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Correlation of exposure to environmental tobacco smoke and periodontitis in passive smokers; a systematic review

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Abstract

Introduction: Smoking is a major health hazard. Cigarette smoke is produced by burning of complex organic compounds that include highly toxic components. Passive smokers are non-smoker individuals that are exposed to second-hand smoke (SHS) or environmental tobacco smoke (ETS). Passive or involuntary smoking is currently a major public health dilemma worldwide. Considering the increasing number of passive smokers in today's world, it is imperative to assess the potential adverse effects of passive smoking on oral health and periodontal tissue.
Objectives: This study aimed to assess the correlation between exposure to ETS and periodontitis in passive smokers.
Methods: In this systematic review, the Google Scholar, PubMed and Scopus databases were searched for relevant articles published from 1990 to 2020. The extracted data were tabulated.
Results: Search of the literature yielded 77 articles based on the searched keywords. After assessing the abstracts and excluding the duplicates, 68 articles remained. After accurate assessment by the examiners, 21 articles were selected for the full-text review. Finally, seven eligible articles remained in the study for the final review.
Conclusion: Cigarette smoke affects the immune system and aggravates periodontal destruction as such. Despite the available evidence regarding the correlation of periodontal parameters and exposure to ETS in passive smokers, more comprehensive studies on this topic are still required to achieve a definite therapeutic protocol considering the small number of available studies on this topic, methodological flaws and the existing controversies.

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Introduction

Cigarette smoking is a major public health dilemma worldwide. Numerous studies have evaluated the adverse effects and hazards of active smoking. Cigarette smoke contains high amounts of carcinogens and toxic compounds such as nicotine, benzopyrene and carbon monoxide. Despite the global attempts to confront the cigarette smoking epidemic, smoking still remains a major cause of morbidity and mortality worldwide, with a global death rate of approximately six million people annually (1). Cigarette smoke has adverse effects on different body organs such as the cardiovascular system, respiratory system, gastrointestinal system, oral mucosa, teeth and periodontium (2-5). Exposure to environmental tobacco smoke (ETS) was first discussed by Surgeon in 1986, since he was the first scientist to report its adverse effects and complications (6). Exposure to ETS is also known as passive smoking, involuntary smoking or second-hand smoking. Active cigarette smoking negatively affects the periodontal health of smokers. Additionally, ETS has a chemical composition similar to that of the mainstream smoke, it may have adverse effects on the periodontal health of non-smokers as well (2). Passive smoker or involuntary smoker refers to non-smoker individuals who are continuously exposed to cigarette smoke in a closed environment. More precisely, passive smokers are individuals exposed to the smoke of at least

Key point

In this review article, the relationship between exposure to environmental tobacco smoke and periodontitis in passive smokers was investigated. Articles were searched in Google Scholar, PubMed and Scopus database from 1990 to 2020. Seven articles met the criteria, although smoking was one of the risk factors for periodontitis, more studies were needed to evaluate the direct impact.

one cigarette daily or spend at least two hours daily in a closed environment polluted with cigarette smoke (7). The risks and adverse effects of passive smoking on the cardiovascular and respiratory systems, the oral cavity, and also the salivary glands have been previously evaluated. Additionally, the role of passive smoking in development of gingival pigmentation, gingivitis, and periodontitis has been previously studied (8-11). The smoke that is inhaled and then exhaled from the smoker's lungs is referred to as the mainstream smoke. The side stream smoke is the smoke released into the environment from the burning end of a cigarette. All these different types of smokes enter the air and can affect both smokers and passive smokers. In general, the side stream smoke accounts for approximately 85% of the ETS (6). Due to the increased number of passive smokers, assessment of the potential adverse effects of passive smoking on oral health and periodontium is imperative (12).

