



Impact of Smoking and Lifestyle on Periodontal Health

Mayssam Adnan Tahir¹, Duha Qais Sabah², Ali Saleh Hatem³, Abeer Ahmed Akhmais⁴,
Mohammed Dhyeaa⁵

¹ College of Dentistry, University of Mashreq, Iraq, Email: mayssam.a.tahir@uom.edu.iq

² College of Dentistry, University of Mashreq, Iraq, Email: duha.q.sabah@uom.edu.iq

³ B.D.S., M.Sc., Lecturer, College of Dentistry, Al Kut University College, Wasit 52001, Iraq,
Email: ali.saleh@uokut.edu.iq

⁴ Pharmacist, Iraqi Ministry of Health, Iraq, Email: Abeerahmed00010@gmail.com

⁵ Pharmacist, Iraq, Email: mohammediaa85@yahoo.com

Abstract

Smoking is a significant risk factor for periodontal disease, profoundly affecting oral health. Smoking remains highly prevalent in many populations despite extensive awareness campaigns by the World Health Organization. Smoking weakens the immune system, reduces blood flow to the gums, and fosters the growth of harmful bacteria, which heightens the risk of gingivitis, periodontitis, and tooth loss in smokers. The likelihood of developing periodontal disease is markedly higher among smokers than among non-smokers. Smokers often experience more severe periodontal disease, with greater bone loss and deeper periodontal pockets compared to non-smokers. Both the frequency and duration of smoking contribute to a higher risk of periodontal disease. More significant loss of the connective tissue that holds teeth in place, leading to tooth mobility and eventually tooth loss if untreated. Smoking accelerated alveolar bone loss. Smokers generally show a poorer response to periodontal treatments. Smoking is a major cause of numerous diseases, affecting nearly every organ in the body. Health conditions caused or exacerbated by smoking are Chronic Obstructive Pulmonary Disease, Lung Cancer, Coronary Heart Diseases, Peripheral Artery Disease, Mouth, Throat, Oesophagus cancer. Tobacco smoking is the leading cause of preventable death globally. It significantly increases the risk of heart disease, lung cancer, stroke and COPD. Annually, around 8 million people succumb to smoking related illnesses. Smoking even one cigarette a day greatly increases the risk of developing coronary heart disease and stroke. Research findings underscore that even minimal tobacco intake substantially increases the risk of heart disease and stroke with light smokers facing a 40-50% higher risk compared to non smokers.

1. Introduction

Periodontal disease, also known as gum disease, is a chronic inflammatory condition affecting the tissues surrounding and supporting the teeth.^[1] Comprising the periodontium are four primary tissues: the gingiva, periodontal ligament (PDL), cementum, and alveolar bone. Smoking has long been recognized as a significant risk factor for periodontal (gum) disease, a chronic inflammatory condition that affects the supporting structures of the teeth. The detrimental effects of smoking on periodontal health are well-documented and profound. Smoking compromises the immune response, impairs wound healing, and reduces the effectiveness of periodontal treatments. Periodontal disease begins with gingivitis, characterized by inflammation and bleeding of the gums. Continued smoking exacerbates this condition, leading to periodontitis, where the gums recede, bone structure supporting the teeth is damaged, and teeth may loosen or even be lost. Nicotine and other compounds in tobacco smoke change the oral environment, fostering the proliferation of harmful bacteria and impairing blood flow to the gums. Periodontal disease primarily caused by bacterial plaque, a sticky, colorless film that constantly forms on teeth. If not removed daily through brushing and flossing, plaque can harden into tartar, which is more challenging to remove and requires professional cleaning. The disease follows a chronic course characterized by periods of activity and remission^[1]. Eventually, it leads to the affected tooth either falling out or being extracted or necessitates the therapeutic removal of dental plaque. The disease is influenced by tooth modifiable and non modifiable factors. Modifiable factors include those that can be changed or controlled such as oral hygiene, diet, stress, and various systemic diseases^[1] While non-modifiable factors include genetic predispositions and age. Indeed, the manifestation and progression of periodontal disease can vary significantly based on individual factors such as bacterial flora composition and specific local and systemic influences. Factors like genetics, oral hygiene practices, smoking habits, systemic conditions like diabetes and medications can all influence the development and severity of periodontal disease^[2]. Smoking is widely recognized as a significant risk factor for periodontitis.

