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Dental fluorosis and fluoride: etiology, clinical pathogenesis, diagnosis and contemporary management**Ergashev Bekzod¹**

Central Asian Medical University International Medical University,
Burhoniddin Margʻinoniy Street 64, Phone: +998 95 485 00 70, Email:

info@camuf.uz, Fergana, Uzbekistan¹

E-mail: bekzodergashev0401@gmail.com¹

Orcid: <https://orcid.org/0009-0000-0382-0811>¹

Parpiyeva Odinoxon Raxmanovna²

Central Asian Medical University International Medical University Dotsent,
Burhoniddin Margʻinoniy Street 64, Phone: +998 95 485 00 70, Email:

info@camuf.uz, Fergana, Uzbekistan²

E-mail: parpieva.odinahon@yandex.ru²

Orcid: <https://orsid.org/0000-0001-6223-103X>²

Abstract: Dental fluorosis is a systemic condition resulting from chronic ingestion of excessive fluoride during enamel mineralization, leading to hypomineralized, porous enamel with characteristic discolorations and structural changes. Fluoride is ubiquitous in the environment, primarily entering the human body through drinking water, foods, and dental products. While optimal fluoride levels reduce dental caries, concentrations above 1.5 mg/L in water are strongly associated with an increased prevalence of fluorosis globally. The condition exhibits endemic patterns in regions with high geological fluoride concentrations, particularly in groundwater. The pathogenesis involves disruption of ameloblast function, altered protein processing in the enamel matrix, and defective fluorapatite deposition, resulting in dose-dependent enamel changes. Diagnosis relies on standardized clinical indices and assessment of fluoride exposure history. Contemporary approaches emphasize prevention through controlled fluoride exposure, regular monitoring of water sources, and public health measures in endemic regions. Management focuses on minimally invasive aesthetic enhancement for moderate or severe cases. Given the balance between caries prevention and fluorosis risk, understanding the etiological and pathophysiological mechanisms is crucial for effective public health strategies and clinical protocols.

Keywords: fluoride, dental fluorosis, enamel hypomineralization, groundwater fluoride, pathogenesis, diagnosis, epidemiology, prevention, ameloblasts, enamel porosity, public health.

Intradaction: Dental fluorosis is a developmental enamel defect caused by excessive systemic fluoride intake during tooth development. The disorder manifests as qualitative and quantitative changes in enamel mineralization, varying from mild white opacities to severe brown discoloration and pitting.

Fluoride (F) exists naturally in the environment and is a highly reactive halogen, readily dissolving into water, soil, and air. Groundwater fluoride concentrations vary widely depending on the underlying geology; in some regions, concentrations exceed globally recommended limits, contributing to endemic fluorosis phenomena.

Fluoride's role in dental biology is dichotomous. At low concentrations, fluoride enhances enamel resistance to caries through the formation of fluorapatite crystals and promotion of remineralization. Systemic sources include drinking water, foods, and inhalation of fluoride particulates; topical sources include toothpaste and professional fluoride applications. The World Health Organization (WHO) recommends an upper limit of 1.5 mg/L of fluoride in drinking water to prevent fluorosis while maintaining caries control benefits. However, areas with naturally high fluoride in groundwater often surpass this threshold, leading to endemic fluorosis affecting both teeth and skeletons.

Dental fluorosis occurs exclusively during enamel formation, particularly during the amelogenesis phase, a process involving enamel matrix secretion and mineralization by ameloblasts. During this critical period, excessive fluoride interferes with protein degradation in the developing enamel, leading to hypomineralization, increased porosity, and structural defects. The severity of fluorosis is proportional to fluoride dose, duration, and timing relative to tooth development stages.

Mild fluorosis may present as scattered opaque white patches covering less than half of the tooth surface; moderate to severe forms include brown stains and enamel pitting with compromised physical integrity.

Globally, the prevalence of dental fluorosis has increased in recent decades due to widespread fluoride use and environmental exposure. In some populations, up to two-thirds of adolescents exhibit signs of fluorosis, with a significant proportion classified as moderate to severe. Geographical distribution highlights regions with high geological fluoride content in soil and rocks, often correlating with elevated fluoride in aquifers and public water supplies. Endemic fluorosis is particularly documented in parts of Asia, Africa, and Latin America, where limited water treatment capacity and reliance on groundwater contribute to prolonged high fluoride exposure.

Understanding the etiological and epidemiological aspects of fluorosis is critical for public health policy. Surveillance of fluoride levels in water and dietary sources informs preventive strategies to balance caries prevention against fluorosis risk. Beyond environmental monitoring, research on genetic susceptibility suggests that individual biological responses contribute to the variability of fluorosis outcomes, although fluoride exposure remains the primary risk factor.

