CODEN [USA]: IAJPBB ISSN : 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES

SJIF Impact Factor: 7.187
https://doi.org/10.5281/zenodo.7562200

Available online at: http://www.iajps.com *Research Article*

EFFECTIVENESS OF FLUORIDE ON CHILDREN ACIDIC PLAQUE

[1]Ibrahim Salem Thaffed, [2]Hamed Manea Abdullah Alshamrani, [3]Abdullah Shalan Saeed Alqarni, [4]Ahmed Mohammed Alghamdi, [5]Majed Nawar Maudh Alharthi, [6]Faris Ahmed Saad Alkhathami, [7]Ali Mohammad Alzahrani, [8]Tareq Saleh Alzahrani, [9]Moadh Abdulhameed Alghamdi

Article Received: October 2022 **Accepted:** November 2022 **Published:** December 2022

Abstract:

Dietary sugars, dental biofilm, and the host interact simultaneously within the framework of the oral environment to cause the multifactorial illness dental caries. Comprehensive literature search utilizing the electronic databases PubMed and Embase for relevant English-language publications published up until the middle of 2022. Fluoride treatments applied directly to dental plaque lowered acid production. In addition, the dissolution of fluoride from enamel treated topically decreased the acid production of oral bacteria in vitro. The benefits are transient and may not be relevant to caries prevention in vivo. Therefore, daily fluoride applications can reduce the acidogenicity of tooth plaque even 8 to 12 hours after treatment. This decrease will likely aid to caries prevention.

Corresponding author:
Ibrahim Salem Thaffed,

QR code

Please cite this article in press Ibrahim Salem Thaffed et al, ***Effectiveness Of Fluoride On Children Acidic Plaque.,*** *Indo Am. J. P. Sci, 2022; 09(12).*

INTRODUCTION:

Early childhood caries (ECC) is the occurrence of decaying, missing, or repaired primary teeth in children under the age of six. ECC is one of the most prevalent chronic childhood disorders worldwide, affecting up to 73% of children from disadvantaged socioeconomic backgrounds [1]. The treatment of young children has proven difficult due to the need for advanced clinical skills and expensive general anesthesia for patient management [2]. A carious lesion that is left untreated can grow, produce discomfort, and become a tooth abscess, resulting in a severe infection [3]. ECC treatments differ from adult treatments in that an atraumatic approach for children can halt the advancement of caries, allowing the stalled rotten tooth to exfoliate prior to producing oral pain [4]. Silver diamine fluoride (SDF) is a topical fluoride solution for age-related dental cavities in men. Clinical studies [2,5] indicate that 38% SDF reduces and arrests coronal caries in preschoolers. According to a systematic evaluation, 81% of active caries lesions in ECC were halted following SDF treatment [6]. Diverse mechanisms of action have been postulated to explain the promising caries-preventing effectiveness of SDF. Some researchers hypothesized that SDF hardens a caries lesion by recovering calcium and phosphate from saliva, and that it inhibits collagenases such dental MMPs and cathepsins to prevent dentine collagen breakdown [7].

Fluorides serve an important function in preventing and controlling tooth cavities. In the middle of the last century, it was widely believed that fluoride had to be absorbed into dental enamel during its formation in order to exhibit its full protective impact. In order to reduce the incidence and severity of caries in youngsters, it was deemed necessary to have a certain prevalence and severity of fluorosis in a community. In the 1980s, a paradigm change was proposed [8] regarding the cariostatic processes of fluorides. This is because the major, if not sole, explanation for how fluorides limit caries development is their topical effect on the de- and re-mineralization processes that occur between the tooth surface and the nearby dental biofilm. This idea gained widespread acceptance [9,10] and made it possible to provide substantial caries prevention without significant fluoride ingestion. With this in mind and being aware of the growth in the prevalence of dental fluorosis in both fluoridated and non-fluoridated locations, researchers from around the world have focused on regulating fluoride consumption [11]. In addition, SDF has demonstrated potent antibacterial effects on particular cariogenic biofilms inside mono-cariogenic species biofilm [10] and multi-cariogenic species biofilm [11,12]. However, these investigations were conducted on in vitro biofilm models, which are in no way representative of the complex microbial environment of the oral cavity. In comparison, the biofilm in the human mouth consists of at least 700 species belonging to 30 different genera [13].