2. Pathophysiology of Periodontal Disease / Periodontal Microbiology of Smokers

Periodontal disease is triggered by an imbalance in the oral micro biome where harmful bacteria outnumber beneficial bacteria^[3]. Secondary bacteria such as *Fusobacterium nucleatum*, take the advantage of that entire oral environment. The oral microbiome, which consists of a complex community of microorganisms, plays a significant role in

maintaining oral health. However, an imbalance in this micro biome, referred to as dysbiosis, can be associated with poor host health (i.e. poor oral hygiene tobacco use[4]. It can cause harmful bacteria to gather below the gum line. These bacteria release molecules known as pathogen-associated molecular patterns (PAMPs), which then spark inflammation in the intermediate area. This leads to a rise in Gingival Crevicular Fluid (GCF) from the bloodstream providing a favorable environment for bacteria to thrive. The bacterial community that forms in the periodontal pocket further drives the immune response. Neutrophils and natural killer (NK) cells are mobilized as part of the innate immune response to fight infection and inflammation. The resulting environment abundant in pro inflammatory cytokines further strengthens the influx of immune cells and the breakdown of nearby tissue[5]. This triggers an adaptive immune response where dendritic cells pick up specific antigens and present them to naive T cells. Consequently, T helper cells type 1,2 and 17 are generated, producing a receptor activator of nuclear factor - K B ligand (RANK-L), which accelerates bone loss[1]. Normally, harmful organisms would be eliminated, and the recruited immune cells would undergo programmed cell death, allowing for reversible tissue damage, characteristics of gingivitis[1]. Smoking affects oral bacteria by altering the oral environment. While smoking is associated with increased plaque accumulation, there isn't substantial evidence to suggest that smoking directly accelerates the rate at which plaque develops. Cigarette smoke contain harmful chemicals that can reduce the mouth's natural defense mechanisms and disrupt the balance of oral organisms. Smoking can lower the oxidation-reduction potential (Eh) in the oral cavity. This change in Eh may create conditions that are more favorable for the growth of anaerobic bacteria, which thrive in low oxygen environments[4].

3.Effect of Smoking on Periodontal Health

3.1.Impact of smoking on gingival inflammation

Gingivitis is an inflammation of the gums, often caused by plaque buildup around the gum line. One of the early signs is bleeding gums, especially during brushing or flossing.

This bleeding indicates that the gums are inflamed and may be a precursor to more severe gum disease if left untreated. Regarding smoking, while it's commonly known to cause vasoconstriction due to nicotine content, the response can vary among individuals. [6] Factors such as how heavily and frequently one smokes, along with personal differences in nicotine response, could influence this variability.

Clinical indicators of gingival inflammation, such as redness, bleeding and exudation, may be less evident in smokers, due to vasoconstrictive actions of nicotine, which reduce gingival blood flow.

Certainly, prolonged and heavy smoking can indeed lead to a reduction in gingival bleeding which can obscure the clinical marker of bleeding on probing that dentists often rely on to monitor periodontal health.

3.2. Smoking and Alveolar Bone Loss

Periodontal damage was noted around canines and premolars, which aren't associated with the Molar- Incisor pattern typically observed in localized juvenile periodontitis. Some studies have shown that smoking decreases the mineral content in bone, yet the connection between smoking, osteoporosis, and periodontitis remains ambiguous. [7] Furcation defects with severe periodontal damage are typically rare, even among young adults. However, when they do, their treatment tends to be intricate & the prognosis often becomes poorer.

3.3. Smoking and Host Immune Response

Smoking impact on the inflammatory response and its impairment of protective mechanisms play a significant role in accelerating periodontal destruction. [8] Immune response in the subgingival (below the gumline) environment plays a crucial role in shaping the microbial community present in the oral cavity.

4. Smoking and oral health

4.1.Smoking and periodontal disease

The role of tobacco smoking as a causative factor in the development of the periodontal disease. Smoking also gives an encouraging environment for microbes in the mouth such as Porphyromonas gingivalis, Prevotella intermedia, and Aggregatibacter actinomycetemcomitans because the byproducts of smoking prevent the mechanisms that limit the growth of harmful bacteria in the oral cavity [9]. Thus, smoking promotes early stages of periodontal disease . Smoking is 11 an independent risk factor for the initiation, extent and severity of periodontal disease. Additionally, smoking can lower the chances for successful treatment[9].

4.2.Smoking and dental biofilm

It has been reported that smokers have a poor level of oral hygiene when compared to nonsmokers, the tooth brushing effectiveness of smokers is much less, and calcium concentration in the dental plaque of smokers has been found to be significantly higher than in nonsmokers, which suggests a direct influence on the rate of calculus formation and deterioration of oral hygiene[10]

It is well known that smokers have higher plaque index scores than nonsmokers. Males have significantly more plaque than females and in both genders, smokers have almost twice the percentage of marginal line with an adherent plaque as nonsmokers[11].

