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BAFFLED AT SEA- AN ORIGINAL REVIEW ARTICLE

Public Health

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ABSTRACT

Fluoride has played a transformative role in modern dentistry by significantly reducing dental caries worldwide. Its incorporation into community water supplies, toothpastes, varnishes, and restorative materials has improved oral health outcomes across diverse populations. However, excessive fluoride exposure is associated with adverse effects such as dental fluorosis, skeletal fluorosis, and potential systemic concerns. This paper explores fluoride as a "double-edged sword" in dentistry, examining its mechanisms of action, benefits in caries prevention, associated risks, ethical considerations, and clinical implications. A critical analysis highlights the balance required between therapeutic efficacy and toxicity. Understanding dosage, exposure sources, and patient-specific risk factors is essential for optimizing benefits while minimizing harm.

KEYWORDS

Fluoride; Dental Caries; Fluorosis; Remineralization; Public Health Dentistry; Toxicity; Preventive Dentistry; Risk-Benefit Analysis.

INTRODUCTION

Fluoride is widely recognized as one of the most effective preventive agents in dentistry. Since the introduction of community water fluoridation in the 20th century, there has been a substantial decline in dental caries prevalence globally. Landmark studies in cities such as Grand Rapids demonstrated the caries-preventive benefits of controlled water fluoridation, influencing global public health policy. Despite its preventive advantages, fluoride's therapeutic window is narrow. Chronic excessive exposure during enamel development may lead to dental fluorosis, while long-term high intake can contribute to skeletal fluorosis. The dual nature of fluoride — protective at optimal levels yet harmful at excessive doses — necessitates careful evaluation of its application in modern dental practice.

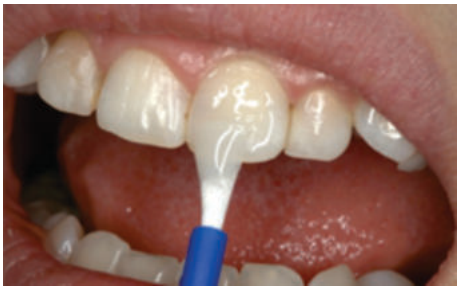

Fig 1 Shows the Application of Fluoride with Applicator Tip

Fig 2 Shows the Armentarium for Fluoride Application

Fig 3 Shows the Application of Fluoride with Disposal Trays

Review of Literature

Y Li (2001) et al conducted a study was to determine the prevalence of bone fracture, including hip fracture, in six Chinese populations with water fluoride concentrations ranging from 0.25 to 7.97 parts per million (ppm). A total of 8266 male and female subjects > or =50 years of age were enrolled. Parameters evaluated included fluoride exposure, prevalence of bone fractures, demographics, medical history, physical activity, cigarette smoking, and alcohol consumption. The results confirmed that drinking water was the only major source of fluoride exposure in the study populations. A U-shaped pattern was detected for the relationship between the prevalence of bone fracture and water fluoride level. The prevalence of overall bone fracture was lowest in the population of 1.00-1.06 ppm fluoride in drinking water, which was significantly lower ($p < 0.05$) than that of the groups exposed to water fluoride levels > or =4.32 and < or =0.34 ppm. The prevalence of hip fractures was highest in the group with the highest water fluoride (4.32-7.97 ppm). The value is significantly higher than the population with 1.00-1.06 ppm water fluoride, which had the lowest prevalence rate. It is concluded that long-term fluoride exposure from drinking water containing > or =4.32 ppm increases the risk of overall fractures as well as hip fractures. Water fluoride levels at 1.00-1.06 ppm decrease the risk of overall fractures relative to negligible fluoride in water; however, there does not appear to be similar protective benefits for the risk of hip fractures.