DISCUSSION:

After observing the short-term (2 weeks) and intermediate (12 weeks) effects on microbiota alterations, Chu et al. [14] reported effective caries arrest following three administrations of SDF at 2-week intervals. One drawback was that long-term samples were not collected since the kindergarten III children graduated shortly after the three-month follow-up. In addition, 16S rRNA gene amplicon sequencing, an NGS application, gives only information regarding relative taxon-specific gene abundances. However, relative gene abundances are frequently utilized to analyze the link between the makeup of microbial communities and environmental conditions [15].

The fluoridation of public drinking water is regarded as one of the ten greatest public health achievements of the 20th century [16]. Despite the availability of other fluoride- containing products, water fluoridation remains the most egalitarian and cost- effective method of distributing fluoride to all members of most communities, regardless of age, income, or educational achievement. In addition, some data suggests that water fluoridation may lessen the disparity in dental health between social strata [16]. The average projected annual cost of water fluoridation per person in the United States is only about $0.72 [17]. Early in the 1940s, it was recognized that approximately 10% of children in locations naturally fluoridated at optimal levels (1.0 ppm) had mild or very mild fluorosis of the permanent teeth, compared to less than 1% in low-fluoride areas [18].

Included in a 2000 comprehensive analysis of 214 papers on water fluoridation were 88 studies on dental fluorosis [19]. The scientists discovered a dose-response relationship between the fluoride concentration in drinking water and the incidence of dental fluorosis. At a fluoride level of 1 ppm in the drinking water, it was estimated that the prevalence of any dental fluorosis was 48%, and that 12.5% of exposed adults had dental fluorosis that caused them aesthetic concern (moderate to severe). This is significantly higher than the number reported by Dean et al. [18] in 1942, who discovered essentially no cases of moderate or severe fluorosis, but the data cannot be directly compared due to the use of differing case

definitions. When examining the effect of fluoridated water on the prevalence of dental fluorosis in a community, research that utilized breaks in water fluoridation to measure dental fluorosis in distinct birth cohorts are of particular relevance. Thus, Burt et al. [20] studied the impact of an unforeseen 11-month interruption in water fluoridation and concluded that changes in fluoride exposure from drinking water influence the prevalence of dental fluorosis. However, in a later study [49], the prevalence of dental fluorosis remained steady, despite a predicted increase in the following cohort due to the reinstatement of water fluoridation. Buzalaf et al. [21] examined the impact of a seven-year break in water fluoridation on the prevalence of dental fluorosis in a Brazilian city. The prevalence of dental fluorosis was observed to be lower in the permanent maxillary central incisors of children who were 36, 27, and 18 months old when water fluoridation was discontinued, compared to those born 18 months after fluoridation was discontinued.

A recent study evaluated the total daily fluoride intake of 1- to 3-year-olds living in an appropriately fluoridated environment from various dietary components and toothpaste. Dentifrice containing a standard concentration of fluoride accounted for an average of 81.5% of the daily fluoride intake, whereas water and reconstituted milk accounted for approximately 60% of the total contribution of the diet [22]. For 4- to 6-year-olds living in the same community, however, the influence of fluoride absorbed from toothpaste was less significant, and water alone delivered an average of 34% of the predicted daily fluoride intake from the diet, which amounts to around 0.014 mg/kg [23,24].

Two studies have explored the changes in microbiota following SDF therapy. Milgrom et al. obtained plaque samples from three children before and 14 days after application of 38% SDF in a clinical experiment. RNA sequencing revealed that there are no consistent changes in the relative abundance or diversity of microorganisms associated with dental caries [25]. Mitwalli et al. examined the impact of 38% SDF on the microbiota profile of root caries plaque [26].

