Curr Pediatr Res 2016; 20 (1&2): 258-261 *ISSN 0971-9032*
www.currentpediatrics.com

Secondhand smoke: A man-made disaster to oral health of children!

Anil Patil

Associate Professor, Pedodontics and Preventive Dentistry, Bharati Vidyapeeth Deemed University Dental College and Hospital, India.

Abstract

Smoking affects almost all body systems, leading to many diseases and ill health of smokers and non-smokers through secondhand smoke (SHS). Few studies proposed an association between SHS exposure and pediatric dental diseases, like dental caries and poorer gingival attachment of teeth and supporting structures. But by so far, an underlying association between SHS and oral health of children has not been completely assessed. Hence, this review will emphasize susceptibility of children to SHS effects, mechanism of action of SHS on oral health, effect of SHS on oral health, and prevention of SHS exposure to children.

Keywords: Dental caries; Oral health; Passive smoking; Secondhand smoke.

Accepted September 29, 2016

Introduction

Oral health is not only intricately related to general health, but also dental caries and gingival diseases may be lead to serious infections and countless grave disorders, for instance diabetes and respiratory diseases. The worldwide prevalence of untreated caries was the highest, with no decreasing trends and very high ranking in global burden [1]. Poor oral health can affect chewing ability, digestion, nutrition, look, confidence and sleeping disorders, in addition to behavioral and developmental problems in children.

Globally, smoking is the foremost reason of preventable fatality. With this progressive manner, smoking will kill millions worldwide and affect billions by direct or indirect way towards end of this century. Smoking affects almost all body systems, leading to many diseases and ill health of smokers and non-smokers through Secondhand Smoke (SHS) [2]. Passive smoking is recognized as involuntary smoking, second hand smoking, exposure to environmental tobacco smoke (ETS), is defined as inhalation of the cigarette smoke of another individual or the exhale of a smoker [3]. Secondhand smoke is mixture of smoke from cigarette's burning end and exhaled smoke. Secondhand smoke has more than 7,000 chemicals out of which hundreds are toxic and may produce cancer [4]. But by so far, an underlying association between SHS and oral health of children has not been thoroughly assessed. Hence, this review will emphasize susceptibility of children to SHS effects, mechanism of action of SHS on oral health, effect of SHS on oral health, and prevention of SHS exposure to children.

Susceptibility of Children to SHS Effects

There is no harmless level of secondhand smoke exposure. Children are comparatively more vulnerable to secondhand smoke effects because of higher breathing rates per body weight, immature lungs and more lung surface area compared with adults [5]. Furthermore, infants and children are generally not capable to manage their environment, consequently unable to perform action to escape from SHS exposure [5]. Children are most probably exposed to SHS at home, since exposure may be so extensive, yet moderately small increase in disease risk might cause a significant disease burden in infancy and childhood [6].

Infant health may be affected by various means of tobacco smoke exposure such as active maternal smoking or maternal exposure to SHS during pregnancy [5,7]; infants exposure to parental SHS at home, third-hand smoke exposure in household dust and interior surfaces, and increased bacterial load exposure of a smoking parent or caretaker [7-11]. Few studies proposed an association between SHS exposure and dental caries and poorer gingival attachment of teeth and supporting structures in children [5,12,13].

Mechanism of Action and Effects of SHS on Oral Health

ETS comprises of more than 4000 chemical agents unfavorably disturbing the oral health [12]. Cotinine (a nicotine biomarker) level measurement is a suitable, reliable objective and quantitative screening tool for ETS exposure determination [12,14]. Passive smoking

alters the normal oral and nasopharyngeal flora leading to upper airway infection [15]. The cigarette smoke products produce edema and inflammation by activity of pro-inflammatory agents and local vasoconstriction. Systemically, these products reduce saliva IgA, serum IgG levels and suppress T helper cells activity in host immunity responses [14]. These mechanisms and oxidative stress may lead to periodontal diseases, alveolar bone density reduction and ultimately tooth loss. Besides, cigarette smoke produces carbon monoxide and cyanides which delays wound healing moreover nicotine content restrain cell proliferation, osteoblastic activity and stimulate alkaline phosphatase activity. It unfavorably affects fibroblast activity and reduces their fibronectin and collagen production [3]. Passive smoking produces vascular destruction, endothelial inflammation, atherosclerosis, pH shift and cytokines production which eventually bring about implant failure [16].

