Indian Journal of Clinical Anatomy and Physiology 2024;11(3):138–145

Content available at: https://www.ipinnovative.com/open-access-journals

Indian Journal of Clinical Anatomy and Physiology

Journal homepage: https://www.ijcap.org/

Review Article

The impact of smoking on periodontal health: A comprehensive review

Ena Sharma[1]*, Maninder Kaur[1], Arshdeep Singh Kooner[2]

[1]*Rayat Bahra Dental College and Hospital, Mohali, Punjab, India*
[2]*Private Practice, Punjab, India*

ARTICLE INFO

Article history:
Received 25-08-2024
Accepted 15-09-2024
Available online 17-10-2024

Keywords:
Smoking
Periodontal disease

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 Health 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

* *Corresponding author*.
E-mail address: dr.sharma_ena@yahoo.co.in (E. Sharma).

https://doi.org/10.18231/j.ijcap.2024.030

environment, fostering the proliferation of harmful bacteria and impairing blood flow to the gums.

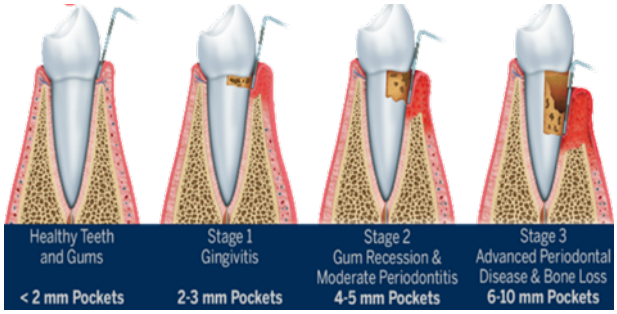

Figure 1: Progression of periodontal disease

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] However, in circumstances not entirely clear, these harmful bacteria persist, evading control by the initial immune response, leading to chronic and unresolved inflammation. This chronic inflammation progresses to fibrous and irreversible damage to gum, tissues, and local bone, defining periodontal disease.[6]

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.[7] Factors such as how heavily and frequently one smokes, along with personal differences in nicotine response, could influence this variability. Understanding these relationships between gingival bleeding, smoking and vascular responses can be crucial for clinicians in diagnosing and managing periodontal diseases effectively. Nicotine stimulates the sympathetic nervous system, leading to the release

of neurotransmitters such as catecholamine.[8] These neurotransmitters act on alpha- receptors on blood vessels, causing vasoconstriction, which is the narrowing of blood vessels. The vasoconstriction induced by smoking affects the blood supply to the Periodontal tissue.[9] As a result, smokers may exhibit fewer obvious signs of gingivitis compared to non-smokers.

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.

Table 1: The study of Bergstrom et al.[10] suggests that the reduced gingival bleeding observed in smokers compared to non-smokers can be attributed to & main factors

Factor description	
Vasoconstriction of Gingival Vessels	Smoking induces vasoconstriction in the blood vessels of the gums. Effect: Reduced blood flow to the gingiva, which can decrease bleeding.
Heavier Keratinization of the Gingiva	Smokers often exhibit thicker and more keratinized gingival tissues. Effect: Increased resistance to mechanical trauma, hence less bleeding.

Smoking is associated with various vascular changes, including vasoconstriction, endothelial dysfunction and impaired microcirculation.[14] These alterations can affect the delivery of oxygen to tissues, leading to reduced oxygen saturation level in the blood of smokers compared to non-smokers.[14] Absolutely, the clinical implications of tobacco consumption on gingival vasculature are significant, regardless of ongoing debate.[14] This reduction in gingival bleeding, often observed in heavy smokers, may obscure signs of inflammation and early stage of periodontal diseases during routine dental examination.

Table 2: The studies mentioned provide insights into the complex effects of nicotine consumption on gingival blood flow

Study	Findings
Clarke et al.[11] Nicotine	Initial increase in gingival blood flow post-nicotine consumption, followed by vasoconstriction and reduced blood flow below baseline levels with prolonged exposure. This suggests that while nicotine initially stimulates blood flow in the gums, prolonged exposure may lead to vasoconstriction & reduced blood flow.
Baab & Oberg[12]	Used Doppler Flowmetry to measure gingival circulation during smoking. Marked and rapid rise in gingival blood flow immediately after smoking. Heightened blood flow returned to baseline levels within approximately 10 minutes.
Palmer et al.[13]	Contrary to expectations, smoking did not show a significant reduction in gingival blood flow. Implication: Challenges the assumption that smoking compromises gingival blood flow and leads to periodontal disease.