Studies in the laboratory have demonstrated that strains of this species can create lactic acid, which causes dental caries. Streptococcus sobrinus is likewise a lactate-producing species, although its incidence is modest and it coexists with Streptococcus mutans [27]. Streptococcus sobrinus was not detected in either original caries lesions (T0) or arrested caries following SDF treatment in the same trial. This aligns with Becker et al [27] findings, Streptococcus sobrinus was surprisingly abundant in active caries following SDF therapy. Lactobacillus sp., which have been identified as secondary infections in deep carious lesions, exhibited a similar tendency. The ability of Lactobacilli to ferment a range of carbohydrates and live in a low-pH environment is the defining characteristic [28] of the caries paradigm. In cases of active caries, SDF therapy raised the number of Streptococcus mutans, Streptococcus sobrinus, and Lactobacillus sp., making the remainder of the treatment more difficult. Veillonella sp. is one of the most common organisms found in saliva and dental plaque in ECC [28]. Veillonella and Streptococcus mutans cause higher acid generation and demineralization than Streptococcus mutans alone, according to in vitro studies [29]. Veillonella transforms lactic acid generated by other organisms into propionic and acetic acids. The combination of acetic and lactic acids exerts demineralizing actions that are additive [30]. Additionally, by nitrate reduction, Veillonella spp. prevent acid-producing bacteria in caries. In acidic settings, a low nitrite concentration (0.2 mM) can eradicate Streptococcus mutans completely [31]. High quantities of Rothia sp. have been recovered from ECC saliva samples [32], but only a small number of writers have researched this species, and its mechanism remains unknown. Increases in acid-producing species may generate an acidic microenvironment that inhibits or eradicates the growth of acidophobic bacteria, hence reducing the variety of the microbiota. Nonetheless, the mechanisms of active caries following SDF treatment require additional exploration [32].

The first method incorporating the use of fluoride for caries management was the addition of fluoride to public water supplies at regulated levels. The good results of this measure led to the suggestion that pregnant women take fluoride supplements to prevent dental cavities in their progeny. Since the initial cariostatic benefits of fluoride were noticed when this element was ingested from "systemic" sources, from the 1940s to the 1970s it was believed that fluoride's cariostatic mechanism relied primarily on its uptake in the enamel-forming process. This would result in the production of fluorhydroxy apatite, a mineral phase that is more resistant to dissolution in the future. Ingestion of fluoride was seen unavoidable for this goal, and dental fluorosis was deemed a required danger in order to obtain the cariostatic effects of fluoride [28,32].

In the 1980s, it was proven [30] that fluoride controls caries lesion formation primarily by its topical action

on demineralization and remineralization processes occurring at the interface of the tooth surface and the oral fluids. Elegant in situ research undertaken in Scandinavia contributed significantly to the development of this theory. In one of the trials, the scientists placed human and shark enamel slabs in detachable orthodontic appliances and covered them with orthodontic bands to permit plaque accumulation. Because shark enamel is constituted of practically pure fluorapatite, it was utilized. Analyses using microradiography demonstrated that carious lesions developed on both substrates, but they were less severe in shark enamel. When daily mouth rinse with 0.2% NaF was employed on human enamel, the investigators compared these results to those from earlier investigations. They noticed that the mineral loss in human enamel treated with a fluoride rinse was less than that of shark enamel untreated [32].

Fluoride exerts its effects on oral bacteria by inhibiting cellular enzymes directly (directly or in combination with metals) or by increasing proton permeability of cell membranes in the form of hydrogen fluoride (HF) [30, 32]. According to the reaction $H^+ + F^-$ HF, HF is generated more easily under acidic conditions (pKa = 3.15), and it is more permeable to bacterial cell membranes, allowing it to enter the cell. In the cytoplasm, which is more alkaline than the outer environment [31], HF dissociates into H^+ and F^-. This intracellular F^- inhibits glycolytic enzymes, resulting in a reduction in glycolysis-derived acid generation. F^- in the cytoplasm also decreases cytoplasmatic pH (which affects total glycolytic activity), influencing both acid generation and acid- tolerance in S. mutans [32]. F^- also inhibits cell membrane-associated H^+-ATPases due to the fact that expelled protons are transported back into the cell, hence reducing H^+ excretion from the cell.

CONCLUSION:

Fluoride primarily functions through topical mechanisms, which include the inhibition of demineralization at the crystal surfaces within the tooth, the enhancement of remineralization at the crystal surfaces (the resulting demineralized layer is highly resistant to acid attack), and the inhibition of bacterial enzymes. Understanding the mechanisms by which fluoride promotes caries management is crucial for maximizing the advantages of this element while minimizing negative effects. Fluoride mostly prevents dental cavities through its topical impact. Fluoride present in low, sustained amounts (sub-ppm range) in oral fluids during an acidic challenge is capable of absorbing into the surface of apatite crystals, so limiting demineralization. When the pH is restored, trace amounts of fluoride in the solution will render it highly supersaturated with regard to fluorhydroxyapatite, hence accelerating the remineralization process. The mineral generated as a result of the nucleating action of the partially dissolved minerals will then preferentially contain fluoride and reject carbonate, increasing the enamel's resistance to future acidic challenges. Topical fluoride can also offer antibacterial activity. In vitro, fluoride concentrations as observed in dental plaque have a biological effect on key virulence components of S. mutans, such as acid generation and glucan synthesis, although the in vivo ramifications of this are not yet known.