Gingival pigmentation is increased by indirect stimulation of melanocytes by activity of polycyclic amines like nicotine and benzoperylene in cigarette smoke which go inside the blood circulation following inhalation [17]. Nicotine furthermore boosts proliferation of cariogenic bacteria like mutans streptococci in smoking mother's oral cavity of which gets transferred to their infants. Additionally, nicotine reduces vitamin C level, which is linked with the streptococcus mutans proliferation causing amplified caries risk. SHS exposure could predispose children to infections through immune system suppression or modulation such as lower salivary IgA levels and higher sialic acid levels with higher activity [18,19]. Sialic acid promotes streptococcus mutans agglutination, leading to dental plaque formation and caries [20]. Cariogenic bacterial colonization on rough tooth surfaces might be improved by SHS. Nicotine also reduces pH, flow and the buffering capacity of saliva whereas increase in lactobacilli count [21,22]. Passive smoking also impedes dental development through numerous mechanisms for instance interference with reciprocal induction of oral ectomesenchymal tissues, interference with tooth mineralization owing to oxidative stress and nutritional deficiency caused by unfavorable effect of SHS on appetite [19]. Enamel hypoplasia in primary and permanent dentition and caries in the primary dentition is linked to SHS exposure in children [23-28].

Children may inhale SHS through the oral cavity owing to nasal congestion, and breastfeeding from a smoker mother may cause toxic substances being transported directly to child's mouth [29,30]. Tobacco smoking was linked with increased levels of *S. mutans* and lactobacilli in saliva [31]. This may predispose to dental caries in children who live with smoking parents because of the early colonization of streptococcus mutans [32]. SHS exposed infants at 4 months old appear more probable to develop caries by 3 years of age [14,33]. SHS may cause unhealthy food choices, oral hygiene care and lifestyle leading to modulation of existing etiology of dental caries [34,35]. Cross sectional studies have proposed association of secondhand smoke with caries in deciduous and permanent teeth SHS is a risk factor for oro-facial clefts too [33,36-39].

An association between SHS exposure and an impact on cognition and behavior, higher likelihood of childhood conduct problems and learning difficulties is hypothesized by few studies [40]. A strong link has been seen between the incidence of respiratory complications in children undergoing general anesthesia and a history of SHS exposure [41-43]. Furthermore, children exposed to SHS have an unusual metabolic response to drugs administered for surgery [44].

Prevention of SHS Exposure to Children

The worldwide prevalence of SHS exposed individuals is projected to be 40% children and more than 30% non-smokers [45]. The monetary costs of smoking addiction and resultant morbidity and mortality are staggering. Increasing the knowledge of smokers about the oral and dental effects attributable to direct or indirect exposure to cigarette smoke can considerably persuade them to quit smoking [15].

Dentistry is transforming from caries management to caries prevention. Hence, most likely etiological risk factors or behaviors related with caries development and progression needs to be considered. Dental health professionals can promote tobacco cessation by counseling patients during the oral examination part of dental visits [14,46]. The American academy of pediatric dentistry advocates screening for tobacco use, tobacco dependence treatment, tobacco use prevention in children and adolescents and educating the public on mammoth health and societal costs of tobacco [47]. Global health organizations should focus on smoking issue to resolve the public health dilemma [3].

Smoking in home, closed public spaces or cars should not be allowed because smoke concentration increases and it will be considered as a normal behavior by child. Parents can assist to guard their children from SHS by implementing following measures [48]:

- Do not permit anybody to smoke in or near your home.
- Do not permit anyone to smoke in your car, even with the window open.
- Verify about children's day care centers and schools are tobacco-free.
- If regulation allows smoking in public areas, seek restaurants and other places that do not permit smoking.

Conclusion

Till now most of the studies on the association of SHS with early childhood caries demonstrated possible etiological alliance. This paper is lacking a critical review of the literature using a robust systematic or Cochrane review process which is the main weakness of article. Consequently, additional research must be accomplished

to SHS's realistic threat to oral health using a cohort design. Worldwide public oral and general health organizations should focus on providing SHS free environment for the infants and children.

