Scienta Press Journals

American Surgeon
Journal of Surgical Science and Medical Advances

Comprehensive Scientific Analysis Of Dental And Skeletal Fluorosis: Pathogenesis, Clinical Manifestations, Ethiological Factors, And Modern Preventive Strategies

Author: Otabekova Gulchehra Otabek qizi
Tashkent International University of Kimyo
3rd-year student, faculty of Dentistry
Muxamedov Jamoliddin Isroil o'g'li
Tashkent International University of Kimyo
3rd-year student, faculty of Dentistry
Otajonova Omina Rustam qizi
Tashkent International University of Kimyo
3rd-year student, faculty of Dentistry
Zokirov Azimjon Umarali o'g'li
Tashkent International University of Kimyo
3rd-year student, faculty of Dentistry

Abstract

Fluorosis is a severe dental and systemic disease that can occur as a result of excessive consumption and ingestion of fluoride in the daily diet. It is well established that fluoride actively affects the mineralized tissues of the body, and when consumed in quantities significantly above the optimal thresholds, it severely disrupts the fundamental processes of tooth and bone mineralization.

Keywords: *Fluorosis, Fluoride Toxicity, Enamel Hypomineralization, Amelogenesis, Skeletal Deformity, Dean's Index, Defluoridation, Public Health.*

1. RELEVANCE OF THE STUDY

Fluorosis is a severe dental and systemic disease that can occur as a result of excessive consumption and ingestion of fluoride in the daily diet. It is well established that fluoride actively affects the mineralized tissues of the body, and when consumed in quantities significantly above the optimal thresholds, it severely disrupts the fundamental processes of tooth and bone mineralization. High fluoride intake can directly lead to either dental or skeletal fluorosis, presenting a dual threat to public health. Other critical causes of excessive fluoride exposure include the intensive use of toothpaste with high fluoride content, the habitual consumption of certain types of milk, and an excessively high consumption of meat products.

On the other hand, dental fluorosis is considerably more widespread in endemic zones and can result from a fluoride intake that is only slightly above the recommended optimal levels. Morphologically, dental fluorosis is characterized as a specific developmental defect of the tooth enamel, which emerges directly during the critical periods of tooth development due to the excessive absorption and accumulation of fluoride ions. Dental fluorosis can deeply affect both the outer aesthetic appearance and the microscopic internal structure of the teeth, causing structural vulnerabilities that persist throughout life.

In the era of modern industrialization, the epidemiological relevance of fluorosis has grown due to localized environmental imbalances. Anthropogenic activities, such as intensive coal combustion, phosphate fertilizer manufacturing, and industrial waste mismanagement, release vast amounts of gaseous and particulate fluorides into the biosphere. These compounds contaminate localized agricultural zones and water tables, expanding the risk boundaries beyond traditionally recognized natural geochemical anomalies. Therefore, understanding the comprehensive epidemiological, physiological, and clinical landscape of fluorosis is vital for designing targeted public health interventions.

2. Objective And Scope

The primary objective of this research is to analyze the structural, biochemically destructive, and global public health implications of fluorosis. In fluorosis, an enamel layer characterized by decreased mineral content and significantly increased porosity is formed, undermining the mechanical resilience of the dentition. Global epidemiological research conducted by the World Health Organization (WHO) has revealed that dental fluorosis affects approximately 70 million people worldwide, with an alarming estimate of about 60 million of these individuals residing within regions of India and China [1].

The pathological process driving dental fluorosis involves the direct replacement and disruption of the biological enamel matrix by inorganic material during amelogenesis, which subsequently results in profound hypomineralization within the mature enamel structure [3]. Clinically, dental fluorosis can cause noticeable and cosmetically distressing tooth enamel discoloration; however, in severe cases, this discoloration is accompanied by physical disintegration, extensive pitting, and loss of structural continuity. This research seeks to correlate various fluoride concentration thresholds with the severity of tissue destruction, evaluating the multi-organ impact of systemic chronic fluoride toxicity.

Furthermore, according to scientific research, the largest source of fluoride-contaminated air is the massive burning of coal; these volatile fluorides are easily absorbed by open food products left in proximity and are directly inhaled through the human respiratory tract [5]. Consequently, fluoride-contaminated food remains a primary and highly significant cause of substantial human exposure to fluoride [5]. This study aims to compile these distinct pathways—waterborne, airborne, and dietary—into a unified epidemiological framework to assist clinical practitioners and public health authorities.

