



Journal Homepage: - [www.journalijar.com](http://www.journalijar.com)

## INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI: 10.21474/IJAR01/11741

DOI URL: <http://dx.doi.org/10.21474/IJAR01/11741>



### RESEARCH ARTICLE

#### DENTAL FLUOROSIS AND PERIODONTAL DISEASE: AN OVERVIEW

Ashish Yadav

#### Manuscript Info

##### Manuscript History

Received: 20 July 2020

Final Accepted: 24 August 2020

Published: September 2020

##### Key words:-

Dental Fluorosis, Fluoride, Fluorine,  
Oral Health, Periodontal Disease

#### Abstract

**Aim:** The aim of this review is to discuss various effects of fluoride on hard and soft tissues of the periodontium and its importance in human life.

**Background :** Fluoride is an essential element for life and is one of the trace elements normally present in the body. It is abundant in the environment and the main source of fluoride to humans is drinking water. Fluoride gets accumulated in hard tissues of the body and has been known to play an important role in mineralization of bone and teeth. The behaviour of fluoride ions in the human organism can be regarded as that of “double-edged sword”. In small amounts, it is known to have beneficial effects on dental health. On the other hand, excessive chronic intakes can result in adverse effects including the development of dental fluorosis in children and/or skeletal fluorosis in both children and adults. Although effect of fluoride on caries has been discussed in painstaking details through various studies but the effect of fluorosis on the periodontium yet remains in shadow.

**Review Results :** Dental fluorosis is a developmental disturbance of dental enamel, caused by successive exposures to high concentrations of fluoride during tooth development, leading to enamel with lower mineral content and increased porosity. Even after continuing with the age old logic of structural changes that take place in mottled enamel it can be said with scientific plausibility that this factor of surface roughness can or must influence some of the variables in this multifactorial disease of periodontitis. This surface roughness is conducive for the bacteria to survive as well as make it difficult for scaling and root planing in fluorosed teeth. This could also jeopardize the effectiveness of the regular oral hygiene procedures.

**Conclusion :** Dental fluorosis is not only a cosmetic problem that impairs social well-being but also affects the oral health related quality of life. Fluorosis continues to be an important problem, both for the affected individuals and for public health. More and more areas are being discovered regularly that are affected by fluorosis in different parts of the country. But ultimate solution for this fluoride menace remains to be the principal of “Precaution is better than cure”.

**Clinical Significance :** Considering the role of fluorosis on hard and soft tissues and all the risk factors of periodontitis, fluorosis can be recommended strongly as an environmental risk factor for periodontitis. To be defined as one of the etiological (environmental) agent of

periodontal disease requires further research studies with greater sample size from varying areas globally.

*Copy Right, IJAR, 2020,. All rights reserved.*

## Introduction:-

### Background:

Fluoride has been described as an essential element needed for normal growth and development of animals and extremely useful for human beings. It is abundant in the environment and the main source of fluoride to humans is drinking water. The Fluoride ion comes from the element fluoride. In the Earth's crust, fluorine is the 17th most abundant element, is a gas and never occurs in a free state in nature.<sup>1</sup>

Negligible concentrations of airborne fluorides are present in atmosphere<sup>2</sup>. Items of food such as fish (Sardines) may contribute to higher dietary fluoride intake. Brewed teas may also contain fluoride concentration of 1-6 ppm depending on the water fluoride concentration, amount of dry tea used and the brewing time<sup>3</sup>. The average daily dietary intake of fluoride (expressed on a body weight basis) by children residing in optionally fluoridated (1 ppm) communities is 0.05 mg/Kg/day, in communities without optionally fluoridated water, average intakes for children are about 50 % lower. Dietary fluoride intake by adults in optionally fluoridated (1 ppm) areas averages 1.4-3.4 mg/day, and in non-fluoridated areas avg. 0.3-1.0 mg/day<sup>4</sup>.

The US Public Health Service (USPHS, 1986) based on extensive research had established the optimum concentration for fluoride in the water in the United States in the range of 0.7-1.2 ppm. This range effectively reduces tooth decay while minimizing the occurrence of dental fluorosis.

The optimum level of fluoride in water is dependent on the annual average of the maximum daily air temperature in the geographic area<sup>5</sup>.