References

1. Marcenes W, Kassebaum NJ, Bernabé E, et al. Global burden of oral conditions in 19902010: A systematic analysis. J Dent Res 2013; 92: 592597.
2. US. Department of Health and Human Services. A report of the surgeon general: How tobacco smoke causes disease: The biology and behavioral basis for smoking-attributable disease. Rockville, MD: U.S. Department of Health and Human Services, Office of the Surgeon General 2010.
3. Batran MM, Soliman NL, Mikael FF. Passive smoking and alveolar bone density. Aust J Basi Appl Sci 2009; 3: 713-9.
4. US Department of Health and Human Services. Let's make the next generation tobacco-free: Your Guide to the 50th Anniversary Surgeon General's Report on Smoking and Health. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health 2014.
5. Office of Environmental Health Hazard Assessment and California Air Resources Board. Health effects of exposure to environmental tobacco smoke: Final report, approved at the Panel's June 24, 2005 meeting. Sacramento: California Environmental Protection Agency 2005.
6. National Health and Medical Research Council. The health effects of passive smoking: A scientific information paper. Canberra: Australian Government Publishing Service 1997.
7. US Department of Health and Human Services. The health consequences of involuntary exposure to tobacco smoke: A report of the Surgeon General. Atlanta, Georgia: US Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health 2006.
8. Matt GE, Quintana PJE, Hovell MF, et al. Households contaminated by environmental tobacco smoke: sources of infant exposures. Tobacco Control 2004; 13: 29-37.
9. Brook I, Gober A. Recovery of potential pathogens in the nasopharynx of healthy and otitis media-prone children and their smoking and nonsmoking parents. Annals of Otology, Rhinology, and Laryngology 2008; 117: 727-730.
10. Arcavi L, Benowitz NL. Cigarette smoking and infection. Archives of Internal Medicine 2004; 164: 2206-2216.
11. Robinson P, Taylor K, Nolan T. Risk-factors for meningococcal disease in Victoria, Australia, in 1997. Epidemiology and Infection 2001; 127: 261-268.
12. Avşar A, Darka O, Topaloğlu B, et al. Association of passive smoking with caries and related salivary biomarkers in young children. Archives of Oral Biology 2008; 53: 969-974.
13. Erdemir E, Sonmez I, Oba A, et al. Periodontal health in children exposed to passive smoking. Journal of Clinical Periodontology 2009; 37: 160-164.
14. Tanaka S, Shinzawa M, Tokumasu H, et al. Secondhand smoke and incidence of dental caries in deciduous teeth among children in Japan: Population-based retrospective cohort study. BMJ 2015; 351: h5397
15. Moravej-Salehi E. Evaluation of the Relationship between Passive smoking and gingival pigmentation in women [DDS thesis]. Dental school, Islamic Azad University of medical Science, Tehran, Iran. 2014.
16. Twito D, Sade P. The effect of cigarette smoking habits on the outcome of dental implant treatment. PeerJ 2014; 2: e546.
17. Hajifattahi F, Azarshab M, Haghgoo R, et al. Evaluation of the relationship between passive smoking and oral pigmentation in children. J Dent (Tehran) 2010; 7: 119-123.
18. Kum-Nji P, Meloy L, Herrod HG. Environmental tobacco smoke exposure: Prevalence and mechanisms of causation of infections in children. Pediatrics 2006; 117: 1745-1754.
19. Avsar A, Topaloglu B, Hazar-Bodrumlu E. Association of passive smoking with dental development in young children. Eur J Paediatr Dent 2013; 14: 215-8.
20. Dong Q, Wu H, Dong G, et al. The morphology and mineralization of dental hard tissue in the offspring of passive smoking rats. Arch Oral Biol 2011; 56: 10051013.
21. Strauss RS. Environmental tobacco smoke and serum vitamin C levels in children. Pediatrics 2001; 107: 540542.
22. Avşar A, Darka O, Bodrumlu EH, et al. Evaluation of the relationship between passive smoking and salivary electrolytes, protein, secretory IgA, sialic acid and amylase in young children. Arch Oral Biol 2009; 54: 457463.
23. Saad AY. Postnatal effects of nicotine on incisor development of albino mouse. J Oral Pathol Med 1990; 19: 426-429.
24. Billings RJ, Berkowitz RJ, Watson G. Teeth. Pediatrics 2004; 113: 1120-1127.
25. Ford D, Seow WK, Kazoullis S, et al. A controlled study of risk factors for enamel hypoplasia in the permanent dentition. Pediatr Dent 2009; 31: 382-388.
26. Leroy R, Hoppenbrouwers K, Jara A, et al. Parental smoking behavior and caries experience in preschool children. Community Dent Oral Epidemiol 2008; 36: 249-257.
27. Hanioka T, Ojima M, Tanaka K, et al. Does secondhand smoke affect the development of dental caries in children? A systematic review. Int J Environ Res Public Health 2011; 8: 1503-1519.

