

# EFFECTS OF DIFFERENT CONCENTRATIONS OF FLUORIDE TOOTHPASTE IN PREVENTION OF DENTAL CARIES

Type of article: Review

Running title: Effect of fluoride toothpaste on dental caries.

**S.S.SHIVANNI**

Undergraduate  
Saveetha Dental College,  
Saveetha University,  
Chennai, India

**Dr.GANESH.J**

Senior lecturer,  
Department of pedodontics  
Saveetha Dental College,  
Saveetha University,  
Chennai, India

**Corresponding Author**

**Dr.Ganesh.J**

Senior lecturer  
Department of pedodontics  
Saveetha Dental College,  
Saveetha University,  
Chennai, India

**Abstract:** Use of fluoride has been a major factor in the decline in the prevalence and severity of dental caries. When used appropriately, fluoride is both safe and effective in preventing and controlling dental caries. Although pit and fissure sealants, meticulous oral hygiene, and appropriate dietary practices contribute to caries prevention and control, the most effective and widely used approaches have included fluoride use. The relative role of enamel fluoride in caries prevention has become increasing during the last two decades. No significant relationship has been demonstrated between caries experience of the individual and fluoride content of the enamel. Furthermore, the fluoride content in surface enamel between teeth developed in low and fluoride areas is too small to explain any significant effect on dissolution rate of the enamel.

**Keywords:** Fluoride, Dental caries, Enamel, prevention, oral hygiene

## INTRODUCTION:

Dental caries is an infectious, multifactorial disease affecting most people in developing countries. Fluoride reduces the incidence of dental caries and slows or reverses the progression of existing lesions (i.e., prevents cavities). Although pit and fissure sealants, meticulous oral hygiene, and appropriate dietary practices contribute to caries prevention and control, the most effective and widely used approaches have included fluoride use. Fluoride modalities are effective, inexpensive, readily available, and can be used in both private and public health settings, their use is likely to continue [1]. Fluoride is the ionic form of the element fluorine, the 13th most abundant element in the earth's crust. Fluoride is negatively charged and combines with positive ions to form stable compounds. Such fluorides are released into the environment naturally in both water and air. Fluoride compounds also are produced by some industrial processes that use the mineral apatite, a mixture of calcium phosphate compounds. In humans, fluoride is mainly associated with calcified tissues because of its high affinity for calcium. Fluoride's ability to inhibit or even reverse the initiation and progression of dental caries is well documented [2-5]. The success of water fluoridation in preventing and controlling dental caries led to the development of fluoride-containing products, including toothpaste (i.e., dentifrice), mouthrinse, dietary supplements, and professionally applied or prescribed gel, foam, or varnish[6]. Dental caries, otherwise known as tooth decay, is one of the most prevalent chronic diseases of people worldwide; individuals are susceptible to this disease throughout their lifetime. Dental caries forms through a complex interaction over time between acid-producing bacteria and fermentable carbohydrate, and many host factors including teeth and saliva. The disease develops in both the crowns and roots of teeth, and it can arise in early childhood as an aggressive tooth decay that affects the primary teeth of infants and toddlers. Risk for caries includes physical, biological, environmental, behavioural, and lifestyle-related factors such as high numbers of cariogenic bacteria, inadequate salivary flow, insufficient fluoride exposure, poor oral hygiene, inappropriate methods of feeding infants, and poverty. The approach to primary prevention should be based on common risk factors. Secondary prevention and treatment should focus on management of the caries process over time for individual patients, with a minimally invasive, tissue-preserving approach [7].