### Facts from India:

In India, fluoride level in ground water varies substantially in different regions. High concentration of fluoride (>1.5 mg/l) have been reported in the states of Haryana, Delhi, Rajasthan, Karnataka, UP, Maharashtra etc<sup>6</sup>.

In India, 20 states had been identified which are affected with endemic fluorosis. About 62 million people, including 6 million children are at risk in India suffering from dental, skeletal and/or non-skeletal fluorosis.<sup>7</sup> The problem has reached alarming proportions affecting various districts of the states. On the basis of districts affected, it can be categorized into 3 categories.<sup>8</sup>

- |             |                        |
|-------------|------------------------|
| 1. 50-100 % | Districts are affected |
| 2. 30-50 %  | Districts are affected |
| 3. <30 %    | Districts are affected |

Fluoride levels in drinking water are also found to be low or normal in certain areas. Ground water is being used for drinking purposes due to lack of central water supply in most of the country. Fluoride mapping has not been carried out in India properly so as to locate areas with normal, low or high levels of fluoride.

Dietary fluoride supplements are available only by prescription and are intended for use by children living in non-fluoridated areas to increase their fluoride exposure so that it is similar to that by children who live in optimally fluoridated areas<sup>9-10</sup>. These supplements are available in two forms: Drops for infants aged six months & up and chewable tablets for children and adolescents<sup>11</sup>. The correct amount of a fluoride supplement is based on the child's age and the existing fluoride level in the drinking water.

### Metabolism of fluoride:

In humans, the predominant route of fluoride absorption is via the gastrointestinal tract. Except for occupational exposure or exposure to fluoride by coal or fuel burning, exposure to fluoride by inhalation is negligible<sup>12,13</sup>. Dermal absorption is insignificant but seen in cases of hydrofluoric acid burns<sup>14</sup>.

Fluoride exists in two forms, unit and bound forms in plasma with the bound form being present in larger quantity. Fluoride concentrations in human saliva are slightly less than those found in plasma, ranging from less than 0.01 to 0.05 ppm.<sup>1</sup>

In adults, about 50 % of daily fluoride intake is associated with the calcified tissues within 24 hours and the remaining 50 % is excreted in urine. This 50:50 distribution is strongly shifted to greater retention in the very early years of life and probably towards greater excretion in the later part.<sup>15</sup> In adults, about 40-60 %<sup>16</sup> of the daily intake of fluoride is excreted in the urine and in children about 45 %<sup>17</sup> in faeces less than 10 % of the daily intake of fluoride is excreted<sup>18,19</sup>. It was estimated that 1 % or less of an ingested dose is excreted in saliva, which returns back to systemic circulation<sup>20</sup>. Only a minor route of fluoride excretion is provided by sweat.<sup>21</sup>

#### **Fluoride in hard tissues:**

##### **Teeth:**

Fluoride is obtained in two forms: Topical and systemic. Topical fluorides help in strengthening the teeth already present in the mouth. Local protection on the tooth surface is provided by topically applied fluorides. It includes toothpastes, mouth rinses and professionally applied fluoride gels.

Sources of systemic fluorides include water, food, beverages and dietary fluoride supplements in the form of tablets, drops or lozenges.

##### **Fluoride's Caries preventive effects:**

Researches have observed three specific mechanisms<sup>22,23</sup>

1. It exerts an influence directly on dental plaque by reducing the ability of plaque organisms to produce acid.
2. It reduces the solubility of enamel in acid by converting hydroxyapatite into less soluble fluorhydroxyapatite / fluorapatite.
3. Promotes remineralization or repair of tooth enamel in areas that have been demineralized by acids.

##### **Bone:**

It has been established through numerous studies that fluoride is bound within the bone replacing hydroxyl or bicarbonate groups normally associated with hydroxyapatite structures and it increases the crystallinity or crystal structure of the apatite.<sup>1</sup>

##### **Fluoride toxicity:**

At recommended levels prolonged use of fluoride does not produce any harmful physiological effects in the human. However, there are safe limits beyond which harmful effects can occur. These effects can be classified as acute and chronic toxicity.

##### **Acute toxicity:**

It occurs due to single ingestion of a large amount of fluoride. The amount of fluoride considered lethal when taken actually as 35- 70 mg F per Kg body weight. Symptoms of acute toxicity occur rapidly which are vomiting, diarrhea, diffuse abdominal pain, excess salivation and thirst.