3. Research Materials And Methods

Fluorosis is fundamentally a disease that acquires an endemic character, meaning it is consistently observed among populations residing in specific geographic regions where the fluoride content in the local drinking water significantly exceeds the physiological norm. This study employs an analytical and comparative review of clinical data, toxicological reports, and environmental parameters compiled from various endemic zones.

Subsequent extensive medical examinations, historical follow-ups, and scientific observations have conclusively demonstrated that fluorosis does not merely damage the teeth; rather, the entire skeletal system and bone structure can be profoundly impaired. Crucially, this systemic vulnerability is not exclusive to humans, as domestic and wild animals living in high-fluoride zones exhibit identical skeletal degeneration. In nature, fluorine routinely binds with metallic elements, occurring naturally in the form of various fluorides; once ingested, it replaces elements within all organs of the human body, establishing its primary and most dense concentrations within bone tissues and the crystalline lattice of teeth.

Chemically, the fluorine element stands at the head of the seventh group (halogens) in the Mendeleev Periodic Table, possessing exceptionally high chemical activity and electronegativity. Due to this extreme activity and reactivity, fluoride ions effortlessly pass through the body's natural physiological protective barriers and defensive borders. Once inside, they systematically disrupt a wide variety of metabolic processes within soft and hard tissues, causing extensive damage to multiple organ systems. An above-normal concentration of the fluorine element directly breaks the vital bonds between the organic protein scaffold (matrix) of enamel/bone and the integrating mineral salts. This molecular disruption causes the systematic breakdown, resorption, and gradual erosion of these vital hard tissues.

Fluoride ions fundamentally inhibit the activity of the essential phosphatase enzyme system (particularly alkaline phosphatase), thereby completely deranging the delicate mineralization processes taking place within the tissues, including the developing dental enamel matrix. Interestingly, clinical research has established that the distinct clinical signs and symptoms of fluorosis are not uniformly manifested among all children who reside within an endemic source and consume the exact same water supply. Among these cohorts, perfectly healthy children with undamaged, normal teeth are frequently observed. This clear variation indicates that the organisms of children utilizing the same concentration of fluoridated water possess varying degrees of sensitivity, tolerance, and susceptibility to the fluoride substance. This biological variation is highly dependent on the baseline stability and resistance of the child's organism, their nutritional status, and the history of past childhood diseases they have overcome.

4. Results And Their Analysis

According to established GOST regulatory standards, the mandatory normative concentration of fluoride in public drinking water must strictly fall within the range of 0.7 to 1.0 mg/liter. Maintaining this precise fluoride level in water plays a crucial and proven role in the population-wide prevention of dental caries (tooth decay).

However, the overall quantity of fluoride entering the organism via water must never exceed the total daily physiological requirement of the human body. The standard daily requirement of the human body for fluoride is precisely 3.0 to 3.5 mg. When the fluoride concentration in drinking water rises to 1.2–1.5 mg/l and above, it begins to trigger various degrees of dental fluorosis within the tooth enamel. At even higher concentrations (8–10 mg/l), fluoride causes severe non-dental systemic anomalies; in addition to devastating changes in the tooth enamel, it induces persistent joint pain, lower back agony, pathological muscle rigidity, profound difficulty in standing or walking upright, numbness and weakness in the limbs, and sudden episodes of fainting or syncope. Extremely elevated concentrations of fluoride in water (10–15 mg/l) induce extensive calcification (ossification) of soft tissues, particularly targeting critical blood vessels, ligaments, and tendons; this severe systemic maternal toxicity ultimately leads to catastrophic reproductive outcomes, including high rates of stillbirths and elevated infant mortality.

Table 1. Pathological Impact of Diverse Fluoride Concentrations in Water on Human Health

Fluoride Concentration	Clinical & Pathological Impact	Epidemiological Categorization
0.7 – 1.0 mg/l	Optimal level for dental health. Provides highly effective prevention against dental caries (tooth decay) without causing tissue damage.	Normative / Standard (GOST)
1.2 – 1.5 mg/l	Initiation of dental fluorosis. Causes mild to moderate enamel changes, loss of	Mild to Moderate Endemic Threshold