## RELATION BETWEEN FLUORIDE AND DENTAL CARIES:

Dental caries is an infectious, transmissible disease in which bacterial by-products dissolve the hard surfaces of teeth. Unchecked, the bacteria can penetrate the dissolved surface, attack the underlying dentin, and reach the soft pulp tissue. Dental caries can result in loss of tooth structure, pain, and tooth loss and can progress to acute systemic infection. Cariogenic bacteria reside in dental plaque, a sticky organic matrix of bacteria, food debris, dead mucosal cells, and salivary components that adheres to tooth enamel. Plaque also contains minerals, primarily calcium and phosphorus, as well as proteins, polysaccharides, carbohydrates, and lipids. Cariogenic bacteria colonise on tooth surfaces and produce polysaccharides that enhance adherence of the plaque to enamel. Left undisturbed, plaque will grow and harbour increasing numbers of cariogenic bacteria. An initial step in the formation of a carious lesion takes place when cariogenic bacteria in dental plaque metabolise a substrate from the diet and the acid produced as a metabolic by-product demineralises the adjacent enamel crystal surface. Demineralisation involves the loss of calcium, phosphate, and carbonate. These minerals can be captured by surrounding plaque and be available for reuptake by the enamel surface. Fluoride, when present in the mouth, is also retained and concentrated in plaque. Fluoride works to control early dental caries in several ways. Fluoride concentrated in plaque and saliva inhibits the demineralisation of sound enamel and enhances the remineralization of demineralised enamel [8,9]. As cariogenic bacteria metabolize carbohydrates and produce acid, fluoride is released from dental plaque in response to lowered pH at the tooth-plaque interface [10]. The released fluoride and the fluoride present in saliva are then taken up, along with calcium and phosphate, by demineralized enamel to establish an improved enamel crystal structure. This improved structure is more acid resistant and contains more fluoride and less carbonate [11-15].

Fluoride also inhibits dental caries by affecting the activity of cariogenic bacteria. As fluoride concentrates in dental plaque, it inhibits the process by which cariogenic bacteria metabolize carbohydrates to produce acid and affects bacterial production of adhesive polysaccharides [16]. In laboratory studies, when a low concentration of fluoride is constantly present, one type of cariogenic bacteria, *Streptococcus mutans*, produces less acid [17-20]. Whether this reduced acid production reduces the cariogenicity of these bacteria in humans is unclear [21].

Saliva is a major carrier of topical fluoride. The concentration of fluoride in ductal saliva, as it is secreted from salivary glands, is low — approximately 0.016 parts per million (ppm) in areas where drinking water is fluoridated and 0.006 ppm in nonfluoridated areas [22]. This concentration of fluoride is not likely to affect cariogenic activity. However, drinking fluoridated water, brushing with fluoride toothpaste, or using other fluoride dental products can raise the concentration of fluoride in saliva present in the mouth 100- to 1,000-fold. The concentration returns to previous levels within 1–2 hours but, during this time, saliva serves as an important source of fluoride for concentration in plaque and for tooth remineralization [23].

Applying fluoride gel or other products containing a high concentration of fluoride to the teeth leaves a temporary layer of calcium fluoride-like material on the enamel surface. The fluoride in this material is released when the pH drops in the mouth in response to acid production and is available to remineralize enamel [24].

In the earliest days of fluoride research, investigators hypothesized that fluoride affects enamel and inhibits dental caries only when incorporated into developing dental enamel (i.e., pre-eruptively, before the tooth erupts into the mouth) [25,26]. Evidence supports this hypothesis [27-29], but distinguishing a true pre-eruptive effect after teeth erupt into a mouth where topical fluoride exposure occurs regularly is difficult. However, a high fluoride concentration in sound enamel cannot alone explain the marked reduction in dental caries that fluoride produces [30,31]. The prevalence of dental caries in a population is not inversely related to the concentration of fluoride in enamel [32], and a higher concentration of enamel fluoride is not necessarily more efficacious in preventing dental caries [33]. The prevalence and severity of dental caries in the United States have decreased substantially during the preceding 3 decades [34]. National surveys have reported that the prevalence of any dental caries among children aged 12–17 years declined from 90.4% in 1971–1974 to 67% in 1988–1991; severity (measured as the mean number of decayed, missing, or filled teeth) declined from 6.2 to 2.8 during this period [35-38].