##### **Chronic toxicity:**

Is caused due to long term ingestion of smaller amounts of fluoride in drinking water. Excessive fluoride more than 8 ppm in drinking water daily for many years can lead to skeletal fluorosis. Severe cases are normally found only in warm climates where drinking water contains very high levels of fluoride. Due to chronic toxicity bone density slowly increases, the joints stiffen and become painful.

At higher levels of ingestion (2-8 mg daily), skeletal fluorosis may arise. Dental fluorosis is easily recognized but skeletal involvement is not clinically obvious until the advanced stage and also early cases may be misdiagnosed as rheumatoid arthritis or osteoarthritis.<sup>24</sup>

##### **Fluorosis:**

It occurs in humans as dental and skeletal fluorosis. They are separated by a prolonged relatively symptom free interval during which the skeleton does not stop accumulating fluoride. Skeletal fluorosis causes crippling deformities and neurological complications in its advanced stages. The effects of fluoride intoxication are related to

the total amount of fluoride ingested, although only water was taken into account earlier presumably because supply of fluoride by food was deemed negligible.

**The severity of fluorosis has a definite relationship with the following factors :**

1. Fluoride concentration in drinking water
2. Fluoride ingestion through other sources.
3. Period of exposure
4. Climatic factors ( Temperature)
5. Nutritional status
6. Occupation

**Table 1:-** Concentration of fluoride in drinking water and its effects on human health<sup>25</sup>.

Fluoride Concentration in (mg/l)	Effects
Nil	Limited growth and fertility
< 0.5	Dental Caries
0.5-1.5	Promotes dental health, prevents tooth decay
1.6-4.0	Dental fluorosis (mottling and pitting of teeth)
4.1-10.0	Dental fluorosis, Skeletal fluorosis (Pain in neck and back)
>10.00	Crippling fluorosis

**Dental Fluorosis:**

Fluorosis is irreversible and only occurs with exposure to fluoride where enamel is developing. During the early childhood years excessive ingestion of fluoride may damage the tooth forming cells, leading to a defect in the enamel known as dental fluorosis. The teeth affected by fluorosis have visible discoloration ranging from white chalky spots to brown and black stains. Fluorosed teeth also have increased porosity of the enamel. Both sexes are equally affected<sup>25</sup>

In India itself, an estimated 60 million people are at risk and 6 million people are disabled<sup>26</sup> Fluorosis is known to cause hypoplastic changes in tooth enamel. Discolorations, pits and striations are common observations in fluorosed teeth<sup>27</sup>. Apart from this, fluorosis can even induce gross morphological changes in the form of root malformations<sup>28</sup>

Instead of normal creamy- white translucent in color, fluorosed enamel is porous and opaque, teeth can resemble a ghastly white chalk color. Cloudy striated (lines of demarcation) enamel, white specks or blotches, snow-capping'' yellowish-brown spots or brown pits on teeth are all characteristic of fluorosis. Fluorosed enamel is structurally weak (brittle) and prone to erosion and breakage in its more severe form. Even in the milder forms, there is increased enamel attritions<sup>1</sup>.

Fluorosis is a toxic manifestation of chronic (low dose, long term) fluoride intake. To prevent fluorosis from occurring in the most prominent and /or most susceptible teeth, the most critical time to avoid fluoride exposure is the first 3-6 years of a child's life<sup>1</sup>.

Effect of fluoride on enamel formation can follow several possible pathogenic pathways ( Fejerskov et al.1970)<sup>29</sup> :

1. Effect on ameloblast.
2. Effect on calcium homeostasis generally with dental fluorosis as an indirect result.
3. Effect on nucleation and crystal growth in all stages of enamel formation.

Mottled appearance and altered form are produced only when excessive amount of fluorides are ingested during the period of formation and calcification i.e. During the first eight years of life. After the tooth erupts and calcification has been completed, ingested fluoride does not have adverse dental consequences.

Fluorosis affect mainly permanent dentition and very high fluoride levels (>10 ppm) are required in drinking water for it to cross placental barrier and affect primary dentition. Dental fluorosis might be more than a cosmetic defect if enough fluorotic enamel is fractured and lost to cause pain, compromise chewing efficiency, adversely affect food choices and require complex dental treatment.<sup>1</sup>

Fluoride toxicity at high levels has been associated with thyroid changes, growth retardation with thyroid changes, kidney changes and even urolithiasis. Present data indicate that some subsets of the population may be unusually susceptible to the toxic effects of fluoride and its compounds. These population include the elderly people with magnesium, calcium and/or vitamin C deficiency and also people with kidney and cardiovascular problems<sup>1</sup>.