Approximately 50% of the United States residents have experienced one form of gingivitis while 80% suffer from some degrees of periodontitis. Thus, it is important to find the possible etiologies for these conditions (13). Periodontitis is an inflammatory disease of the tooth supporting structures. It is caused by specific microorganisms (mainly anaerobes and Gram-negative bacteria). Periodontitis is characterized by progressive destruction of the periodontal ligament and alveolar bone, pocket formation and/or gingival recession (14). Evidence shows that the type of microbial flora is different in different parts of the tooth and even in healthy and diseased gingiva. In addition, some reports are available regarding the higher prevalence and severity of periodontal disease in smokers and passive smokers (15). The exact mechanism of effect of cigarette smoke on the periodontium has not yet to be clarified. No significant difference has been reported in the amount of dental plaque in smokers and passive smokers; however, the microbial challenge in smokers is due to qualitative, rather than quantitative, changes in dental plaque (16). Akinkugbe et al showed a positive correlation between exposure to ETS and periodontitis in passive smokers. However, the authors declared the methodological flaws in study designs, assessment of ETS and periodontitis and adjusted variables and suggested some strategies to improve the quality of such studies (2).

The present study aimed to comprehensively review the available articles published from 1990 to 2020 regarding the correlation of exposure to ETS and periodontitis in passive smokers.

Methods

An electronic search of the literature was carried out in the Google Scholar, PubMed, and Scopus databases for relevant articles published from 1990 to 2020. The titles, abstracts, and bibliography of the selected articles were evaluated to find the most relevant topics. The following keywords were searched according to PEO:

PEO;

P: {Mesh term}: Tobacco Smoke Pollution / {Free term}: Passive Smoker, Second Hand Smoker

E: {Mesh term}: Tobacco Smoke Pollution / {Free term}: Environmental Tobacco smoke, Pollution

O: {Mesh term}: Periodontal, Pocket, Periodontal Index, Periodontal Attachment Loss, Tooth Loss, Gingival Recession, {Free term}: Probing Pocket Depth, Clinical Attachment Loss, Missing Teeth, Gingival Recession

The statistical population of this systematic review included all quantitative research articles regarding the correlation of exposure to ETS and periodontitis in passive smokers published from 1990 to 2020.

The search strategy included a literature search for relevant original articles in English with a clear methodology, clinical trials, and in vitro and in vivo studies.

The exclusion criteria were animal studies, studies with unavailable full-texts, review articles, case reports, and letter to editors, questionnaires, and also poor-quality articles. The title of all articles was first evaluated and those out of the scope and objectives of the study were excluded. Next, the abstract and then the full-text of the retrieved articles were read to exclude the irrelevant articles and those that met the exclusion criteria.

After final selection of the articles, the required information was extracted by using an extraction table in Excel software. Endnote X8 was used to organize the topics and abstracts, and to identify the duplicates. Finally, seven relevant articles were selected for the final review. The Joanna Briggs Institute (JBI) critical appraisal tool was used to assess the risk of bias:

JBI critical appraisal checklist for analytical cross sectional and cohort studies:

1. Were the criteria for inclusion in the sample clearly defined?
2. Were the study subjects and the setting described in detail?
3. Was the exposure measured in a valid and reliable way?
4. Were objective, standard criteria used for measurement of the condition?
5. Were confounding factors identified?
6. Were the two groups similar and recruited from the same population?
7. Were the exposures measured similarly to assign people to both exposed and unexposed groups?
8. Were the confounding factors identified?

Results

Search of the databases with the aforementioned keywords yielded 77 articles. After assessment of the titles and abstracts, 68 articles were selected among the initially retrieved 77 for further assessment. By more detailed evaluation by the examiners, 21 articles were selected among the 68 for full-text review. Finally, seven articles

that met the eligibility criteria were selected for the final review. Of the enrolled seven articles, four articles were analytical, cross-sectional, two of them were cohort, and one was a review article. Figure 1 presents the PRISMA flow diagram of the study.

A systematic review was retrieved regarding periodontal indices including the pocket depth (PD), clinical attachment loss (CAL), bleeding on probing (BOP), gingival index (GI) and periodontal index (PI). However, it had some methodological flaws with respect to study design, ETS and periodontitis assessment, and adjusted variables and offered some suggestions for improvement of the quality of future studies on this topic.