REFERENCES:

1. J. Xiao, N. Alkhers, D.T. Kopycka-Kedzierawski, R.J. Billings, T.T. Wu, D. A. Castillo, L. Rasubala, H. Malmstrom, Y. Ren, E. Eliav, Prenatal oral health care and early childhood caries prevention: a systematic review and meta-analysis, Caries Res. 53 (4) (2019) 411–421.
2. C.H. Chu, E.C. Lo, Promoting caries arrest in children with silver diamine fluoride: a review, Oral Health Prev. Dent. 6 (4) (2008) 315–321.
3. P.S. Casamassimo, S. Thikkurissy, B.L. Edelstein, E. Maiorini, Beyond the dmft: the human and economic cost of early childhood caries, J. Am. Dent. Assoc. 140 (6) (2009) 650–657.
4. D. Duangthip, K.J. Chen, S.S. Gao, E.C.M. Lo, C.H. Chu, Managing early childhood caries with atraumatic restorative treatment and topical silver and fluoride agents, Int. J. Environ. Res. Public Health 14 (10) (2017).
5. M.H.T. Fung, D. Duangthip, M.C.M. Wong, E.C.M. Lo, C.H. Chu, Arresting dentine caries with different concentration and periodicity of silver diamine fluoride, JDR Clin. Trans. Res. 1 (2) (2016) 143–152.
6. S.S. Gao, I.S. Zhao, N. Hiraishi, D. Duangthip, M.L. Mei, E.C.M. Lo, C.H. Chu, Clinical trials of silver diamine fluoride in arresting caries among children: a systematic review, JDR Clin. Trans. Res. 1 (3) (2016) 201–210.
7. M.L. Mei, L. Ito, Y. Cao, E.C. Lo, Q.L. Li, C.H. Chu, An ex vivo study of arrested primary teeth caries with silver diamine fluoride therapy, J. Dent. 42 (4) (2014) 395–402.
8. Fejerskov O, Thylstrup A, Larsen MJ: Rational use of fluorides in caries prevention: a concept based on possible cariostatic mechanisms. Acta Odontol Scand 1981;39:241– 249.