Also, fluoride influences the pattern of distribution of serum proteins<sup>30</sup> It also contributes to genetic alterations inducing up-regulation and down regulation of genes<sup>31</sup>

**Table II:-** Dean's Fluorosis Index<sup>32</sup>(Modified).

Score	Criteria
<b>Normal (0)</b>	The enamel represents the usual translucent semivitriform type of structure. The surface is smooth, glossy and usually of a pale, creamy white color.
<b>Questionable (0.5)</b>	The enamel discloses slight aberrations from the translucency of normal enamel, ranging from a few white flecks to occasional white spots.
<b>Very mild (1.0)</b>	Small, opaque, paper white areas scattered irregularly over the tooth, but not involving as much as approximately 25% of the tooth surface.
<b>Mild (2.0)</b>	The white opaque areas in the enamel of teeth are more extensive, but do not involve as much as 50% of tooth.
<b>Moderate (3.0)</b>	All enamel surfaces of the teeth are affected and surfaces subject to attrition show wear. Brown stain is frequently a disfiguring feature.
<b>Severe (4.0)</b>	All enamel surfaces of the teeth are affected and hypoplasia is so marked that the general form of the tooth may be affected. The major diagnostic sign of this classification is discrete or confluent pitting. Brown stains are widespread and teeth often present a corroded-like appearance.

#### **Dental fluorosis and Periodontium:**

Dental fluorosis is known for the changes it induces in the hard tissues of the body but not much attention has been given to its relation with the surrounding periodontal structures. Similarly even though a limited studies does consider the prevalence of periodontitis in fluorosis subjects, a little or no literature is present regarding the progress of periodontal disease in fluorosis affected population.

Many studies along with published documents have demonstrated that increased fluoride exposure is directly linked to increased periodontal disease. From a public health perspective, any study that demonstrates increased fluoride exposure may increase the risk of developing periodontal disease or other illness are of major importance.

#### **Historical evidence:**

In 1936, Dean H.T<sup>33</sup> wrote in the Journal of the American Medical Association: 'From observations that I made in areas of relatively high fluoride concentration (more than 4 ppm) there is sufficient evidence to suggest that there is an apparent tendency toward a higher incidence of gingivitis (periodontal disease) .

Similarly such findings were seen by Murray J John<sup>34</sup> (1972), Haikel T et al<sup>35</sup> (1989), Vandana KL et al<sup>36</sup> (2007) Vora et al<sup>37</sup> (2013) and Priyanka J Dalvi et al<sup>38</sup> (2017) in their studies.

Murray J John (1972)<sup>34</sup> conducted a study to compare gingivitis and gingival recession between Hartlepool (1.2 – 2.0 ppm) and York (0.15 – 0.2ppm) and found that these two conditions were more prevalent in high fluoride areas than low fluoride areas.

In 1989 Haikel T et al<sup>35</sup> performed the study to determine the periodontal status using CPITN index in a population aged 7-60 years residing in the fluoride area of Khouribga and non-fluoride area of Beni-Mellal, Morocco. A total of 2378 subjects were studied . Results showed that extensive gingivitis , low to moderate prevalence of shallow pockets with increasing age and very low prevalence of deep pockets at all ages.

Vandana KL et al (2007)<sup>36</sup> conducted a study to determine the periodontal status using CPITN index in a population residing in the high fluoride areas of Davangere district. Results of this study showed that as the degree of fluorosis increased, severity of gingivitis reduced and periodontitis increased ie with A degree of fluorosis, gingivitis was 89.4

% and periodontitis 8.5 % but with F degree of fluorosis the former was 64 % and the latter 35.8 % which was statistically significant.

In 2013 Voraet al<sup>37</sup> in his cross-sectional survey, showed that there is a statistically significant correlation between severity of dental fluorosis and severity of periodontal diseases and he concluded, dental fluorosis may have significant effect on periodontal condition.

