Review: The Impact of Second-Hand Smoking on Children Oral Health: A Narrative Review

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Abstract

Second-hand smoking (SHS) is associated with certain deleterious oral conditions like delayed development, periodontitis, loss of teeth, early childhood dental caries (EAC), orofacial clefts, oral pigmentation, and gingival pigmentation in children. Children are inclined to SHS due to their smaller bronchial tubes and weak immune systems. The autonomous connotation of SHS with the various dental and oral disease conditions in children. The impact of SHS in children has been reported by various researchers on different aspects of oral health. However ever, there is a lacuna regarding SHS and its consequences on the child's oral health. Hence, the purpose of the present manuscript is to perform a narrative review on the impact of second-hand smoking on children's oral health.

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Introduction

Oral diseases have proven to be one of the leading health concerns for many countries, including the hazardous influences of non-communicable illnesses (encompassing tobacco, insalubrious dietary habits, physical dormancy, illicit liquor usage, etc.). The acknowledgment of the relationship between tobacco usage and oral disease, and the paybacks of tobacco termination, on oral health consequences forestalls the National oral health programs to aggressively implement actions to ban tobacco/smoking usage at clinical and community levels. For ages, it has been documented that long-term exposure to smoking has deleterious effects on oral and overall health and can be associated with conditions like oral mucosal lesions and malignancies, orofacial clefts, gingivitis, and periodontitis, dental caries, oral pigmentation, and early tooth loss.[1] Second-hand smoking (SHS) denotes to released fume from the red-hot cigarette, and non-smokers inhale that.[2] Second-hand smoking exposure may initiate *in utero*, and it may continue all over a childhood with increased breathing rates and larger surface area of the lungs being exposed and defenseless to the ill effects of SHS.[2]

Second-hand smoking is considered a grave risk at the community health level. It has been assessed that approximately a billion adult smokers are prevalent worldwide, and almost.[7] billion children are exposed to involuntary tobacco fume. Children are more prone to SHS due to their minor bronchial tubes and poorly developed immune systems. Children breathe faster and inhale more harmful chemicals per kilogram of body weight than adults.[3] Children of the younger age group are especially susceptible to ETS exposure. They have advanced ventilation rates and can gasp in increased amounts of ETS for the same level of external exposure.[4] Second-hand smoking causes several ill effects on children by inclining them to cancer, CVD, other respiratory diseases, and neurological illnesses and hamper their intellectual capabilities. More than 4000 harmful components have been recognized in tobacco smoke comprising tar, CO, HCN, phenols, NH3, CH2O, C6H6, H_2N_2O, and nicotine.[3]Therefore, it is imperative to assess active and SHS for further clinical research accurately.[5] The impact of SHS in children had have been reported by various researchers on different aspects of oral health. However ever, there is a lacuna regarding SHS and its consequences on the oral health of children.

Hence, the purpose of the present manuscript is to perform a narrative review on the impact of second-hand smoking on children's oral health.

Literature review:

Effect on early tooth eruption:

It is well-documented that SHS has deleterious effects on general health; however, only very little information is accessible regarding oral health. One of the most significant disturbances in tooth development due to SHS and nicotine exposure affect the primary and permanent dentition and make them more prone to dental caries (DC) upon eruption.[6] There are few studies reported that a minimal risk for deciduous teeth caries due to SHS.[7,8,9] Hence, restoring the primary teeth has become an important aspect, and studying the correlation between SHS and DC of the permanent tooth has become imperative wherein the permanent second molar teeth that develop later in life is expected to be the most susceptible to postnatal nicotine exposure.[10]

It is a well-known fact that early childhood caries (ECC) are a common health issue. They are accompanied by odontalgia, difficulty in chewing, problems associated with facial growth, and final tooth arrangement in children. ECC occurs due to the approximation between premature enamel crystals in erupting teeth and acid produced by oral bacterial fermentation present on the tooth surface. Nicotine contact upsurges the vulnerability to acid on developmental imperfections on the tooth's surface due to the following reasons: inhibiting the enamel matrix calcification8, premature growth of enamel crystals, and increased production of acid-producing plaques.[11,12] The tooth development in the initial stages may also be a causative factor for dental caries because early tooth eruption could enhance the probability of cariogenic interaction.[13]

Second-hand smoke and dental caries (DC)