Riccardo Mazzoli (2025) et al conducted a systematic review and dose-response meta-analysis by searching literature in online databases (MEDLINE/PubMed, Embase and Web of Science) up to August 26, 2025, and by pooling results of studies examining fluoride exposure and its associations with bone health outcomes, namely fracture risk and BMD. We also conducted stratified analyses by sex, age, and fracture site. This study is registered in PROSPERO (CRD42022321899). They included 37 studies published between 1945 and 2024. 10 pertained to bone density only; 19 to bone fractures only; and eight to both outcomes. We found a non-linear, positive relation between fluoride exposure and fracture risk, with an indication of an approximate threshold around 1.5 mg/L of fluoride in drinking water and a nearly linear increasing fracture risk above that concentration (risk ratios of 1.06, 1.19 and 1.35 at 2.0, 3.0 and 4.0 mg/L, respectively, compared to null exposure). Sex-specific dose-response analyses, available only for fragility fractures, indicated an effect in females but little or nothing in males. The association between fluoride and BMD was inconsistent, showing opposite trends for different bone sites (hip and spine) and amounts of exposure, and by sex. Among females aged over 50 years, an association of drinking water fluoride with fragility fracture risk started as early as around 0.5 mg/L (risk ratio of 1.26 at 1.0 mg/L). This study indicates increased risk of fractures with fluoride exposure > 1.5 mg/L in drinking water, with an enhanced susceptibility in postmenopausal females. These findings, which appear consistent with those yielded by recent pooled analysis related to other endpoints in children, may be helpful in assessing the risk-benefit profile for fluoride exposure.

Eida Medjedovic (2015) et al conducted a included school children aged 8 to 15 years who visited health and dental services dependent in Mostar. It is obvious that after the implementation of treatment with 5% NaF by the method of topical fluoridation, health status of subjects from the experimental group significantly improved, so that at the final review 89.71% or 61 subjects of the experimental group had healthy (cured teeth), tooth with dental caries only 5.88% or 4 respondents tooth with dental caries and filling 4.41% or 3 respondents, extracted baby tooth 14.71% or 10 respondents, while for 13.24% of respondents was identified state with still unerupted teeth. Our findings are indirectly confirmed that the six-month treatment of fluoridation with 5% NaF, contributed to statistically significant improvement in overall oral health of the experimental group compared to the control group which was not treated by any dental treatment. It can be concluded that there is a statistically significant difference in the evaluated parameters of oral health of children in the control group compared to the studied parameters of oral health the experimental group of children at the final dental examination.

Xin – Hai Yin (2015) et al conducted a study where PubMed and EMBASE databases were searched to identify relevant observational studies from the time of inception until March 2014 without restrictions. Data from the included studies were extracted and analyzed by two authors. Summary relative risks (RRs) with corresponding 95% confidence intervals (CIs) were pooled using random- or fixed-effects models as appropriate. Sensitivity analyses and meta-regression were conducted to explore possible explanations for heterogeneity. Finally, publication bias was assessed. Fourteen observational studies involving thirteen cohort studies and one case-control study were included in the meta-analysis. Exposure to fluoride in drinking water does not significantly increase the incidence of hip fracture (RRs, 1.05; 95% CIs, 0.96–1.15). Sensitivity analyses based on adjustment for covariates, effect measure, country, sex, sample size, quality of Newcastle–Ottawa Scale scores, and follow-up period validated the strength of the results. Meta-regression showed that country, gender, quality of Newcastle–Ottawa Scale scores, adjustment for covariates and sample size were not sources of heterogeneity. Little evidence of publication bias was observed. The present meta-analysis suggests that chronic fluoride exposure from drinking water does not significantly increase the risk of hip fracture. Given the potential confounding factors and exposure misclassification, further large-scale, high-quality studies are needed to evaluate the association between exposure to fluoride in drinking water and hip fracture risk.