For the risk of bias assessment, the JBI checklist was used for analytical, cross-sectional and cohort studies. Most of the studies did not have appropriate inclusion criteria, did not control for the effect of confounding factors, and did not standardize the exposure time and conditions. Thus, the risk of bias was relatively high. In addition, irrespective of the objectives of the study, three articles were retrieved regarding microbiological and biochemical indices related to periodontitis and exposure to ETS, the results of which were assessed as secondary objectives.

Discussion

Periodontitis is a common chronic inflammatory disease with a multifactorial etiology. It is caused by gram-negative anaerobic bacteria and is influenced by a number of factors such as an immunocompromised state and smoking (25).

Passive smokers are non-smoker individuals that are exposed to second-hand smoke (SHS) or ETS, which is a combination of side-stream smoke and mainstream smoke. Some similarities exist between the mainstream smoke and SHS, which supports the risk factors of passive smoking (26). Inability to precisely estimate the duration of exposure of passive smokers to ETS is an important concern in epidemiological studies. Evaluation of passive exposure to cigarette or tobacco smoke is much harder than the assessment of smoking rate.

Cigarette smoke affects the immune response and can aggravate periodontal destruction. Cigarette smoking decreases the immune response to bacterial invasion, and impairs the response of neutrophils to periodontal infection. Moreover, it increases the release of tissue degrading enzymes (27,28). A large body of scientific evidence supports the role of nicotine in progression of periodontal disease. Smokers experience a higher prevalence and intensity of periodontal disease, compared with non-smokers. Nicotine has toxic effects on the peripheral blood circulation and causes gingival vasoconstriction. As a result, the number of immune cells in the gingival tissue decreases, compromising the immune system and healing mechanisms. Nicotine can also decrease the primary and secondary immune responses by reduction of the chemotactic and phagocytic activity of leukocytes. Clinically, smoking is associated with increased probing pocket depth (PPD), calculus deposition, alveolar bone loss, acute necrotizing ulcerative gingivitis, and osteoporosis in menopaused women. Despite the attempts of researchers, the exact mechanism of effect of cigarette smoke on periodontal status and wound healing has yet to be fully elucidated (29).

Yamamoto et al, reported that in active and passive smokers, the number of teeth with CAL ≥3.5 mm was significantly higher than that in non-smokers. They estimated that the odds of periodontitis were 2.87 (ng/mL) in passive and 4.91 (ng/mL) in active smokers, compared with non-smokers, after controlling for the effect of confounders such as the lifestyle (17).

Erdemir et al demonstrated that CAL in children exposed to the cigarette smoke of their parents was significantly lower than that in children with non-smoker parents (0.09 mm) (19). In their study, although the PPD and CAL values in passive smokers were lower than the corresponding values in active smokers and higher than the corresponding values in non-smokers, these differences did not reach statistical significance. In their study, PPD and CAL were correlated with the concentration of cotinine (18).

Tanaka et al found no significant correlation between passive smoking and prevalence of periodontal disease (21). Chatrchaiwiwatana and Ratanasiri found no significant correlation between the exhaled carbon monoxide and dental caries, periodontitis and oral health in active or passive smokers (20). Cotinine is among the main metabolites of nicotine break down. Etter et al reported that the concentration of cotinine in non-smokers with smoker friends or spouses was 1.5 times the rate in non-smokers with non-smoker friends or spouses (30). Cotinine is a nicotine metabolite, which can be quantified in urine, blood or saliva. It can serve as a valid and reliable index for differentiation of smokers and non-smokers. Although measurement of the level of cotinine may not be an accurate indicator of the level of nicotine in all individuals due to metabolic differences, the salivary cotinine is extensively measured for assessment of nicotine exposure, because collection of saliva is less invasive and easier compared with blood and urine collection (31). Akinkugbe et al, in their review study found a significant correlation between exposure to ETS and risk of development of periodontitis (2). Some studies evaluated the clinical periodontal parameters and microbiological indices in patients exposed to ETS. Accordingly, Kanmaz et al reported Treponema denticola in 17.4% of smokers, 0% of passive smokers, and 9.5% of non-smokers (18). According to Karasneh et al (32) tobacco, use affects the sub-gingival bacterial profile by decreasing the count of beneficial bacteria and increasing the count of periopathogenic bacteria. In patients with chronic periodontitis, sub-gingival bacteria (particularly Treponema species) play an important role in development of periodontitis.