Previous studies have shown that smoking by family members could be a significant jeopardy pointer for the development of DC in permanent teeth, especially in teenagers.[10] Literature have documented the correlation between SHS and oral health in children have also stated about the socioeconomic status, poor oral hygiene and diet has also shown to cause adverse behaviors associated with tobacco use leading to insalubrious behaviours.[14] A systematic review conducted by Hanioka reported the presence of a causal correlation between SHS and ECC that was made conceivable by the evaluations grounded on consistent fundamentals without certain obvious biological believability.[13] There are four cohort studies reported depicting a favorable implication among environmental tobacco smoke or SHS and ECC.[15.16,17,18]

Nicotine produces cotinine being a direct metabolite related explicitly to environmental tobacco exposure. The concentration of salivary cotinine or other body fluids can serve as an active method to evaluate the correlation of environmental tobacco smoke exposure with dental caries (DC).[19] There are not many studies that validated the relationship of cotinine with DC except one. Since its level mirrors short-term exposure to nicotine, it is noteworthy to gather accurate data on prolonged tobacco exposure.[20]

Three significant mechanisms explain the chances of formation of DC by exposure to SHS:

- Direct contact and acquaintance of the emerging tooth buds to various compounds in the smoke, thus, causing delayed formation and impairment in mineralization;[21]
- Salivary gland damage caused due to smoke chemicals, leading to a reduced salivary flow, thus, affecting the buffering capacity and cleansing;[22] and
- impairment of the immune system of children and increased colonization of *S mutans*, that helSHS in the formation of DC[23,24]

The main causative factors associated with DC were related to diet control, oral hygiene practices, and several other demographics. Behavioral influences associated with the progression of DC.[25] SHS have been established to have an adversative consequence on children's decayed, missing, and filled teeth (DMFT) and affect the gingival index scores due to increased cotinine levels and bacterial colonies.[2,25]

Effects on periodontium

Periodontitis, a chronic condition, is typified by irritation of the supporting tooth tissue structures, causing the breakdown of the surrounding supporting CT and the jawbones thus, leading to irreparable loss of the particular bone.[26]

The exact process that aids in forming periodontal pathology due to smoking is still unspoken. However, various epidemiological studies conducted previously denote the connotation between smoking and periodontitis. Individuals who smoke may portray a suggestively increased plaque index, and the common bleeding sites in smokers are much less than that of non-smokers.[27] A

greater frequency of bacterial species is allied with the disease of the periodontium when compared to non-smokers that consist of *Porphyromonas gingivalis, Aggregatibacter actinomycetencomitans, Bacteroides forsythus, Prevotella intermedia, Fusobacterium nucleatum,* etc.[28] Nevertheless, few authors showed no significant alterations amongst smokers and non-smokers in connotation to the periodontal microorganisms with their frequency and sub-gingival amount.[29] A strong association is established amid the degree of smoking, the bacterial quantity of bacteria, and probing depth using real-time PCR.[30,31] It has been documented that the strength of the responses of the blood vessels, especially in gingivitis (caused due to plaque accumulation) in smokers, was approximately half that in non-smokers. The majority of information portrayed that cigarette smoke might reduce bleeding gums that could occur due to vascular alterations in the periodontium.[32]

Nicotine is thought to have an adverse effect on the propagation, attachment, and chemotaxis of the cells of periodontal ligament and persuade the production of pro-inflammatory cytokine by human gingival fibroblasts harmoniously with lipopolysaccharide released from *Escherichia coli* and *Porphyromonas gingivalis,*.[33,34] Thus, studies have suggested that nicotine singularly could result in periodontal bone loss owing to periodontitis.[35] The cigarette smoke inhalation due to SHS augments demolition of bone in the ligature-induced periodontal lesion. The levels of matrix metalloproteinase (MMP)- 2 were also found to be more unprotected versus non-exposed individuals; thus, suggesting that MMP-2 particles could be accountable for the enhanced cell damage seen in the PDL region of smokers.[36] A few studies showed that nicotine, with or without lipopolysaccharide (LSHS) association, from periodonto-pathogenic bacteria, could increase interleukins 6 & 8 protein formation by the gingival fibroblasts. However, pro-and anti-inflammatory cytokines have been described to have a minimal relationship with tobacco components in other studies. Cigarette smoke consists of effective inhibitors of both gene expression and protein production (like IL-1β, IL-8, IL-2, and TNF-α), It has been reported in studies with smokers versus non-smokers with moderate to severe chronic periodontitis, the pro-and anti-inflammatory cytokines, and few anti-resorptive gingival tissue agents of expresses lesser number of interleukins (1β, 8, 10, TNF-α, MMP-8, and OPG) in smokers whereas type 6, 1ra47 and INF-γ were higher comparatively. Hence, smoking initiates bone obliteration in periodontal disease and embraces decreased amount of anti-inflammatory/resorptive factors, and might also encompass an increase in pro-inflammatory cytokines (IL-6 and INF-γ).[37]

