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Letter to the Editor

Passive Smoking: Oral and Dental Effects

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Dear Editor-in-Chief

Passive smoking, also known as involuntary smoking, second hand smoking or exposure to environmental tobacco smoke (ETS), is defined as inhalation of the cigarette smoke of another individual or the exhale of a smoker (1). Passive smoking can adversely affect the health of non-smokers of all age groups (1). The association of passive smoking with life threatening conditions such as lung cancer, cardiovascular diseases, and sudden infant death syndrome has been well confirmed. Besides, passive smoking is correlated with low birth weight, asthma, bronchitis, pneumonia, otitis media, increased incidence of tuberculosis, Crohn's disease, learning disorders, developmental retardation, high systolic and diastolic blood pressure, child behavior disorders and spontaneous abortion (2-4).

ETS contains over 4000 chemical agents adversely affecting the oral health of passive smokers (5). Cotinine is a nicotine biomarker with a half-life longer than that of nicotine. Measurement of cotinine level is a suitable and reliable objective and quantitative screening tool for determination of exposure to ETS as it is for active smoking (5, 6). A dose-dependent correlation exists between the number of cigarettes smoked by a smoker and the plasma and saliva cotinine levels of his/her non-smoker companion (5). Passive smoking changes the normal oral and nasopharyngeal flora and may cause upper airway infection (2). It may decrease alveolar bone density (1) or cause severe periodontitis (7), implant failure (8), gingival pigmentation in children and adults (2, 4), primary and permanent tooth decay (2, 5) and tooth loss (6). It may also delay tooth development (9). Passive smoking is a risk factor for occurrence of or facial clefts as well (10).

The cigarette smoke products in active and passive smoking result in edema and inflammation via the activity of pro-inflammatory agents and local vasoconstriction. Systemically, these products decrease the level of saliva IgA and serum IgG and suppress the function of T helper cells in host immunity responses (6). It appears that passive smoking, via the above-mentioned mechanisms and oxidative stress, can cause periodontal disease like severe periodontitis, decrease the alveolar bone density and lead to tooth loss. In active smokers, in comparison to passive smokers, plaque accumulation is among the main causes of periodontal disease (7). Moreover, cigarette smoke, via the generation of products such as carbon monoxide and cyanides, delays wound healing and its nicotine content inhibits cell proliferation and osteoblastic activity and stimulates alkaline phosphatase activity. It adversely affects fibroblast activity and decreases the production of fibronectin and collagen by them (1). Passive smoking leads to vascular destruction, endothelial inflammation, atherosclerosis, PH changes, and production of cytokines and therefore cardiovascular diseases. Via the same mechanism, it also causes implant failure and increases the risk of failure of dental implants in passive smokers 2.3 times bigger than the risk of those who are not exposed to passive smoking (8).

The mechanism of action of cigarette smoke in increasing gingival pigmentation is via the activity of polycyclic amines such as nicotine and benzoperylene that are present in cigarette smoke and enter into the blood circulation following inhalation. They indirectly stimulate the melanocytes (4).

Nicotine also enhances the proliferation of cariogenic bacteria such as mutans streptococci in the oral cavity of smoking mothers and the mothers transfer these bacteria to their infants. Moreover, nicotine decreases the level of vitamin C, which is associated with the proliferation of *S. mutans* and results in subsequently increased risk of caries. Furthermore, it lowers the saliva pH and decreases the salivary flow and the buffering capacity of saliva while increasing other cariogenic bacteria such as lactobacilli (5).

Passive smoking also delays dental development via several mechanisms such as interference with reciprocal induction of oral ectomesenchymal tissues, interference with tooth mineralization due to oxidative stress and nutritional deficiency due to the adverse effects of passive smoking on appetite (causing loss of appetite)(9).

Enhancing the knowledge of smokers about the oral and dental problems caused by direct or indirect exposure to cigarette smoke can significantly encourage them to quit smoking (2). Rate of cigarette smoking and passive smoking is widely variable in different countries and districts. But, clearly, the prevalence of passive smoking is higher than active smoking and attempts must be made to control it due to its adverse health effects. Global health organizations must focus on this issue to resolve this public health dilemma (1). Further investigations are required on the role of passive smoking in development of oral cancer and precancerous lesions. Effect of passive smoking on oral and dental health must be further scrutinized in future studies.

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