Priyanka J Dalvi et al<sup>38</sup> in 2017 did a study with an aim to investigate whether fluorosis acts as a risk factor for periodontal disease and to assess salivary oxidative stress in fluorosed and nonfluorosed patients with periodontitis contributing to periodontal disease. Results showed that gingivitis was significantly higher in nonfluorosed than in fluorosed group in contrast, periodontitis was significantly higher in fluorosed group than in nonfluorosed group. Gingivitis appeared to decline as the fluoride status worsened, while periodontitis showed an increasing gradient from lower fluoride score to higher fluoride score.

Human studies conducted to analyze the effect of fluoride on periodontal status have elucidated varying results suggestive of no relation between periodontal health and consumption of fluoride (Zimmerman 1955)<sup>39</sup>, decrease in periodontal index score and attachment loss (Grembowski 1993)<sup>40</sup> among adults living in a natural fluoride area, increase in gingival bleeding index score (Parviainen K 1977)<sup>41</sup> gingivitis and gingival recession (Murray J John 1972)<sup>34</sup> among individuals from high fluoride area.

Even after continuing with the age old logic of structural changes that take place in mottled enamel it can be said with scientific plausibility that this factor of surface roughness can or must influence some of the variables in this multifactorial disease of periodontitis.

Surface roughness was revealed to be exceedingly high in fluorosis subjects, with roughness increasing with degree of fluorosis after analyzing with atomic microscopy.<sup>42</sup>

This roughness of tooth surface is conducive for the bacteria to survive as well as make it difficult for scaling and root planing in fluorosed teeth. Also this could jeopardize the effectiveness of the regular oral hygiene procedures.<sup>43</sup>

#### **Prevention and mitigation of fluorosis:**

Dental fluorosis is irreversible in nature which requires complex and expensive procedures which are time consuming and are not easily available to rural population. There is no specific treatment in case of skeletal fluorosis. Therefore, due to lack of sustainable treatment measures for any form of fluorosis, prevention and control through interventions (provision of safe water and food) is said to be the best approach to mitigate fluorosis.<sup>44</sup>

There are some interventions to practice for the management of the disease. Fluorosis can be totally prevented and the individual can lead a normal, healthy life. Fluorosis can be prevented or minimized by using alternative water sources, removing excessive fluoride from drinking water and by improving the nutritional status of population at risk. The simple interventions include provision of surface water, rain water and consumption of low-fluoride ground water.<sup>45</sup>

Other interventions are defluoridation of water through flocculation and adsorption. Similarly, health education programmes and better nutrition are some of the cost-effective intervention measures.<sup>45</sup>

#### **Levels of prevention:**

##### **Primary level:**

Limitation of the fluoride content of drinking water to <0.5 ppm is the only practical and effective public health measure for the prevention and control of dental fluorosis and caries by using deep bore drinking water supplies and adequate calcium intake (dietary calcium > 1g/day).<sup>46</sup>

##### **Secondary level:**

Improving the nutritional status, especially of expecting mothers, newborns and children up to the age of 12 years is of high importance. Treating other causes of fluoride toxicity such as kidney and thyroid diseases etc.. Food and other substances rich in fluoride should be avoided. eg. Kala namak, Black tea, Tobacco, Supari and use of fluoride containing water. Also people should consume adequate amount of calcium, Vitamin E and antioxidants.<sup>47</sup>

**Tertiary level:**

Teeth once affected by dental fluorosis cannot be reversed to normal. But the discoloured teeth can be masked by bleaching and/or by other methods. For fluorosed teeth various treatments available are:

1. Bleaching of teeth
2. Light cure filling and laminated veneering
3. Crowns placement on teeth with metals like chrome, cobalt, gold, porcelain and acrylic.

**Conclusion:-**

Dental fluorosis is not only a cosmetic problem that impairs social well-being but also affects the oral health related quality of life. Both for the affected individuals and for public health, fluorosis continues to be an important problem. In different parts of the country, more and more areas are being discovered regularly that are affected by fluorosis. But ultimate solution for this fluoride menace remains to be the principal of "Precaution is better than cure".

It is very clear that fluoride in recommended concentrations is definitely beneficial to health. So as to capitalize on the beneficial effects of fluoride, judicious use of fluoride supplements is mandatory. Measures should be taken to use fluoride to our advantage in achieving optimal health.

Dentists as well as public health dentists are at the center of this problem and can help in spreading awareness about ill effects of excessive amount of fluoride in water therefore, significantly contributing to society and preventing them from the harmful effects of dental and skeletal fluorosis.