These decreases in caries prevalence and severity have been uneven across the general population; the burden of disease now is concentrated among certain groups and persons. For example, 80% of the dental caries in permanent teeth of U.S. children aged 5–17 years occurs among 25% of those children. To develop and apply appropriate and effective caries prevention and control strategies, identification and assessment of groups and persons at high risk for developing new carious lesions is essential [39]. Caries risk assessment is difficult because it attempts to account for the complex interaction of multiple factors. Although various methods for assessing risk exist, no single model predominates in this emerging science. Models that take multiple factors into account predict the risk more accurately, especially for groups rather than persons. However, for persons in a clinical setting, models do not improve on a dentist's perception of risk after examining a patient and considering the personal circumstances [40]. Children and adults who are at low risk for dental caries can maintain that status through frequent exposure to small amounts of fluoride (e.g., drinking fluoridated water and using fluoride toothpaste). Children and adults at high risk for dental caries might benefit from additional exposure to fluoride (e.g., mouthrinse, dietary supplements, and professionally applied products). All available information on risk factors should be considered before a group or person is identified as being at low or high risk for dental caries. However, when classification is uncertain, treating a person as high risk is prudent until further information or experience allows a more accurate assessment [41].

### **RISK FOR ENAMEL FLUOROSIS**

The proper amount of fluoride helps prevent and control dental caries. Fluoride ingested during tooth development can also result in a range of visually detectable changes in enamel opacity (i.e., light refraction at or below the surface) because of hypomineralization. These changes have been broadly termed enamel fluorosis, certain extremes of which are cosmetically objectionable [42]. (Many other developmental changes that affect the appearance of enamel are not related to fluoride [43]. Severe forms of this condition can occur only when young children ingest excess fluoride, from any source, during critical periods of tooth development. The occurrence of enamel fluorosis is reported to be most strongly associated with cumulative fluoride intake during

enamel development, but the severity of the condition depends on the dose, duration, and timing of fluoride intake. The transition and early maturation stages of enamel development appear to be most susceptible to the effects of fluoride [44]. These stages occur at varying times for different tooth types. For central incisors of the upper jaw, for example, the most sensitive period is estimated at age 15–24 months for boys and age 21–30 months for girls [45,46]. The very mild and mild forms of enamel fluorosis appear as chalklike, lacy markings across a tooth's enamel surface that are not readily apparent to the affected person or casual observer [47,48]. In the moderate form, >50% of the enamel surface is opaque white. The rare, severe form manifests as pitted and brittle enamel. After eruption, teeth with moderate or severe fluorosis might develop areas of brown stain [49]. In the severe form, the compromised enamel might break away, resulting in excessive wear of the teeth. Even in its severe form, enamel fluorosis is considered a cosmetic effect, not an adverse functional effect [50,51]. Some persons choose to modify this condition with elective cosmetic treatment.

#### Fluoride Toothpaste

Fluoride is the only nonprescription toothpaste additive proven to prevent dental caries. When introduced into the mouth, fluoride in toothpaste is taken up directly by dental plaque [52–54] and demineralized enamel [55,56]. Brushing with fluoride toothpaste also increases the fluoride concentration in saliva 100- to 1,000-fold; this concentration returns to baseline levels within 1–2 hours [57]. Some of this salivary fluoride is taken up by dental plaque. The ambient fluoride concentration in saliva and plaque can increase during regular use of fluoride toothpaste. Studies of 2–3 years duration have reported that fluoride toothpaste reduces caries experience among children by a median of 15%–30% [58,59]. This reduction is modest compared with the effect of water fluoridation, but water fluoridation studies usually measured lifetime — rather than a few years' — exposure. Regular lifetime use of fluoride toothpaste likely provides ongoing benefits that might approach those of fluoridated water. Combined use of fluoride toothpaste and fluoridated water offers protection above either used alone [60]. Children who begin using fluoride toothpaste at age <2 years are at higher risk for enamel fluorosis than children who begin later or who do not use fluoride toothpaste at all [61,62]. Because studies have not used the same criteria for age of initiation, amount of toothpaste used, or frequency of toothpaste use, the specific contribution of each factor to enamel fluorosis among this age group has not been established.