9. ten Cate JM, Duijsters PP: Influence of fluoride in solution on tooth demineralization. I. Chemical data. Caries Res 1983;17:193– 199.
10. Khan A, Moola MH, Cleaton- Jones P: Global trends in dental fluorosis from 1980 to 2000: a systematic review. SADJ 2005;60:418– 421.
11. Whelton HP, Ketley CE, McSweeney F, O'Mullane DM: A review of fluorosis in the European Union: prevalence, risk factors and aesthetic issues. Community Dent Oral Epidemiol 2004;32(suppl 1):9– 18.
12. C.H. Chu, L. Mei, C.J. Seneviratne, E.C. Lo, Effects of silver diamine fluoride on dentine carious lesions induced by Streptococcus mutans and Actinomyces naeslundii biofilms, Int. J. Paediatr. Dent. 22 (1) (2012) 2–10.
13. M.L. Mei, C.H. Chu, K.H. Low, C.M. Che, E.C. Lo, Caries arresting effect of silver diamine fluoride on dentine carious lesion with S. mutans and L. acidophilus dualspecies cariogenic biofilm, Med. Oral Patol. Oral Cir. Bucal 18 (6) (2013) e824–31.
14. M.L. Mei, Q.L. Li, C.H. Chu, E.C. Lo, L.P. Samaranayake, Antibacterial effects of silver diamine fluoride on multi-species cariogenic biofilm on caries, Ann. Clin. Microbiol. Antimicrob. 12 (2013) 4.
15. R.J. Palmer Jr., Composition and development of oral bacterial communities, Periodontology 2000 64 (1) (2014) 20–39.
16. CDC: Achievements in public health. 1900– 1999: fluoridation of drinking water to prevent dental caries. Morb Mort Wkly Rep 1999;48:933– 940.
17. Jones S, Burt BA, Petersen PE, Lennon MA: The effective use of fluoride in public health. Bull WHO 2005;83:670– 676.
18. Dean HT, Arnold FA, Elvolve E: Additional studies of the relation of fluoride domestic waters to dental caries experience in 4,425 white children aged 12– 14 years in 13 cities in 4 states. Public Health Rep 1942;57:1155– 1179.
19. McDonagh MS, Whiting PF, Wilson PM, Sutton AJ, Chestnutt I, Cooper J, Misso K, Bradley M, Treasure E, Kleijnen J: Systematic review of water fluoridation. BMJ 2000;321:855– 859.
20. Burt BA, Keels MA, Heller KE: The effects of a break in water fluoridation on the development of dental caries and fluorosis. J Dent Res 2000;79:761– 769.
21. Buzalaf MA, de Almeida BS, Olympio KP, da SCVE, de CSPSH: Enamel fluorosis prevalence after a 7- year interruption in water fluoridation in Jaú, Sao Paulo, Brazil. J Public Health Dent 2004;64:205– 208.
22. de Almeida BS, da Silva Cardoso VE, Buzalaf MA: Fluoride ingestion from toothpaste and diet in 1- to 3- year- old Brazilian children. Community Dent Oral Epidemiol 2007;35:53– 63.
23. Rodrigues MH, Leite AL, Arana A, Villena RS, Forte FD, Sampaio FC, Buzalaf MA: Dietary fluoride intake by children receiving different sources of systemic fluoride. J Dent Res 2009;88:142– 145.
24. Buzalaf MAR, Rodrigues MHC, Pessan JP, Leite AL, Arana A, Villena RS, Forte FD, Sampaio FC: Biomarkers of fluoride in children exposed to different sources of systemic fluoride. J Dent Res 2011;90:215– 219.
25. P. Milgrom, J.A. Horst, S. Ludwig, M. Rothen, B.W. Chaffee, S. Lyalina, K. S. Pollard, J.L. DeRisi, L. Mancl, Topical silver diamine fluoride for dental caries arrest in preschool children: a randomized controlled trial and microbiological analysis of caries associated microbes and resistance gene expression, J. Dent. 68 (2018) 72– 78.
26. H. Mitwalli, M.D.A. Mourao, J. Dennison, P. Yaman, B.J. Paster, M. Fontana, Effect of silver diamine fluoride treatment on microbial profiles of plaque biofilms from root/cervical caries lesions, Caries Res. 53 (5) (2019) 555–566.
27. M.R. Becker, B.J. Paster, E.J. Leys, M.L. Moeschberger, S.G. Kenyon, J.L. Galvin, S. K. Boches, F.E. Dewhirst, A.L. Griffen, Molecular analysis of bacterial species associated with childhood caries, J. Clin. Microbiol. 40 (3) (2002) 1001–1009.
28. P.W. Caufield, C.N. Schon, P. Saraithong, Y. Li, S. Argimon, Oral Lactobacilli and Dental Caries: A Model for Niche Adaptation in Humans, J. Dent. Res. 94 (Suppl. 9) (2015) 110S–118S.
29. E. Kanasi, F.E. Dewhirst, N.I. Chalmers, R. Kent Jr., A. Moore, C.V. Hughes, N. Pradhan, C.Y. Loo, A.C. Tanner, Clonal analysis of the microbiota of severe early childhood caries, Caries Res. 44 (5) (2010) 485–497.
30. W.D. Noorda, D.J. Purdell-Lewis, A.M. van Montfort, A.H. Weerkamp, Monobacterial and mixed bacterial plaques of Streptococcus mutans and Veillonella alcalescens in an artificial mouth: development, metabolism, and effect on human dental enamel, Caries Res. 22 (6) (1988) 342–347.
31. J.D. Featherstone, B.E. Rodgers, Effect of acetic, lactic and other organic acids on the formation of artificial carious lesions, Caries Res. 15 (5) (1981) 377–385.
32. L.S. Silva Mendez, R.P. Allaker, J.M. Hardie, N. Benjamin, Antimicrobial effect of acidified nitrite

on cariogenic bacteria, Oral Microbiol. Immunol.
14 (6) (1999) 391–392.