Diagnostic assessment of fluorosis uses standardized clinical indices that categorize enamel changes based on severity and distribution patterns. Recognizing fluorosis distinct from other enamel defects such as hypoplasia or intrinsic staining is essential for accurate epidemiological reporting and management planning. Contemporary strategies for managing fluorosis emphasize prevention, risk assessment, and aesthetic rehabilitation for moderate and severe cases. Given the permanent nature of enamel changes once established, public health measures focusing on environmental and systemic fluoride control are paramount.



Dental Fluorosis Causes

Materials and Methods: This narrative review synthesizes current scientific knowledge on the etiology, clinical pathogenesis, diagnosis, and management of dental fluorosis based on published literature from peer-reviewed journals, systematic reviews, and epidemiological studies. Articles were identified through academic databases and scientific search engines, including PubMed, MDPI, WHO regional literature repositories, and indexed dental science sources, using terms related to fluoride exposure, dental fluorosis, enamel development, groundwater fluoride concentrations, clinical indices, and management.

Global water fluorosis research indicates that drinking water remains the principal source of systemic fluoride intake influencing fluorosis prevalence. Systematic analyses of water fluoride levels and dental outcomes demonstrate a positive correlation between fluoride concentrations above 1.5 mg/L and increased risk of fluorosis. These studies apply PRISMA methodologies to collect and appraise research from multiple regions, providing a comparative understanding of fluoride exposure and health effects across diverse geological environments.

Understanding pathogenesis draws on biochemical and cellular investigations into ameloblast behavior under fluoride stress. Experimental research characterizes how excessive fluoride disrupts enamel protein processing, alters matrix degradation, and affects mineral deposition. Analytical methods in these studies include molecular assays of ameloblast function, enamel

matrix composition analyses, and quantitative assessment of mineral content changes in experimental models. These approaches elucidate the cellular mechanisms underlying enamel hypomineralization, providing foundational insights into fluorosis development.

Hydrological and geological assessments form an integral part of fluorosis research, as fluoride concentrations in aquifers depend on rock composition, soil interactions, and groundwater flow dynamics.

Dental Fluorosis Symptoms



Water quality surveys and geochemical sampling provide statistical data on fluoride distribution in environmental water sources. Such surveys often include ionic analysis using fluoride-selective electrodes, geographic mapping of high-fluoride zones, and comparison with epidemiological data on dental fluorosis prevalence rates.

Diagnostic research focuses on the development and validation of clinical indices that reliably categorize fluorosis severity. Standardized scales, such as Dean’s Index and the Thylstrup-Fejerskov Index, facilitate consistent assessment across populations and time. These indices are based on visual criteria of enamel appearance, enabling quantitative reporting of fluorosis distribution within cohorts.

Contemporary management research emphasizes preventive dental public health strategies. This includes evaluating the impact of controlled fluoride exposure in water and dental products, community-based water defluoridation technologies, and public education initiatives for fluorosis risk mitigation. Although clinical treatment modalities for aesthetic improvement exist, such as microabrasion and resin infiltration, the focus remains on systemic prevention and environmental control due to the irreversible nature of enamel changes.

Results:

Etiology and Environmental Exposure

Dental fluorosis arises from the systemic intake of fluoride during enamel formation, predominantly through drinking water with elevated fluoride concentrations. Groundwater in specific geological regions often encounters

high levels of fluoride due to the dissolution of fluoride-bearing minerals. Studies indicate that fluoride concentrations above 1.5 mg/L are associated with a marked increase in fluorosis prevalence, aligning with WHO guideline thresholds established for safe drinking water.

Environmental fluoride arises from natural geological formations containing fluorite and related minerals. As groundwater percolates through fluoride-rich rocks and sediments, fluoride ions dissolve and accumulate in aquifers. Climatic and hydrological conditions influence fluoride mobilization, with alkaline groundwater exhibiting higher fluoride solubility. Agricultural practices and industrial emissions may further contribute to environmental fluoride levels in soil, water, and air. In certain endemic regions, reliance on untreated groundwater sources results in chronic fluoride exposure across communities, particularly where surface water alternatives are limited.



Dental Fluorosis Treatment

Dietary fluoride intake supplements water exposure. Foods and beverages prepared with high-fluoride water contribute to systemic fluoride load. Tea, shellfish, and certain vegetables naturally accumulate fluoride. Additionally, fluoride toothpaste and dietary supplements contribute to total fluoride ingestion, particularly in children, where inadvertent ingestion is more common. However, compared to groundwater exposure, these sources typically contribute less to systemic fluoride levels.