4.3. Smoking and gingivitis

Cigarette smoking also causes a lowering of the oxidation reduction potential (Eh), which causes an increase in anaerobic plaque bacteria. Furthermore, tobacco smoke contains phenols and cyanides which can account for antibacterial and toxic properties. Smokers harbor significantly higher levels of these and are at significantly greater risk of infection with *Bacteroides forsythus* than nonsmokers [12]. *P. gingivalis* is also more likely to sub gingival infect smokers than nonsmokers. However, the relative risk for infection due to this bacterium is significantly higher. It has been found that three species of Gram-negative bacteria, *Branhamella catarrhalis*, *Neisseria perflava*, and *Neisseria sicca* are more susceptible to cigarette smoke than three species of Gram positive bacteria *Streptococcus mitis*, *Streptococcus salivarius*, and *Streptococcus sanguinis* [13].

4.4. Smoking and dental caries

Dental caries is a multi-microbial disease caused by various associations and is not infectious. It is considered a diet- and pH-dependent process due to the acid demineralization of the tooth enamel by sugar-fermenting microorganisms [14]. The data on dental caries prevalence in tobacco smokers and chewers were not completely known. The literature discussed both the increased and decreased prevalence of dental caries in tobacco users [15].

4.5. Smoking and saliva

Saliva is a complex and important body fluid which is very essential for oral health. Saliva is required for protecting the oral mucosa, teeth remineralization, digestion, taste sensation, pH balance and phonation. It includes a variety of electrolytes, peptides, glycoproteins, and lipids which have antimicrobial, antioxidant, tissue repair, and buffering properties. Saliva is the first biological fluid that is exposed to cigarette smoke, which contains numerous toxic compositions responsible for structural and functional changes in saliva [16]. Smoking causes a short-term increase in salivary secretion, the long-term effects of tobacco use are unclear.

4.6. Smoking and halitosis

Bad breath, also known as halitosis, is a symptom in which a noticeably unpleasant breath odor is present [17]. A special reason that smoking tobacco causes halitosis. In fact, there are several reasons why people who smoke are far more likely to suffer from a particular type oral odor called, what else, smoker's breath. The most immediate way that cigarettes cause bad breath is by leaving smoke particles in the throat and lungs.

4.7. Passive smoking

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, Passive smoking can adversely affect the health of non-smokers of all age groups [18].

4.8. Passive smoking and oral health

Environmental tobacco smoke (ETS), contains over 4000 chemical agents adversely affecting the oral health of passive smokers [19]. 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 [20,19],(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 [19]

Passive smoking changes the normal oral and nasopharyngeal flora and may cause upper airway infection [21]It may decrease alveolar bone density [18] or cause severe periodontitis, implant failure, gingival pigmentation in children and adults, primary and permanent tooth decay and tooth loss. It may also delay tooth development. Passive smoking is a risk factor for occurrence of or facial clefts as well [22].

The cigarette smoke products in active and passive smoking result in edema and inflammation via the activity of pro-inflammatory agents and local vasoconstriction.

4.9. Smoking and Cessation

A recent epidemiological study from the United States highlighted a significant drop in teenage smoking after a peak in the mid-1990s, attributed to successful campaigns aimed at deterring smoking among adolescents[23]. Many young smokers are addicted to nicotine, and like adults, they face similar challenges with relapse when attempting to quit. Effective prevention programs have shown promise in reducing adolescent smoking rates, emphasizing that preventing smoking initiation during youth can greatly reduce future tobacco use. However, it's important to note that damage done to periodontal tissues from previous smoking cannot be reversed.

5. Periodontal Treatment in Smokers

Following non-surgical therapy, which includes scaling, root planing and professional teeth cleaning, smokers experienced less favorable healing outcomes in terms of gingival bleeding reduction and pocket depth reduction compared to non-smokers[24].

Non-surgical treatments for periodontal disease have shown promising results in both smokers and non-smokers. However, smokers tend to have deeper periodontal pockets. A 5-year study examining non-surgical periodontal treatments in 40 patients has revealed significant findings regarding smokers & their oral health outcomes. Those who smoked were more prone to requiring new surgical interventions compared to non-smokers. Smokers in contrast to non-smokers demonstrated poorer outcomes following flap debridement surgery regarding reduction in pocket depth and improvement in attachment levels, particularly in areas with initially deep periodontal pockets.

Conclusion

Smoking generally has a negative health effects, because smoke inhalation inherently poses challenges to various physiologic processes. Diseases related to tobacco smoking have been shown to kill approximately half of long-term smokers when compared to average mortality rates faced by non-smokers. Passive smoking, also known as involuntary smoking, second hand smoking or exposure to environmental tobacco smoke (ETS), can adversely affect the general and oral health of non-smokers of all age groups. Smoking is well-established risk factor for periodontal disease. It changes the human micro flora, human immune response that leads to destruction of the supporting tissues of the tooth, the vasoconstriction of peripheral blood vessels which is caused by smoking have less overt signs of gingivitis than nonsmokers and clinical signs of gingival inflammation such as redness, bleeding, and exudation are not as apparent in smokers. Contrary to the number of studies showing dental caries in smokers, the studies demonstrated that the smokeless tobacco (ST) chewers had more caries experience when compared to tobacco smokers. In the present scenario, tobacco is a major killer, thus a major drive against tobacco should be started, and a public health, dentist, shall be a model role player in this and dentists have an important role in creating awareness among the public regarding the detrimental effect of smoking on oral health.