**Clinical Significance:**

Considering the role of fluorosis on hard and soft tissues and all the risk factors of periodontitis, fluorosis can be recommended strongly as an environmental risk factor for periodontitis. To be defined as one of the etiological (environmental) agent of periodontal disease, further research studies with greater sample size are required from varying areas globally.

**References:-**

1. V Dhar, M Bhatnagar. Physiology and toxicity of fluoride. Indian J Dent Res, 2009; 20(3): 350-355
2. Hodge HC, Smith FA. Occupational Fluoride exposure. J Occup Med 1977; 19: 12-39
3. Whitford GM. The Metabolism and Toxicity of Fluoride 1996, 2nd Ed, Karger, Basel, Switzerland
4. Institute of Medicine, Food and Nutrition Board. Dietary Reference intakes for calcium, phosphorus, magnesium, Vitamin D and fluoride. Report of the standing committee on the scientific evaluation of dietary reference intakes. Washington DC. National Academy Press.
5. US Department of Health and Human Services, Centers for Disease Control, Dental Disease Prevention Activity. Water Fluoridation: A manual for engineers and technicians. Atlanta; 1986
6. Susheela AK. Fluorosis: Indian scenario. A Treatise on Fluorosis. Fluorosis Research and Rural Development Foundation; 2001. p. 13-50
7. Srikant, Chandra S. Endemic Fluorosis in Five Villages of The Palamau District, Jharkhand, India. Fluoride. 2008; 41(3): 206-211
8. Joshi V, Joshi N K. Fluorosis and Its Impact on Public Health in Jodhpur. International Journal of Basic and Applied Medical Sciences 2014; 14(2): 87-92
9. Largent E. The supply of fluorine to man: 1. Introduction. Fluorides and human health. Geneva: World Health Organization Monograph Series No. 59; 1970. p. 17-80
10. Rugg-Gunn AJ. Nutrition and dental health. New York: Oxford University Press; 1993.
11. American Dental Association, Council on Access Prevention and Interprofessional Relations. Caries Diagnosis and risk assessment: A review of preventive strategies and management. J Am Dent Assoc 1995; 126
12. L Li, K Luo, Y Tang, Y Liu. The daily fluorine and arsenic intake for residents with different dietaries and fluorosis risk in coal-burning fluorosis area, Yunnan, Southwest China. Environ Sci Pollut Res 2015; 22: 2031-2040
13. World Health Organization (WHO), Air Quality Guidelines for Europe 2nd Ed, World Health Organization, Geneva, Switzerland 2000
14. World Health Organization (WHO), Environmental Health Criteria 36, Fluorine and Fluorides, World Health Organization, Geneva, Switzerland 1984
15. Whitford GM. Intake and metabolism of fluoride. Adv. Dent Res 1994; 8: 5-14