Fluoride toothpaste contributes to the risk for enamel fluorosis because the swallowing reflex of children aged <6 years is not always well controlled, particularly among children aged <3 years. Children are also known to swallow toothpaste deliberately when they like its taste. A child-sized toothbrush covered with a full strip of toothpaste holds approximately 0.75–1.0 g of toothpaste, and each gram of fluoride toothpaste, as formulated in the United States, contains approximately 1.0 mg of fluoride. Children aged <6 years swallow a mean of 0.3 g of toothpaste per brushing and can inadvertently swallow as much as 0.8 g. As a result, multiple brushings with fluoride toothpaste each day can result in ingestion of excess fluoride. For this reason, high-fluoride toothpaste (i.e., containing 1,500 ppm fluoride) is generally contraindicated for children aged <6 years.

#### **CONCLUSION:**

From this study we conclude that, fluoride is a safe and effective agent that can be used to prevent and control dental caries. Using higher concentration of fluoride in toothpaste helps in the prevention of dental caries than low concentration.

#### **REFERENCES:**

- [1] Bratthall D, Hänsel Petersson G, Sundberg H. Reasons for the caries decline: what do the experts believe? *Eur J Oral Sci* 1996;104:416–22.
- [2] Blaney JR, Tucker WH. The Evanston Dental Caries Study. II. Purpose and mechanism of the study. *J Dent Res* 1948;27:279–86.
- [3] Ast DB, Finn SB, McCaffrey I. The Newburgh-Kingston Caries Fluorine Study. I. Dental findings after three years of water fluoridation. *Am J Public Health* 1950;40:716–24.
- [4] Dean HT, Arnold FA, Jay P, Knutson JW. Studies on mass control of dental caries through fluoridation of the public water supply. *Public Health Rep* 1950;65:1403–8.
- [5] Hutton WL, Linscott BW, Williams DB. The Brantford fluorine experiment: interim report after five years of water fluoridation. *Can J Public Health* 1951;42:81–7.
- [6] Pao EM. Changes in American food consumption patterns and their nutritional significance. *Food Technol* 1981;35:43–53.
- [7] panelDrRobert H Selwitz DDS<sup>ab</sup> Amid I Ismail DrPH<sup>c</sup> Nigel BPitts BDS<sup>d</sup>, Dental caries, vol 365, issue 9555, January 2007.
- [8] Featherstone JDB. Prevention and reversal of dental caries: role of low level fluoride. *Community Dent Oral Epidemiol* 1999;27:31–40.
- [9] Koulourides T. Summary of session II: fluoride and the caries process. *J Dent Res* 1990;69(special issue):558.
- [10] Tatevossian A. Fluoride in dental plaque and its effects. *J Dent Res* 1990;69(special issue):645–52.
- [11] Chow LC. Tooth-bound fluoride and dental caries. *J Dent Res* 1990;69(special issue):595–600.
- [12] Ericsson SY. Cariostasis mechanisms of fluorides: clinical observations. *Caries Res* 1977;11(suppl 1):2–23.
- [13] Kidd EAM, Thylstrup A, Fejerskov O, Bruun C. Influence of fluoride in surface enamel and degree of dental fluorosis on caries development in vitro. *Caries Res* 1980;14:196–202.
- [14] Thylstrup A. Clinical evidence of the role of pre-eruptive fluoride in caries prevention. *J Dent Res* 1990;69(special issue):742–50.
- [15] Thylstrup A, Fejerskov O, Bruun C, Kann J. Enamel changes and dental caries in 7-year-old children given fluoride tablets from shortly after birth. *Caries Res* 1979;13:265–76.