Table 1. Qualitative data retrieved from the studies regarding the effect of exposure to ETS on periodontal health

Author (year)	Title	Study population	Study design	Assessment method	Outcome criteria
Yamamoto et al (2005) (17)	Association between passive and active smoking evaluated by salivary cotinine and periodontitis	Active smokers, Passive smokers, Non-smokers	Analytical cross-sectional	Level of salivary cotinine and severity of periodontitis were compared between active and passive smokers	PPD, CAL, Salivary cotinine
Kanmaz et al (2019) (18)	Microbiological and biochemical findings in relation to clinical periodontal status in active smokers, non-smokers and passive smokers	Active smokers, Passive smokers, Non-smokers	Analytical cross-sectional	Microbiological and biochemical findings regarding periodontal status	PPD, CAL, porphyromonas gingivalis and Treponema denticola counts, and salivary levels of MMP-8 and IL-8
Erdemir et al (2010) (19)	Periodontal health in children exposed to passive smoking	6-12-year-old children	Analytical cross-sectional	Effect of parents' smoking on periodontal indices of their children	PI, GI, BOP, CAL, PPD, salivary cotinine
Chatrchaiwiwatana et al (2011) (20)	Association between exhaled carbon monoxide and oral health status in active and passive smokers	Active and passive smokers	Analytical cross-sectional	Correlation of dental caries and periodontitis with exhaled carbon monoxide	Exhaled carbon monoxide
Akinkugbe et al (2016)(2)	Systematic review and meta-analysis of the association between exposure to environmental tobacco smoke and periodontitis endpoints among nonsmokers	Passive smokers	Review 2013-2015	Correlation of exposure to ETS and periodontitis risk factors	CAL, PPD, tooth loss

PPD: Probing pocket depth; CAL: Clinical attachment loss; GI: Gingival index; PI: Plaque index; BOP: Bleeding on probing; MMP-8: Matrix metalloprotenaise-8; IL-8: Interleukin-8

Table 2. Qualitative data retrieved from the cohort studies on the effect of exposure to ETS on periodontal health

Author (year)	Title	Study population	Study design	Methodology	Outcome measure
Tanaka et al (2013) (21)	Active and passive smoking and prevalence of periodontal disease in young Japanese women.	Female active and passive smokers and non-smokers	Retrospective cohort	Correlation of periodontal disease with active and passive smoking	Pocket depth
Tanaka et al (2005)(22)	Active and passive smoking and tooth loss in Japanese women: baseline data from the Osaka maternal and child health study	Active and passive smoker pregnant women	Retrospective cohort	Correlation of active and passive smoking with tooth loss	Tooth loss

Table 3. Results of studies on the effects of exposure to ETS on periodontal health

Author	Results
Yamamoto et al (2005)	In active and passive smokers, the number of teeth with CAL ≥3.5 mm was significantly higher than that in non-smokers. The odds of periodontitis were 2.87 in passive smokers and 4.91 in active smokers, compared with non-smokers after controlling for the effect of confounding factors including the lifestyle.
Kanmaz et al (2019)	The smoker group showed higher PPD and CAL compared with non-smokers (P<0.05). Passive smokers showed lower PPD and CAL than active smokers and higher PPD and CAL than non-smokers. However, these differences were not significant. The PPD and CAL values were correlated with the concentration of cotinine (P<0.05). The salivary levels of MMP-8 and IL-8 in smokers were lower than the corresponding values in non-smokers and passive smokers, but not significantly.
Erdemir et al (2010)	The mean salivary concentration of cotinine was significantly higher in children exposed to ETS than controls. The mean CAL was significantly lower in children exposed to ETS compared with controls (0.09 mm, *P*<0.05).
Chatrchaiwiwatana et al (2011)	No significant correlation was noted between the exhaled carbon monoxide and dental caries, periodontitis, and oral health in active and passive smokers.
Akinkugbe et al (2016)	A positive correlation was noted between exposure to ETS and periodontitis in passive smokers.
Tanaka et al (2013)	The frequency of periodontal disease in smokers was higher than that in non-smokers (1.56). No significant correlation was noted between periodontal disease and exposure to ETS.
Tanaka et al (2005)	The results indicated that active and passive smoking may be associated with an increase in tooth loss in Japanese female young adults.