Table 3: Summarizes the studies of effect of smoking on alveolar bone

Study	Findings
KA Hollenbach et al.[15]	Smoking associated with accelerated alveolar bone loss and increased severity of periodontal disease.
Mullally et al.[16]	Smoking exacerbates bone loss in aggressive periodontitis, with a greater impact in the maxillary arch.
Linder and Mullally[17]	Furcation defects were exclusively observed in individuals who smoked.
Persson et al.[18]	Smoking associated with altered bone metabolism markers, indicating increased bone resorption.

4. 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.[15] 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.

In a radiographic investigation conducted within an older adult population referred to a specialist periodontal clinic. The study revealed that among cigarette smokers, the prevalence of molar furcation was twice as high as compared to non-smokers.[16] The present findings conclude that tobacco smoking significantly impacts alveolar bone loss.[19] The analysis reveals the influence of tobacco extends beyond soft tissue alone, impacting alveolar bone as well. Tobacco consumption may serve as a complicating factor in the etiology of periodontal disease.

5. 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.[20] Immune response in the subgingival (below the gumline) environment plays a crucial role in shaping the microbial community present in the oral cavity.

Polymorphonuclear leukocyte PMNs (Neutrophils) are indeed the primary type for defending the body against bacterial invasion through a process called phagocytosis.[20] Corberand in 1980 highlights the complex effects of tobacco smoke on PMNs.According to the study, PMN motility was severely depressed by a solution of tobacco smoke concentrate, indicating impairment in the ability of these immune cells to migrate to sites of infection / inflammation.[20] Smokers generally have elevated blood levels of PMNs (polymorphonuclear leukocytes) compared to non-smokers. However, the suppression of chemotaxis in smokers relative to now smokers is concerning.[16]

Cigarette smoking has been shown to activate the release of elastase, an enzyme (released by neutrophils) that plays a role in breakdown of elastin, a key component of connective tissue.[2] Elastase has the capacity to cause tissue damage. Destructive effects of elastase, particularly in the context of cigarette smoke exposure, are prominently displayed in lung diseases such as emphysema. When exposed to cigarette smoke, elastase- defined animal models do not develop emphysema. This demonstrates the crucial role of clastase in mediating the tissue damage associated with cigarette smoke exposure and the pathogenesis by emphysema. Moreover, the heightened production of oxygen species induced by cigarette smoking can diminish the tissue levels of α- 1 protease inhibitors. Consequently, this enables elastase & other enzymes, which have the potential to inflict tissue damage, to persist unchecked at active sites within the tissue. Smokers cohibit lower levels of functional elastase & its inhibitor-complex in gingival crevicular fluid compared to non-smoking controls.

Nicotine metabolites have been found to accumulate within the periodontium. This contribute to vasoconstriction, a narrowing of blood vessels, which in turn reduces blood flow to the gums and surrounding tissues. This affects the activity of crucial immune cells including Polymorphonuclear leukocytes and macrophages.

Tobacco use has been associated with elevated levels of neutrophils in peripheral blood. Furthermore, it facilitates their migration through capillary walls.[20]

6. Smoking ang Serum IgG

P. gingivalis has the capability to modulate innate host defense functions, including immune subversion of IL-8 secretion, complement activity TLRA activation, through various virulence factors. This modulation ultimately leads to impaired host defense against periodontal pathogens.

Table 4: Summarize the effect of smoking on serum IgG

Study	Findings
Jiang et al.[21] 2020	- Current smokers in a US adult population showed lower IgG antibody titers against periodontal bacteria (P. gingivalis, Campylobacter rectus, Prevotella nigrescens). - Adjustment for confounding factors still showed decreased antibody levels in smokers. - Decreased IgG antibodies may compromise host immune response, potentially protecting against periodontal pathogens.
Darveau et al.,[4] 2010	- P. gingivalis can modulate innate host defense functions, impairing host defense against periodontal pathogens.

In vitro studies have highlighted that nicotine exposure can hinder antimicrobial activities, such as the production of superoxide anion and hydrogen peroxide.[13]

Separate investigations have also revealed a reduced number of helper lymphocytes among cigarette smokers. This decline could potentially affect both B cell function & antibody production.

7. Toothbrushing Behavior in Smokers and Non-Smokers

While the exact reasons why smokers tend to have more plaque buildup than non-smokers aren't fully understood.[20] One significant factor is the impact of smoking and oral hygiene behaviors. Smokers may be likely to engage in proper oral hygiene practices such as regular brushing, flossing and dental checkups.