- [16] Bowden GHW. Effects of fluoride on the microbial ecology of dental plaque. *J Dent Res* 1990;69(special issue):653–9.
- [17] Bowden GHW, Odlum O, Nolette N, Hamilton IR. Microbial populations growing in the presence of fluoride at low pH isolated from dental plaque of children living in an area with fluoridated water. *Infect Immun* 1982;36:247–54.
- [18] Marquis RE. Diminished acid tolerance of plaque bacteria caused by fluoride. *J Dent Res* 1990;69(special issue):672–5.
- [19] Rosen S, Frea JI, Hsu SM. Effect of fluoride-resistant microorganisms on dental caries. *J Dent Res* 1978;57:180.
- [20] Van Loveren C. The antimicrobial action of fluoride and its role in caries inhibition. *J Dent Res* 1990;69(special issue):676–81.
- [21] Oliveby A, Twetman S, Ekstrand J. Diurnal fluoride concentration in whole saliva in children living in a high- and a low-fluoride area. *Caries Res* 1990;24:44–7.
- [22] Rølla G, Ekstrand J. Fluoride in oral fluids and dental plaque. In: Fejerskov O, Ekstrand J, Burt BA, eds. *Fluoride in dentistry*. 2nd ed. Copenhagen: Munksgaard, 1996:215–29.
- [23] LeGeros RZ. Chemical and crystallographic events in the caries process. *J Dent Res* 1990;69(special issue):567–74.
- [24] Dean HT, Dixon RM, Cohen C. Mottled enamel in Texas. *Public Health Rep* 1935;50:424–42.
- [25] McClure FJ, Likins RC. Fluorine in human teeth studied in relation to fluorine in the drinking water. *J Dent Res* 1951;30:172–6.
- [26] Marthaler TM. Fluoride supplements for systemic effects in caries prevention. In: Johansen E, Taves DR, Olsen TO, eds. *Continuing evaluation of the use of fluorides*. Boulder, CO: Westview, 1979:33–59. (American Association for the Advancement of Science selected symposium no. 11).
- [27] Murray JJ. Efficacy of preventive agents for dental caries. Systemic fluorides: water fluoridation. *Caries Res* 1993;27(suppl 1):2–8.
- [28] Groeneveld A, Van Eck AAMJ, Backer Dirks O. Fluoride in caries prevention: is the effect pre- or post-eruptive? *J Dent Res* 1990;69(special issue):751–5.
- [29] Levine RS. The action of fluoride in caries prevention: a review of current concepts. *Br Dent J* 1976;140:9–14.
- [30] Margolis HC, Moreno EC. Physicochemical perspectives on the cariostatic mechanisms of systemic and topical fluorides. *J Dent Res* 1990;69(special issue):606–13.
- [31] Clarkson BH, Fejerskov O, Ekstrand J, Burt BA. Rational use of fluorides in caries control. In: Fejerskov O, Ekstrand J, Burt BA, eds. *Fluorides in dentistry*. 2nd ed. Copenhagen: Munksgaard, 1996:347–57.
- [32] Arends J, Christoffersen J. Nature and role of loosely bound fluoride in dental caries. *J Dent Res* 1990;69(special issue):601–5.
- [33] Burt BA, Eklund SA. *Dentistry, dental practice, and the community*. 5th ed. Philadelphia, PA: W.B. Saunders, 1999.
- [34] National Institute of Dental Research. The prevalence of dental caries in United States children, 1979–1980. Bethesda, MD: U.S. Public Health Service, Department of Health and Human Services, National Institutes of Health, 1981; NIH publication no. 82-2245.
- [35] Kelly JE, Harvey CR. Basic dental examination findings of persons 1–74 years. In: *Basic data on dental examination findings of persons 1–74 years, United States, 1971–1974*. Hyattsville, MD: US Department of Health, Education, and Welfare, Public Health Service, Office of Health Research, Statistics, and Technology, National Center for Health Statistics, 1979; DHEW publication no. (PHS) 79-1662. (Vital and health statistics data from the National Health Interview Survey; series 11, no. 214).
- [36] National Institute of Dental Research. Oral health of United States children. The National Survey of Dental Caries in U.S. School Children: 1986–1987. National and regional findings. Bethesda, MD: US Department of Health and Human Services, Public Health Service, National Institutes of Health, National Institute of Dental Research, 1989; NIH publication no. 89-2247.
- [37] Kaste LM, Selwitz RH, Oldakowski RJ, Brunelle JA, Winn DM, Brown LJ. Coronal caries in the primary and permanent dentition of children and adolescents 1–17 years of age: United States, 1988–1991. *J Dent Res* 1996;75(special issue):631–41.
- [38] Meskin LH, ed. *Caries diagnosis and risk assessment: a review of preventive strategies and management*. *J Am Dent Assoc* 1995;126(suppl):15–245.
- [39] Pitts NB. Risk assessment and caries prediction. *J Dent Educ* 1998;62:762–70.
- [40] Recommendations for Using Fluoride to Prevent and Control Dental Caries in the United States, August 17, 2001 / Vol. 50 / No. RR-14
- [41] Fejerskov O, Manji F, Baelum V. The nature and mechanisms of dental fluorosis in man. *J Dent Res* 1990;69(special issue):692–700.
- [42] Avery JK. Agents affecting tooth and bone development. In: Avery JK, ed. *Oral development and histology*. 2nd ed. New York, NY: Theime Medical Publishers, 1994:130–41.
- [43] DenBesten PK, Thariani H. Biological mechanisms of fluorosis and level and timing of systemic exposure to fluoride with respect to fluorosis. *J Dent Res* 1992;71:1238–43.
- [44] Evans RW, Stamm JW. Dental fluorosis following downward adjustment of fluoride in drinking water. *J Public Health Dent* 1991;51:91–8.
- [45] Dean HT. The investigation of physiological effects by the epidemiological method. In:
- [46] Moulton FR, ed. *Fluorine and dental health*. Washington, DC: American Association for the Advancement of Science, 1942;19:23–31.
- [47] Fejerskov O, Manji F, Baelum V, Møller IJ. *Dental fluorosis—a handbook for health workers*. Copenhagen: Munksgaard, 1988.