16. Ekstrand J, Ehrnebo M. The relationship between plasma fluoride, urinary excretion rate and urine fluoride concentration in man. *J Occup. Med.* 1983;25(10):745-748
17. Villa A, Anabalon M, Zohouri V, Maguire A, Franco Am, Pug-Gunn A. Relationship between fluoride intake, Urinary fluoride excretion and fluoride retention in children and adults: An analysis of available data. *Caries Res.* 2010;44:60-68
18. Ekstrand J, Hardell LI, Spak CJ. Fluoride balance studies on infants in a 1 ppm water fluoride area. *Caries Res.* 1984;18:87-92
19. Ekstrand J, Ziegler EE, Nelson SE, Fomon SJ. Absorption and retention of dietary and supplemental fluoride by infants. *Adv Dent Res* 1994;8:175-180
20. Oliveby A, Lagerlof F, Ekstrand J, Dawes C. Studies on fluoride concentration in human submandibular/sublingual saliva and their relation to flow rate and plasma fluoride levels. *J Dent. Res.* 1989;68:146-149
21. Whitford GM. *The Metabolism and Toxicity of Fluoride* 1996, 2nd Ed, Karger, Basel, Switzerland
22. De Paola PF, Kashket S. Prevention of dental caries. In: Fluorides, effects on vegetation, animals and humans. Schupe JL, Peterson HB, Leone NC. Editors. Salt lake city: Paragon Press;1983. P. 199-211
23. Melberg JR, Ripa LW. Fluoride in preventive dentistry: Theory and clinical applications. Chicago: Quintessence; 1983. P. 41-80
24. Melberg JR, Ripa LW. Fluoride in preventive dentistry: Theory and clinical applications. Chicago: Quintessence; 1983. P. 81-102
25. Ranjan R, Maiti SB, Vala D, Jain A, Shrivastava S. Fluorides in health and disease – A Review. *Journal of Advanced Medical and Dental Sciences Research* 2016;4(6):43-51
26. Reddy DR. Neurology of endemic skeletal fluorosis. *Neurol India.* 2009;57:7-12
27. Makarov SV, Spitsyn VA, Kravchuk OI, Bychkovskaia LS. Qualitative and quantitative variation of serum proteins in fluorosis patients. *Genetika.* 1999;35(9):1305-1308
28. Hou GQ, Liu JL, Yu YY, Xia T. Screening of environmental response genes related to dental fluorosis. *Wei Sheng Yan Jiu.* 2005;34(5):543-546
29. Fejerskov O, Thylstrup A, Lasen MJ. Rational Use of Fluorides in Caries Prevention-A Concept Based on Possible Cariostatic Mechanism. *Acta Odontol Scand* 1981;39:241-249
30. Thylstrup A. Post-eruptive development of isolated and confluent pits in fluorosed enamel in a 6-year old girl. *Scand J Dent Res* 1983;91(4):243-246
31. Vazirani SJ, Singh A. Endemic fluorosis: radiological features of dental fluorosis. *J Indian Dent Assoc.* 1968;40(11):299-303
32. Soben Peter. *Essentials of preventive and community dentistry* (5th ed.) Arya publishing house, New Delhi, India.
33. Dean HT, Elvove e. epidemiological aspects of chronic endemic dental fluorosis. *Am. J public health* 1936; 26:567-575
34. Murray J John. Gingivitis and Gingival Recession in Adults from High Fluoride and Low Fluoride Area *Arch Oral Biol.* 1972; 17:1269-1277
35. Haikel Y, Tiirlot JC, Cahen PM, Frank R. Periodontal treatment needs in populations of high and low fluoride areas of Morocco. *J Clin Periodontol* 1989;16:596-600
36. Vandana KL, Reddy SM. Assessment of periodontal status in dental subjects using CPITN. *Ind J Dent Res* 2007;18:67-71
37. Vora KS, Vora PK. Assessment of periodontal status of the patients with dental fluorosis in area with natural high levels of fluoride : A cross sectional survey
38. Dalvi PJ, Vandana KL, Gosh S, Joshi VM, Bhat K, Prakash VH. Fluorosis: Environmental Risk Factor for Periodontal Disease. *J of Postgraduate Medicine, Education and Research.* 2017; 51(4): 157-161
39. Zimmermann ER, Leone NC, Arnold FA. Oral Aspects of Excessive Fluoride in A Water Supply. *J Am Dent Assoc* 1955;50:272-275
40. Grembowski D, Fiset L, Spadafora A, Milgrom P. Fluoridation Effects on Periodontal Disease Among Adults. *J Periodont Res* 1993; 28: 166
41. Parvialwen K, Nordling H, Ainamoj. Occurrence of Dental Caries and Gingivitis in Low Medium and High Fluoride Areas in Finland. *Comm Dent Oral Epidemiol* 1977; 5:287
42. Susheela AK, Bhatnagar M, Gnanasundram N, Saraswathy TR. Structural Aberrations In Fluorosed Human Teeth. *Biochemical And Scanning Electron Microscopic Studies.* *Curr. Sci* 1999; 77:1-6
43. Singh P, Gupta ND, Bey A. Dental fluorosis and periodontiom: A game of shadows. *Journal of Oral Biology and Craniofacial Research.* 2014;4:47-48



44. Khairnar MR, Dodamani AS, Jadhav HC, Naik RG, Deshmukh MA. Mitigation of fluorosis – A Review. Journal of Clinical and Diagnostic Research 2015;9(6):5-9
45. UNICEF Position on Water Fluoridation. Fluoride in water: An overview. [http://www.nofluoride.com/Unicef\\_fluor.cfm](http://www.nofluoride.com/Unicef_fluor.cfm)
46. Habbu N. et al. Esthetic management of dental fluorosis. International journal of dental clinics. 2011;3(2):80-81
47. Shinde A, Shinde M. Environmental fluorine and associated morbidities. Journal of Environmental Research and Development. 2006;1(1):32-34.