Nichole R Johnston (2020) et al conducted a Fluoride is ubiquitously present throughout the world. It is released from minerals, magmatic gas, and industrial processing, and travels in the atmosphere and water. Exposure to low concentrations of fluoride increases overall oral health. Consequently, many countries add fluoride to their public water supply at 0.7-1.5 ppm. Exposure to high concentrations of fluoride, such as in a laboratory setting often exceeding 100 ppm, results in a wide array of toxicity phenotypes. This includes oxidative stress, organelle damage, and apoptosis in single cells, and skeletal and soft tissue damage in multicellular organisms. The mechanism of fluoride toxicity can be broadly attributed to four mechanisms: inhibition of proteins, organelle disruption, altered pH, and electrolyte imbalance. Recently, there has been renewed concern in the public sector as to whether fluoride is safe at the current exposure levels. In this review, we will focus on the impact of fluoride at the chemical, cellular, and multisystem level, as well as how organisms defend against fluoride. We also address public concerns about fluoride toxicity, including whether fluoride has a significant effect on neurodegeneration, diabetes, and the endocrine system.

Joice Tom Job (2021) et al conducted fluoride ions are an important environmental contaminant and pollutant found in a wide variety of environmental conditions. The fluoride in drinking water is evident to induce toxic effects including neurodegeneration, skeletal and dental fluorosis as well as organ damage. Nutraceuticals and functional foods are emerging as possible preventive agents against fluoride toxicity. Hence, the possible use of an emerging functional food-the coconut haustorium is being evaluated against sodium fluoride-induced toxicity in intestinal cells (IEC-6). The cells exposed to fluoride showed significant cell death mediated through the increased lipid peroxidation and glutathione depletion. The glutathione biosynthetic enzymes were inhibited by the exposure to fluoride and the apoptotic genes (caspases 3/7 and apaf-1) were upregulated. The CHE pre-treatment improved the activity of enzymes involved in the de novo biosynthesis of glutathione and subsequently improved the intracellular GSH pool. The improved antioxidant defence was also evident from the reduced expression of apoptotic genes ($p < 0.05$). Overall, the study concludes that fluoride ions induce oxidative stress-mediated apoptosis in intestinal epithelial cells, via inhibiting glutathione biosynthesis. Methanol extract of coconut haustorium increased glutathione biosynthesis and subsequently prevented fluoride toxicity in IEC-6 cells by virtue of its antioxidant potentials.

Conceptual Sections

1. Mechanism of Action of Fluoride
   Fluoride exerts its cariostatic effect through:
- Enhancement of remineralization: Promotes formation of fluorapatite, which is more acid-resistant than hydroxyapatite.
- Inhibition of demineralization: Reduces enamel solubility during acid attacks.
- Antibacterial action: Inhibits bacterial enzymes, particularly in Streptococcus mutans, reducing acid production.
  Post-eruptive topical effect: The primary benefit is topical rather than systemic.
  Optimum Fluorides levels: 0.7 ppm to 1.2 ppm.

2. Benefits of Fluoride in Dentistry

a. Caries Prevention
- Reduction in caries prevalence in fluoridated communities.
- Effective in high-risk populations.
- Cost-effective public health intervention.

b. Modes of Delivery
- Community water fluoridation
- Fluoridated toothpaste
- Professional topical applications (varnishes, gels)
- Fluoride-releasing restorative materials
- Organizations such as the World Health Organization and the American Dental Association endorse optimal fluoride use for caries prevention.

3. Adverse Effects of Excess Fluoride

a. Dental Fluorosis
- Occurs during enamel development (up to 8 years of age).
- Ranges from mild white opacities to severe brown staining and pitting.
- Primarily aesthetic concern in mild forms.

b. Skeletal Fluorosis
- Results from prolonged ingestion of high fluoride levels.
- Causes bone pain, stiffness, and structural changes.

c. Acute Fluoride Toxicity
- Rare but possible with ingestion of high-dose supplements or dental products.
- Symptoms: nausea, vomiting, abdominal pain.
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Crtitcal Analysis Sections

The characterization of fluoride as a double-edged sword arises from its dose-dependent effects. At optimal concentrations (e.g., ~0.7 ppm in community water), fluoride significantly reduces caries incidence with minimal risk. However, increasing availability through multiple sources — water, toothpaste, mouth rinses, dietary intake — has raised concerns about cumulative exposure.