- [48] Kaminsky LS, Mahoney MC, Leach J, Melius J, Miller MJ. Fluoride: benefits and risks of exposure. *Crit Rev Oral Biol Med* 1990;1:261–81.
- [49] Clark DC, Hann HJ, Williamson MF, Berkowitz J. Aesthetic concerns of children and parents in relation to different classifications of the Tooth Surface Index of Fluorosis. *Community Dent Oral Epidemiol* 1993;21:360–4.
- [51] Duckworth RM, Morgan SN, Burchell CK. Fluoride in plaque following use of dentifrices containing sodium monofluorophosphate. *J Dent Res* 1989;68:130–3.
- [52] Duckworth RM, Morgan SN. Oral fluoride retention after use of fluoride dentifrices. *Caries Res* 1991;25:123–9.
- [53] Sidi AD. Effect of brushing with fluoride toothpastes on the fluoride, calcium, and inorganic phosphorus concentrations in approximal plaque of young adults. *Caries Res* 1989;23:268–71.
- [54] Reintsema H, Schuthof J, Arends J. An in vivo investigation of the fluoride uptake in partially demineralized human enamel from several different dentifrices. *J Dent Res* 1985;64:19–23.
- [55] Stookey GK, Schemehorn BR, Cheetham BL, Wood GD, Walton GV. In situ fluoride uptake from fluoride dentifrices by carious enamel. *J Dent Res* 1985;64:900–3.
- [56] Bruun C, Givskov H, Thylstrup A. Whole saliva fluoride after toothbrushing with NaF and MFP dentifrices with different F concentrations. *Caries Res* 1984;18:282–8.
- [57] Horowitz HS, Law FE, Thompson MB, Chamberlin SR. Evaluation of a stannous fluoride dentifrice for use in dental public health programs. I. Basic findings. *J Am Dent Assoc* 1966;72:408–22.
- [58] James PMC, Anderson RJ. Clinical testing of a stannous fluoride-calcium pyrophosphate dentifrice in Buckinghamshire school children. *Br Dent J* 1967;123:33–9.
- [59] O'Mullane DM, Clarkson J, Holland T, O'Hickey S, Whelton H. Effectiveness of water fluoridation in the prevention of dental caries in Irish children. *Community Dent Health* 1988;5:331–44.
- [60] Lalumandier JA, Rozier RG. The prevalence and risk factors of fluorosis among patients in a pediatric dental practice. *Pediatr Dent* 1995;17:19–25.
- [61] Pendrys DG, Katz RV, Morse DE. Risk factors for enamel fluorosis in a nonfluoridated population. *Am J Epidemiol* 1996;143:808–15.
- [62] Levy SM. A review of fluoride intake from fluoride dentifrice. *J Dent Child* 1993;61:115–24