Toothbrushing behavior indeed plays a significant role in oral cleanliness. People who brush their teeth regularly tend to have less plaque buildup compared to those who brush less frequently or inconsistently.[22]

Smokers often exhibit lower toothbrushing efficiency compared to non-smokers. This could be due to various factors such as the adverse effects of smoking on gum health, reduced saliva production and possibly the presence of tobacco stains that may make it harder to visually assess plaque removal.

Furthermore, there is higher calcium concentration in the dental plaque of smokers compared to non-smokers.[23] The Male smokers tend to spend less time brushing their teeth compared to age-matched male non-smokers.[24]

Danielsen's findings between 13 smokers and 16 non-smokers.They indicate that within the confines of an experimental gingivitis model and among healthy adults who already maintain good oral hygiene,there were no

significant differences observed in the rate of plaque accumulation between smokers and non-smokers.[20]

The correlation you've highlighted between smoking behavior and grooming habits, particularly in adolescence, is fascinating. It suggests that while smokers may exhibit less well developed health related behaviors compared to now smokers, they may place a higher emphasis on grooming behaviors.

Individuals with extroverted trails may engage in more impulsive behaviors, such as smoking. Behavioral differences between smokers and non-smokers likely play a significant role in the observed disparities in oral cleanliness.

8. Smoking and Genetic Variability

Variations among individuals within the species can arise from typical genetic diversity or variations known as polymorphisms. Differences in how individuals respond to conditions or treatments can also be attributed to unique genetic variations among individuals. Having a positive IL-1 genotype boosts the risk of tooth loss by 2.7 times, whereas smoking raises it by 2.9 times. The combined effect results in an estimated 7.7 times higher risk of tooth loss. There was significant attachment loss in genotype positive patients who were smokers.[25]

In a study involving 323 maintenance patients, researchers observed a correlation between bleeding upon probing and a positive IL-1 genotype among non-smokers.However, the association between bleeding on probing and a positive IL-1 genotype did not achieve statistical significance among smokers. The authors propose that smoking might have a more pronounced effect on the likelihood of bleeding, which could overshadow any detectable influence of the IL-1 genotype within this subgroup.[25]

In a study involving 154 patients, the authors examined the potential pharmacogenetic interaction between arylamines found in tobacco smoke and the N-acetyltransferase 2 (NAT2) profile in individuals diagnosed with periodontal disease. The process of acetylation plays a role in detoxifying arylamines present in tobacco smoke. NAT2 polymorphisms in the population determine whether individuals are rapid or slow acetylators, influencing this detoxification process. The study results showed that patients with the most severe periodontal disease tended to be slow acetylators, (risk ratio of more than 2). The authors suggest that the impaired metabolism of arylamines could potentially impact the immune response, possibly acting as immunosuppressants.[26]

In another study, patients with a positive NAT2 genotype were significantly linked to greater severity of bone loss, particularly notable among smokers aged 40-55 years.[26] In summary, polymorphisms appear to account for some of the variability observed in disease among patients. However, further studies are needed to fully understand the specific roles these genetic variations play.

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.[27] 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.

In a literature review focusing on health promotion and behavioral strategies to prevent periodontal diseases, Kallio[28] outlines the various approaches available to dental professionals at the chair side and within society. Key chair side interventions include taking a brief smoking history and offering advice on quitting smoking, assessing how family and peer influences impact patient behavior, and providing written educational materials.

Collaboration between dental professionals, along with other healthcare providers and administrators, in shaping school policies to implement smoking bans has been highlighted as a successful model of community engagement. A youth intervention study[29] demonstrated significant impact, revealing a 22% reduction in lifetime cigarette consumption among participants in the test group compared to controls over a span of 15 years.

10. 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.[30]

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.

Investigators studying the longevity of osseointegrated implants in patients with a positive IL-1 genotype observed

a notable impact of patients smoking habits on implant survival. They found that smoking influenced the success rate of implants. Smokers explained a 2.5 times higher rate of implant failure, whereas the genetic status (IL-1 genotype) did not emerge as a significant factor affecting implant survival.[25]

In a follow up study of 3-5 years, focusing on patients with severely resorbed maxilla rehabilitated with implants, researchers noted that the failure rate stabilized after initial 2 years, smokers exhibited a higher incidence of implant failures compared to non-smokers during this period.[25]

Complications following implant placement were found to be more frequent in smokers, especially in implants where the cover screw was placed. Additionally, the severity of complications was linked to the duration of smoking habits. Smokers (65.3%) exhibited a lower success rate compared to non-smokers (82.7%) implants severely placed in grafted maxillary sinuses.[25] Previous studies examining healing outcomes associated with connective tissue grafting have consistently reported a negative influence and poorer root coverage among smokers. Nevertheless, outcomes from guided tissue regeneration procedures in Miller Class 1 or 2 Buccal recessions remained stable over a 4-year period, regardless of whether the patients were smokers and non-smokers.[31] The success rate of implant loading in augmented ridges using deproteinized bone mineral after 4-6 years was reported as 43% for smokers and 100% for non-smokers.