28. Arora M, Weuve J, Schwartz J, et al. Association of environmental cadmium exposure with pediatric dental caries. Environ. Health Perspect 2008; 116: 821-825.

29. De S, Fenton JE, Jones AS, et al. Passive smoking, allergic rhinitis and nasal obstruction in children. J Laryngol Otol 2005; 119: 955-957.

30. Dahlström A, Ebersjö C, Lundell B. Nicotine exposure in breastfed infants. Acta Paediatr 2004; 93: 810-816.

31. Sakki T, Knuuttila M. Controlled study of the association of smoking with lactobacilli, mutans streptococci and yeasts in saliva. Eur. J. Oral Sci. 1996; 104: 619-622.

32. Wan AK, Seow WK, Purdie DM, et al. A longitudinal study of Streptococcus mutans colonization in infants after tooth eruption. J Dent Res 2003; 82: 504-508.

33. Hanioka T, Ojima M, Tanaka K, et al. Does secondhand smoke affect the development of dental caries in children? A systematic review. Int J Environ Res Public Health 2011; 8: 15031519.

34. Ruottinen S, Karjalainen S, Pienihäkkinen K, et al. Sucrose intake since infancy and dental health in 10 year old children. Caries Res 2004; 38: 142-148.

35. Alm A, Wendt LK, Koch G, et al. Oral hygiene and parent related factors during early childhood in relation to approximal caries at 15 years of age. Caries Res 2008; 42: 28-36.

36. Aligne CA, Moss ME, Auinger P, Weitzman M. Association of pediatric dental caries with secondhand smoking. JAMA 2003; 289: 125864.

37. Majorana A, Cagetti MG, Bardellini E, et al. Feeding and smoking habits as cumulative risk factors for early childhood caries in toddlers, after adjustment for several behavioral determinants: A retrospective study. BMC Pediatr 2014; 14: 45.

38. Nakayama Y, Mori M. Association of environmental tobacco smoke and snacking habits with the risk of early childhood caries among 3 year old Japanese children. J Public Health Dent 2015; 75: 157162.

39. Taghavi N, Mollaian M, Alizadeh P, et al. Orofacial clefts and risk factors in Tehran, Iran: A case control study. Iran Red Crescent Med J 2012; 14: 25-30.

40. Anderko L, Braun J, Auinger P. Contribution of tobacco smoke exposure to learning disabilities. Journal of Obstetric, Gynecologic & Neonatal Nursing 2010; 39: 111-117.

41. Skolnick E, Vomvolakis M, Buck K, et al. Exposure to environmental tobacco smoke and the risk of adverse respiratory events in children receiving general anesthesia. Anesthesiology 1998; 88: 1144-1153.

42. Stasic A. Perioperative implications of common respiratory problems. Seminars in Pediatric Surgery 2004; 13: 174-180.

43. Lyons B, Frizelle H, Kirby F, et al. The effect of passive smoking on the incidence of airway complications in children undergoing general anaesthesia. Anaesthesia 1996; 51: 324-326.

44. Jones D, Bhattacharyya N. Passive smoke exposure as a risk factor for airway complications during outpatient pediatric procedures. Otolaryngology and Head and Neck Surgery 2006; 135: 12-16.

45. Oberg M, Jaakkola MS, Woodward A, et al. Worldwide burden of disease from exposure to secondhand smoke: a retrospective analysis of data from 192 countries. Lancet 2011; 377: 139146.

46. Carr AB, Ebbert J. Interventions for tobacco cessation in the dental setting. Cochrane Database Syst Rev 2012; 6: CD005084.

47. Policy on Tobacco Use; Oral Health Policies; Reference Manual; American Academy of Pediatric Dentistry 2015; 37; 15-16.

48. US Department of Health and Human Services. How tobacco smoke causes disease: What it means to you. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health 2010.

Correspondence to:

Anil Patil,
Associate Professor,
Pedodontics and Preventive Dentistry,
Bharati Vidyapeeth Deemed University Dental College and Hospital,
Sangli, 416414,
India.
Tel: +91 9850983500
E-mail: dranilp0888@gmail.com

Special issue: Pediatric Research
Editor: Abdulla A Alharthi, Department of pediatric nephrology, Taif University, Saudi Arabia