Key issues include:
- Narrow therapeutic index: Small margin between beneficial and harmful doses.
- Ethical debates: Community water fluoridation raises questions regarding individual autonomy versus collective benefit.
- Risk stratification challenges: Uniform public health measures may not suit all individuals equally.
- Overexposure in children: Swallowing fluoridated toothpaste contributes to fluorosis risk.
- While the caries-preventive benefits are well established, emerging discussions explore potential systemic associations,

though high-quality evidence remains inconclusive for most claimed risks.

Clinical Implications

- Dentists must adopt a patient-centred approach:
- Caries Risk Assessment: Tailor fluoride use based on individual risk.
- Age-Specific Recommendations:
  Smear layer of toothpaste (<3 years)
  Pea-sized amount (3–6 years)
- Supervised Brushing in children.
- Avoid unnecessary supplementation where water is fluoridated.
- Professional topical fluoride for high-risk patients only.
- Monitoring cumulative fluoride exposure.
  Balancing benefit and safety require precise dosage control and patient education.

Future Research Directions

- Long-term epidemiological studies on cumulative fluoride exposure.
- Biomarkers for early fluoride toxicity detection.
- Personalized preventive dentistry models.
- Alternative remineralizing agents (e.g., bioactive materials).
- Improved public health strategies minimizing fluorosis while preserving caries prevention.

DRAWBACKS

- Risk of dental fluorosis in children.
- Potential for skeletal fluorosis in endemic areas.
- Public mistrust and misinformation.
- Ethical controversy regarding mass fluoridation.
- Dependence on compliance in topical applications.

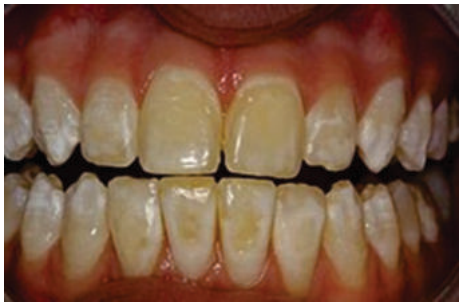

Fig 4 Shows the Dental Fluorosis

Fig 5 Shows the Skeletal Fluorosis

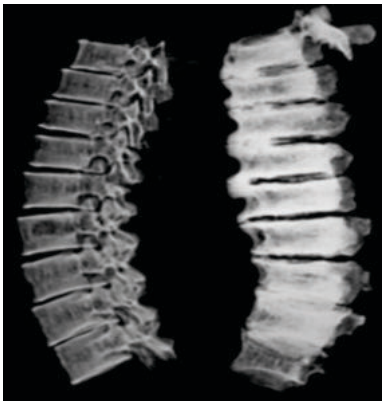

Fig 6 Shows the Radiographic Image of Skeletal Fluorosis

CONCLUSION

Fluoride remains a cornerstone of preventive dentistry due to its proven effectiveness in reducing dental caries. However, its benefits are dose-dependent, and excessive exposure carries measurable risks. The concept of fluoride as a double-edged sword underscores the importance of balanced application, evidence-based guidelines, and individualized patient care. Optimal fluoride use — neither deficient nor excessive — is essential to maximize oral health benefits while minimizing adverse effects. Looking at global scenario and available evidence, it is highly recommended that Government of India should make some policies to regulate the use of fluorides from time to time.

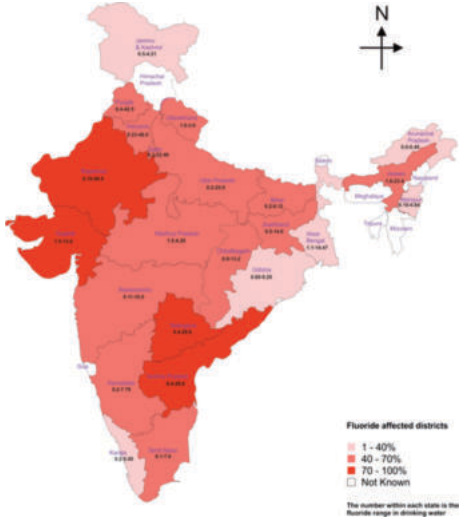

Fig 7 Shows the Fluoride Mapping of India

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