References:

1. Bergström J, Eliasson S, Dock J. Exposure to tobacco smoking and periodontal health. *J Clin Periodontol*. 2000;27(1):61-8. [Google Scholar]
2. Silva H. Tobacco Use and Periodontal Disease—The Role of Microvascular Dysfunction. *Biology (Basel)*. 2021;10(5). [Google Scholar]
3. Khan S. Effect of Smoking on Periodontal Health. *Dis Mon*. 2011;57(4):214-7. [Google Scholar]
4. Dyke T. Inflammation and Periodontal Diseases: A Reappraisal. *J Periodontol*. 2008;79(8):1501-2. [Google Scholar]
5. Darveau R. Periodontitis: a polymicrobial disruption of host homeostasis. *Nat Rev Microbiol*. 2010;8(7):481-90. [Google Scholar]
6. Graves D, Oates T, Garlet G. Review of osteoimmunology and the host response in endodontic and periodontal lesions. *J Oral Microbiol*. 2011;3. [Google Scholar]
7. Naderi N, Semyari H, Elahinia Z. The impact of smoking on gingiva: a histopathological study. *Iran J Pathol*. 2015;10(3):214-20. [Google Scholar]
8. Arno A, Schei O, AL, Wæehaug J. Alveolar bone loss as a function of tobacco consumption. *J Clin Periodontol*. 1959;17(1):3-10. [Google Scholar]
9. Bagaitkar J, CA, Patel CK, Renaud DE, Demuth DR, Scott DA. Tobacco smoke augments Porphyromonas gingivalis Streptococcus gordonii biofilm formation. *PLoS One*. 2011; 6 (11):e27386.
10. Tymkiw KD, Thunell DH, Johnson Gk, Joly S, Burnell KK, Cavanaugh JE, Brogden KA, Guthmiller JM. Influence of smoking on gingival crevicular fluid cytokines in severe chronic periodontitis. *J Clin Periodontol*. 2011 Mar;38(3):219-228.
11. Kinane DE, Chestnutt IG. Smoking and periodontal disease. *Crit Rev Oral Biol Med* 2000;11:356-65.
12. Sham AS, Cheung LK, Jin LJ, Corbet EF. The effect of tobacco use on oral health. *Hong Kong Med J*. 2003 ;9:271-7.
13. Calsina G, Ramón JM, Echeverría JJ. Effects of smoking on periodontal tissues. *J Clin Periodontol*. 2002; 29(8):771-6.
14. Simon-Soro A, Belda-Ferre P, Cabrera-Rubio R, Alcaraz LD, Mira A. A tissue-dependent hypothesis of dental caries. *Caries Res* 2013;47:591-600
15. Sathyanarayanan R, Carounnanidy U. Classification and management dental caries. New concepts. *Indian J Dent Res* 2002;13:21-5.
16. Kelbouskas E, Kelbauskiene S, Nedzelskiene I. The influence of smoking on oral health. *Medicina*. 2005, 41: 418-26.
17. Kapoor, U; Sharma, G; Juneja, M; Nagpal, A "Halitosis: Current concepts on etiology, diagnosis and management". *European Journal of Dentistry*. (2016). 10 (2): 292-300. .
18. El_Batran MM, Soliman NL, Mikael FF. Passive smoking and alveolar bone density. *Aust J Basic Appl Sci*, 2009. 3 (2): 713-9
19. Tanaka K, Miyake Y, Sasaki S, Ohya Y, Miyamoto S, Matsunaga I, Yoshida T, Hirota Y, Oda H. Active and passive smoking and tooth loss in Japanese women: baseline data from the Osaka Maternal and Child Health Study. *Ann Epidemiol*, 2005; 15 (5):358 -64.
21. Hajifattahi F, Azarshab M, Haghgoo R, Lesan S. *Dent (Tehran)*, 2010, 7(3): 119-23.
22. Taghavi N, Mollaian M, Alizadeh P, Moshref M, Modabernia S, Akbarzadeh A. Orofacial clefts and risk factors in Tehran, Iran: a case control study. *Iran Red Crescent Med J*, .2012;. 14(1): 25- 30.

23. Haffajee A, Socransky S. Relationship of cigarette smoking to the subgingival microbiota. *J Clin Periodontol.* 2001;28(5):377-88. [Google Scholar]
24. Preshaw P, Lauffart B, Zak E, Jeffcoat M, Barton I, Heasman P. Progression and treatment of chronic adult periodontitis. *J Periodontol.* 1999;70(10):1209-20. [Google Scholar]