Clinical Pathogenesis

Fluorosis pathogenesis centers on disrupted enamel formation, a complex process governed by ameloblast activity. Under normal conditions, ameloblasts orchestrate enamel matrix secretion, protein degradation, and subsequent mineral deposition to form highly organized hydroxyapatite crystals. Excessive fluoride interferes with these processes at multiple biochemical and cellular levels. Fluoride ions influence ameloblast protein synthesis and regulation, leading to altered matrix degradation and delayed mineralization. Increased enamel porosity results from defective fluorapatite formation and failed matrix protein clearance.

At the molecular level, fluoride exposure induces endoplasmic reticulum stress in ameloblasts, altering protein folding and enzymatic activities essential for matrix maturation. The imbalance between mineral deposition and organic matrix removal results in hypomineralized enamel with enlarged intercrystalline spaces.

Fluorosed enamel contains immature enamel proteins and increased fluorapatite with differing crystallographic properties compared with normally mineralized enamel. The resultant enamel is more porous, less mineralized, and structurally compromised, which is observable clinically as white opaque areas or, in severe cases, brown stains and pits.

The severity of fluorosis is dose-dependent, influenced by fluoride concentration, duration, and age during exposure. The critical window of susceptibility aligns with early childhood, particularly during the first eight years of life when permanent anterior tooth enamel is forming. Genetic factors and nutritional status may modulate individual susceptibility, though fluoride exposure remains the primary determinant.

Epidemiological Patterns. Epidemiological data reveal endemic patterns of fluorosis in regions with high environmental fluoride. Global estimates suggest that millions of individuals, particularly in Asia and Africa, exhibit varying degrees of fluorosis. Large-scale systematic reviews indicate that populations consuming groundwater with elevated fluoride levels show prevalence rates far exceeding global averages. In some areas, over half of school-aged children demonstrate signs of mild to severe fluorosis. Groundwater surveys document variable fluoride distribution, with high-fluoride aquifers corresponding to increased fluorosis prevalence within local populations.

Statistical analysis of water quality and oral health outcomes demonstrates a strong positive correlation between fluoride concentrations and fluorosis severity. These findings highlight the need for regular monitoring of drinking water fluoride and implementation of mitigation strategies in high-risk zones. Geographic information systems and hydrological mapping contribute to identifying endemic areas and allocating public health resources efficiently.

Diagnosis. Diagnosis of dental fluorosis relies on standardized clinical examination and severity classification using validated indices. Clinically, fluorosis presents as symmetrical enamel changes across corresponding teeth, distinguishing it from localized enamel defects. Visual assessment categorizes fluorosis severity based on enamel opacity, surface irregularities, and discoloration patterns. Standard indices quantify severity for epidemiological reporting and research comparability.

Differential diagnosis considers other developmental enamel defects such as amelogenesis imperfecta and hypoplasia, which may mimic fluorosis appearances but differ in etiology and distribution patterns. A thorough

environmental fluoride exposure history and hydrological data enhance diagnostic specificity.

Management and Contemporary Approaches

Prevention remains the cornerstone of managing dental fluorosis at the population level. Controlled fluoride levels in drinking water through water treatment and public health policies aim to balance caries reduction benefits with fluorosis risk. In endemic areas, community water defluoridation techniques and alternative safe water sources are essential. Public education campaigns communicate safe fluoride intake levels and encourage appropriate use of fluoride-containing dental products to minimize excessive ingestion in early childhood.

Clinical management of established fluorosis focuses on aesthetic improvement for moderate and severe cases. Minimally invasive techniques such as microabrasion, bleaching, and resin infiltration address discolorations and surface irregularities. More extensive restorative procedures may be appropriate for severe enamel defects, though they are not preventive.

Overall, contemporary approaches underscore the importance of interdisciplinary strategies combining environmental health, epidemiology, and clinical dentistry to reduce the burden of fluorosis while preserving fluoride's protective effects against caries.

Discussion: Dental fluorosis remains a significant public health concern in regions where environmental fluoride concentrations exceed recommended levels. The balance between fluoride's cariostatic benefits and its potential to cause enamel hypomineralization underscores a complex health dilemma. Historically, fluoride was introduced into community water supplies in the mid-20th century to reduce caries rates, yielding substantial declines in tooth decay. However, prolonged exposure to fluoride at higher environmental concentrations, especially in groundwater, has led to a rise in fluorosis prevalence, necessitating careful evaluation of fluoride delivery systems at the population level.