ISSN: 3071-1819 Volume 01, Issue 05, June, 2026

	luster, and appearance of white spots or opaque streaks.	
3.0 – 3.5 mg/l	Exceeds the total safe daily physiological requirement (3.0-3.5 mg). Accelerates enamel hypomineralization and severe dental coloration.	Borderline Toxic Accumulation Level
8.0 – 10.0 mg/l	Severe enamel destruction, deep brown staining. Systemic toxicity: severe joint pain, spinal stiffness, muscle rigidity, limb numbness, and syncope.	Severe Skeletal & Systemic Fluorosis
10.0 – 15.0 mg/l	Extensive calcification of soft tissues, blood vessels, ligaments, and tendons. Causes severe reproductive disasters, including high stillbirth and infant mortality rates.	Lethal / Critical Systemic Toxicity

4.1. Clinical Classification of Dental Fluorosis (Dean's Index)

To standardize the diagnosis and epidemiological assessment of dental fluorosis, clinicians utilize Dean's Fluorosis Index. This classification categorizes the severity of enamel changes into six distinct stages:

ISSN: 3071-1819 Volume 01, Issue 05, June, 2026

- **Questionable (Score 0.5):** The enamel represents a smooth, glossy, and semi-translucent pale creamy-white crystalline surface. No pathological alterations or hypomineralization are visible.
- **Normal (Score 0):** The enamel shows slight aberrations from the translucency of normal enamel, ranging from a few white flecks to occasional light white spots. This is used when a definitive diagnosis of mild fluorosis is warranted but not absolutely certain.
- **Very Mild (Score 1):** Small, opaque, paper-white areas scattered irregularly over the tooth surface, involving less than 25% of the labial or buccal tooth surface.
- **Mild (Score 2):** The white opaque lines or patches are more extensive, but completely cover less than 50% of the total tooth surface area.
- **Moderate (Score 3):** Practically all enamel surfaces of the teeth are affected; distinct opaque white chalky surfaces are evident, and brown staining frequently begins to manifest as an aesthetic defect.
- **Severe (Score 4):** All enamel surfaces are entirely affected, characterized by marked hypomineralization, severe brown discoloration, and widespread physical pitting. The pits are confluent, giving the tooth a corroded appearance, and the brittle enamel is highly prone to fracture.

4.2. Advanced Prevention and Therapeutic Interventions

Controlling endemic fluorosis requires a dual approach balancing community-wide engineering and individualized medical care. Public health interventions focus primarily on water defluoridation techniques. The Nalgonda technique, which involves sequential precipitation using aluminum salts and lime, remains a cost-effective community strategy. In advanced municipal frameworks, reverse osmosis (RO) filtration systems are deployed to reliably strip excess fluoride ions from potable water sources.

On an individual level, clinical management depends on the severity of the condition. For mild to moderate fluorosis, aesthetic defects are successfully managed via microabrasion (using hydrochloric acid and silicon carbide paste) combined with intensive remineralization therapy using calcium phosphate or casein phosphopeptide-amorphous calcium phosphate (CPP-ACP) formulations. For severe cases with surface pitting and structural loss, restorative dentistry—including composite bonding, porcelain veneers, or full-coverage crowns—is required to restore function and aesthetics.

5. CONCLUSION AND RECOMMENDATIONS

In summary, the intake of excessive fluoride into the organisms of children aged 7 to 15 years results in pronounced growth retardation, while in adolescents aged 13 to 15 years, a severe arrest of sexual development and maturation is frequently observed. Long-term scientific observations have confirmed that a concurrently low concentration of calcium in drinking water (below 30 mg/l) or its inadequate representation in food products (300–400 mg/kg) drastically exacerbates the clinical course of fluorosis, accelerating its progression into severely destructive forms.

The clinical manifestation and morphological forms of fluorosis are highly dependent on the absolute concentration of fluoride in drinking water, the duration of the patient's residence in that specific endemic zone (particularly during the critical periods of tooth mineralization), the unique biological sensitivity of the individual organism to fluoride intoxication, their general systemic health, and their baseline physiological resistance to poisoning processes.

If a child has lived in a region with high fluoride concentration for only a limited, brief period, enamel fluorosis will manifest exclusively on the specific teeth that were actively developing and mineralizing during that timeframe. Conversely, if a child resided in that endemic area during the fetal gestation period and continued to live there for several consecutive years after birth, the fluorosis pathology will be clinically visible across all teeth without exception.

Furthermore, even when the drinking water contains a borderline safe fluoride level of approximately 1 mg/l, the high volume of water consumption typical in hot arid climates can easily trigger the development of mild forms (such as speckled, spotted, or striated patterns) of dental fluorosis.

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