Second-hand smoking and alveolar bone density

Smoking and periodontal pathology have a strong relationship that has been established by several authors that explain the triggering action of nicotine products for the cytokine overproduction in the body due to depressed oxygen levels. The higher levels of cytokines lead to a breakdown in the supporting tissues structures of the teeth. Smoking also adversely impacts bone metabolism in individuals with good oral hygiene. The amalgamation of the tobacco smoke and decreased bone density destructively disturbs alveolar bone height and density that could be elicited by the estrogen deficiency associated with IL-1, IL-6, TNF α elevation, thus affecting both jaw and systemic bones.[38,39] It has been shown by radiographic analysis that deleterious effects on periodontal health and the reduction in bone loss are comparatively abridged in people who quit smoking. The plausible explanation of this could be due to the impact on specific bone resorptive mediators that get affected by tobacco components (e.g., the combination of nicotine and bacterial lipopolysaccharide (LSHS) augmented prostaglandin (PGE) production by peripheral monocytes.[40]

Second-hand smoking and oral pigmentation

The occurrence of oral cavity pigmentation due to excessive melanin production has an antagonistic effect on the appearance and poses a significant implication in the differential diagnosis of such cases since the pigmentation due to melanin deposits might be a clinical indicator of certain general ailments and use of medication.[41] Smoker's melanosis is one of the most familiar findings occurring due to melanin pigmentation that might be encouraged by the motivation of melanin-producing cells due to the induction of existing tobacco products (like nicotine and benzopyrene). There appears to be a positive connection between tobacco smoke and melanin pigmentation because the pigmentation decreases with the cessation of the habit.[13] few researchers have shown a strong association between SHS and specific side effects like childhood asthma, DC,[8] unprompted abortions, periodontitis, the child's attitude, cognitive problems, and juvenile malignancies.[42,43,44] A Japanese study has elicited increased melanin pigmentation incidence in the kids whose parents were smokers.[13] The plausible mechanism for this could be attributed to the polycyclic amines in cigarette smoke like nicotine and $C_{20}H_{12}$, which have triggering influences for melanin synthesis. Subsequently, gingival melanocytes get affected easily by cigarette smoke. The stimulant materials reach the gingival melanocytes from the mucosa, saliva, and the systemic course into the blood flow. Hence, the smoke primarily enters the circulation through the nose, carrying out the melanocytes indirectly.[13,45] It can be presumed

that pigmentation in the oral mucosa may employ a defensive outcome by combining with specific materials in tobacco smoke or food that can enter the tissue. Therefore, these aggravating issues might travel towards the mucosa and blood-producing similar consequences in small kids like that of the smoker because children breathe in rapidly compared to adults.[46]

Second-hand smoke and orofacial clefts

Orofacial clefts (OFCs) are the most typical types of major congenital defects, accounting for about 1.5 to 2 per 1000 births.[47] There are shreds of evidence from previous literature that is unswerving in establishing a helpful correlation amongst active smoking by mother and non-syndromic orofacial clefts.[48] Nevertheless, cigarette smoking by the mother and its hazardous effects may be misjudged at times, and women who are non-smokers might get uncovered to SHS, which is usually not considered to be necessary, especially in developing countries.[49]

The orofacial clefts require a multidisciplinary treatment approach from natal life until adulthood. Individuals with their family members might agonize psychologically and entail substantial health and related services disbursement.[50]

Conclusion

SHS has been shown to cause serious deleterious actions on children's overall general and oral health, ranging from delayed dental development to the prevalence of early childhood caries, severe effects of the periodontium, and alveolar bone. These adverse impacts of SHS on kids and younger individuals can be mirrored by elevated levels of cotinine level thereby enhancing the microbial accumulation, poor DMFT, and gingival index scores. Hence, proper counselling should be provided to parents and guardians regarding S and its ill effects.

IMPLICATIONS FOR TOBACCO REGULATION:

It was evident there a significant impact of SHS on children oral health. Counselling for parent and guardian is imperative to avoid such delitoriosu effects in children caused by SHS. Appropriate preventive and treatment protocols should be adopted to avert such deleterious hazards on the oral health of pediatric patients. Significantly, this review provides the evidence of SHS has an impact of on children oral health. Finally, the information regarding impact of SHS on tooth eruption, dental caries, periodontium, bone density, oral facial clefts, and oral pigmentation obtained from this review will be used to advise the population modelling of long-term health impacts.

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