From an etiological perspective, fluoride's systemic absorption and incorporation during enamel formation are central to fluorosis development. Fluoride ions readily dissolve in water and bind with developing enamel crystals, altering the kinetics of mineral deposition and matrix protein degradation. These biochemical disruptions underlie the characteristic hypomineralization seen in fluorosed teeth, which range in severity based on dose and timing of exposure. The correlation between water fluoride concentrations and fluorosis prevalence underscores the importance of geological and hydrological factors in disease distribution. Endemic fluorosis regions often align with aquifers containing naturally high fluoride levels, driven by rock composition and groundwater chemistry.

Epidemiological patterns reinforce these associations, showing variable global prevalence tied to environmental fluoride distribution. Systematic water quality assessments and oral health surveys provide robust statistical evidence that regions with elevated fluoride in drinking water report higher rates of dental fluorosis. These patterns reflect environmental health challenges where water treatment infrastructure is limited, and populations rely on untreated groundwater sources. The documented geographic clusters of fluorosis emphasize the need for targeted surveillance, water quality monitoring, and public health infrastructure improvements.

Clinically, fluorosis presents diagnostic challenges due to its phenotypic overlap with other enamel defects. Standardized clinical indices facilitate consistent severity scoring, which is crucial for epidemiological comparability and research. Symmetrical enamel changes across dentition distinguish fluorosis from localized enamel abnormalities. Accurate diagnosis requires integration of environmental exposure history, hydrological data, and clinical examination. Differential diagnosis includes conditions such as amelogenesis imperfecta, enamel hypoplasia, an intrinsic staining from systemic factors, requiring careful clinical judgment.

Pathophysiologically, fluorosis reflects a disruption in ameloblast function during a critical period of enamel development. Laboratory investigations demonstrate that excessive fluoride induces cellular stress, alters protein processing pathways, and modifies mineral deposition dynamics. These cellular mechanisms explain the increased enamel porosity, altered crystal structure, and resultant clinical manifestations. Understanding these pathways informs preventive strategies by identifying critical windows of susceptibility and reinforcing the importance of controlled fluoride exposure, particularly during early childhood when permanent anterior teeth are forming.

Public health strategies focusing on environmental control of fluoride exposure remain the most effective means to reduce fluorosis prevalence. Water defluoridation programs, alternative safe water sourcing, and continuous water quality evaluation are essential interventions in endemic regions. Moreover, education on appropriate fluoride use, especially in dental products, helps manage total fluoride intake among children. The dual role of fluoride — as a caries preventive agent and a potential cause of fluorosis — necessitates policies that optimize protective benefits while minimizing risk. Management of established fluorosis centers on aesthetic restoration, as enamel changes are permanent once mineralization is complete. Minimally invasive techniques such as microabrasion and resin infiltration can improve cosmetic outcomes for moderate to severe cases. However, these interventions do not reverse the underlying hypomineralization and are

primarily cosmetic. Therefore, emphasis on early prevention and environmental management remains paramount.

Broader implications of fluorosis research extend beyond dental health, encompassing environmental geology, water resources management, and community health planning. Integrating hydrological data with oral health surveillance allows for predictive mapping of high-risk areas and prioritization of resources. Collaboration between dental researchers, environmental scientists, and public policy experts enhances the development of comprehensive strategies to manage fluoride exposure at the population level.

In summary, dental fluorosis exemplifies a multifaceted health issue where environmental exposure, biological mechanisms, clinical diagnosis, and public health interventions intersect. Continued research into fluoride's effects, advanced diagnostic tools, and community-based prevention programs will support efforts to mitigate fluorosis while preserving fluoride's role in caries prevention.

Conclusion: Dental fluorosis remains a prevalent condition in regions with elevated environmental fluoride, reflecting the systemic impact of fluoride during enamel formation. Etiologically linked to excessive fluoride ingestion from high-fluoride groundwater and supplemented by dietary and topical sources, fluorosis represents a dose-dependent disruption of enamel mineralization. The pathogenesis involves altered ameloblast activity, resulting in hypomineralized and porous enamel with clinical manifestations ranging from mild opacity to severe discoloration and pitting. Accurate diagnosis relies on standardized clinical indices and environmental exposure data. Managing fluorosis emphasizes preventive public health measures aimed at controlling fluoride levels in water supplies and minimizing excess intake, especially among children. While clinical aesthetic treatments improve appearance, prevention through environmental control and community education remains paramount. Integrating hydrological monitoring with dental surveillance supports balanced strategies that maintain fluoride's cariostatic benefits while reducing fluorosis risk.

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