as nutritional deficiencies especially inadequate calcium intake exacerbate fluoride absorption and toxicity, compounding the health crisis in resource-poor settings (Sharma et al., 2021). These figures highlight not just the clinical impact but also the intergenerational and socioeconomic implications of fluorosis in India.

Policy responses, most notably through the NPPCF, have been crucial in identifying endemic districts, conducting surveillance, and raising awareness. However, major challenges remain in ensuring sustainable access to safe water and effective community participation. While interventions such as community defluoridation plants, rainwater harvesting systems, and piped water supply have been introduced, their implementation often suffers from technical failures, maintenance issues, and limited local engagement (Kumar et al., 2022). Epidemiological findings strongly emphasize the need for region-specific solutions that account for climate, water dependence, and nutritional profiles. Strengthening community-based awareness programs, promoting nutritional supplementation with calcium and antioxidants, and expanding long-term surveillance are essential to protect vulnerable groups and reduce the burden of fluorosis across India.

9. Conclusion

Fluoride contamination in drinking water is a major public health challenge in India, particularly in rural and semi-arid regions where millions of individuals are affected. Although low concentrations of fluoride (0.5–1.0 mg/L) promote dental health, exposure above the permissible limit of 1.5 mg/L set by WHO and BIS results in serious health effects, ranging from dental and skeletal fluorosis to systemic toxicity. Groundwater surveys indicate that states such as Rajasthan, Bihar, Andhra Pradesh, and Telangana are severely impacted, with fluoride concentrations often exceeding 2.0 mg/L and in some districts rising above 20 mg/L. Dental fluorosis is especially common among children, leading to enamel mottling, brown staining, and weakened teeth, while skeletal fluorosis affects adults exposed over long durations, causing chronic joint pain, deformities, and, in advanced cases, neurological complications due to spinal cord compression. Beyond skeletal and dental effects, fluoride toxicity has systemic manifestations, including endocrine disruption, renal impairment, neurological dysfunction, and reproductive health risks. Both geogenic processes, such as weathering of fluoride-rich rocks, and anthropogenic factors, including industrial discharge and over-extraction of groundwater, contribute to this crisis. Given fluoride's narrow margin between beneficial and harmful levels, robust monitoring and timely interventions are critical. Addressing this challenge requires a comprehensive approach. Defluoridation methods such as the Nalgonda technique, activated alumina, reverse osmosis, and bone char have shown promise, while rainwater harvesting offers a safe and sustainable alternative. Nutritional strategies, particularly supplementation with calcium, vitamin C, vitamin D, and antioxidants, help mitigate fluoride absorption and toxicity. At the policy level, initiatives like the NPPCF, combined with school-based screening, community awareness, and infrastructure development, are essential. The reducing fluorosis prevalence demands region-specific strategies, coordinated government efforts, and active community participation to ensure safe drinking water, better systemic health, and improved quality of life across India's fluoride-endemic regions.

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