PPD: Probing pocket depth; CAL: Clinical attachment loss; ETS: Environmental tobacco smoke; MMP-8: Matrix metalloprotenaise-8; IL-8: Interleukin-8

Table 4. Qualitative data retrieved from the articles regarding the effect of exposure to ETS on microbiological and biochemical findings related to periodontitis

Author (year)	Title	Study population	Study design	Methodology	Outcome measure
Nishida et al (2006) (23)	Association between passive smoking and salivary markers related to periodontitis	Active smokers, passive smokers, non-smokers	Analytical cross-sectional	Measuring the salivary markers related to periodontitis	IL-1B, lactoferrin, albumin aspartate, aminotransferase
Numabe et al (1998) (24)	Phagocytic function of salivary PMN after smoking or secondary smoking	Active smokers, passive smokers	Retrospective cohort	The number of salivary phagocytic polymorphonuclears before and after cigarette smoking in active and passive smokers	Number of salivary phagocytic polymorphonuclears

IL-1B: Interleukin 1 beta

Table 5. Results of articles regarding the effect of exposure to ETS on microbiological and biochemical indices related to periodontitis

Author (year)	Results
Nishida et al (2006)	No significant difference was noted in the frequency of parameters related to periodontal status such as the level of IL-1beta, albumin, and salivary aspartate transaminase between passive smokers and non-smokers
Numabe et al (1998)	The phagocytic activity of salivary polymorphonuclears significantly increased after both active and passive smoking

Smoking may cause gingival and periodontal problems, dental caries and oral cancer. Saliva is the first biological fluid exposed to cigarette smoke. Saliva can reflect the health status of the human body because it contains a number of proteins, hormones and antibodies. Thus, in many conditions, identification of changes in salivary markers can help in diagnosis or follow-up of the course of oral conditions.

In assessment of biochemical findings related to periodontal status, Kanmaz et al reported lower level of MMP-8 and IL-8 salivary biomarkers in smokers, compared with non-smokers and passive smokers; however, this difference did not reach statistical significance (18). Furthermore, Nishida et al discussed that exposure to ETS increased the salivary level of interleukin 1-beta, albumin, and aspartate aminotransferase. However, they did not find a significant difference in the frequency of periodontal pathogens between passive smokers and non-smokers (23). Numabe et al evaluated the phagocytic activity of salivary polymorphonuclears and noted a significant increase in salivary polymorphonuclear count after active and passive smoking. Polymorphonuclears are a group of white blood cells that are responsible for body defense against pathogenic microorganisms (24).

Conclusion

It appears that further scrutiny of the correlation of exposure to ETS and periodontitis in passive smokers requires an evidence-based investigation. Although ETS is an important risk factor for development of periodontitis, scientific evidence showing the direct effect of ETS on periodontitis is limited and not conclusive.

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Authors' contribution

SR, MSS and VS were the principal investigators of the study. VS, MG, SR, MSS and FS were included in preparing the concept and design. MG and SR revisited the manuscript and critically evaluated the intellectual contents. All authors participated in preparing the final draft of the manuscript, revised the manuscript and critically evaluated the intellectual contents. All authors have read and approved the content of the manuscript and confirmed the accuracy or integrity of any part of the work.

Conflicts of interest

The authors declare that they have no competing interests.

Ethical issues

This systematic review was conducted in accord with the World Medical Association Declaration of Helsinki. The Ethics Committee of Tabriz University of Medical Sciences approved this study. The institutional ethical committee at Tabriz University of Medical Sciences approved all study protocols (Ethical code#IR.TBZMED.VCR.REC.1400.120). Accordingly, written informed consent was taken from all participants before any intervention. This study was extracted from DDS thesis of Vian Shateri at this university (Thesis #66541). Besides, ethical issues (including plagiarism, data fabrication, double publication) were completely observed by the authors.

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