In summary, the majority of evidence indicates that surgical and non-surgical treatments are less successful in smokers compared to non-smokers. Clinical studies consistently demonstrated a significant reduction in pocket depth and the number of diseased sites in both smokers and non-smokers.[32–34] Tobacco smoking hinders the healing process following non-surgical periodontal therapy.

11. Discussion

Smoking is a well-established risk factor for numerous health problems, and oral health is no exception. It significantly increases the risk and severity of periodontal disease, the leading cause of tooth loss in adults. This discussion will delve into the detrimental effects of smoking on each aspect of periodontal health and explore the supporting evidence from various studies.

The foundation of a healthy oral cavity lies in a balanced oral microbiome, where beneficial bacteria coexist with a minimal presence of harmful ones. Smoking disrupts this delicate balance. Study published in the (2020) by Y Jiang et al.[21] found that smoking alters the composition of the oral microbiome, promoting the growth of pathogenic bacteria associated with gum disease. This shift weakens the body's natural defense against infection, making individuals more susceptible to developing gingivitis, the initial stage of gum disease.

Smoking triggers a chronic inflammatory response throughout the body, including the gums. This inflammation, as evidenced by study (2018) by FRM Leite et al.[35] damages the delicate tissues supporting the teeth. This damage weakens the defense ability to fight off infection and can lead to the progression of gingivitis to periodontitis, a more severe form of gum disease characterized by bone loss and receding gums.

Even when individuals with periodontitis attempt to quit smoking, the damage may already be done. Study (2022) by PM Duarte et al[36] demonstrate that smoking hinders the body's ability to heal from periodontal treatment. This can make it more challenging to control periodontal disease and prevent tooth loss Smoking can mask the early signs of gum disease. Healthy gums typically exhibit a pink color and firm texture. However, smoking can constrict blood vessels in the gums, making them appear pale, even when inflammation is present. Consequently, this can delay the identification and management of the condition, allowing the disease to develop further.

The severity of the impact of smoking on periodontal health is directly related to the intensity and duration of the habit. According to the study by Tomar et al. (2000),[33] the likelihood of developing periodontitis grows with the number of cigarettes smoked per day and the length of time a person has been smoking. The consequences of neglecting oral health due to smoking extend beyond periodontal diseases.

11.1. Challenges in periodontitis treatment

Smoking significantly decreases the effectiveness of periodontal treatment. Study (2021) by J Chang et al.[37] show that smokers have poorer outcomes following scaling and root planing (deep cleaning) compared to non-smokers. Smokers are more likely to experience a recurrence of periodontitis after successful treatment. This highlights the ongoing detrimental effect of smoking on maintaining healthy gums and bone.

11.2. Hindered tissue regeneration

Smoking disrupts the function of fibroblasts, the cells responsible for tissue repair and regeneration. This makes it difficult for the gums and bone to heal effectively after periodontal treatment or injury. Study (2015) by TK Ng et al[38] This study found that smokers with periodontitis had significantly lower levels of growth factors necessary for tissue regeneration compared to non-smoking counterparts. Another study by A Queiroz et al[39] highlighted the negative impact of smoking on stem cells, crucial for tissue repair. Smoking was shown to decrease the number and function of these stem cells, further hindering regeneration.

Smoking significantly hinders the effectiveness of treatment and tissue regeneration. Quitting smoking is

not only essential for overall health but also crucial for maintaining healthy gums and bone, maximizing the success of periodontal treatment, and promoting optimal healing.

12. Conclusion

Although the exact ways in which smoking affects gum tissues aren't fully understood, it remains the most significant avoidable risk factor for periodontal disease. The impact varies based on how long and how much a person smokes. Additionally, the smoking habits of family members may influence behavior and could potentially lead to passive smoking effects, an area that requires more investigation. In addition to its broader systemic effects that can change how the body responds, smoking seems to have substantial local effects that could contribute to the early onset of periodontal disease in young adults who are susceptible.

To effectively pursue our goals of prevention, early disease detection, and timely intervention, the dental profession must prioritize educating young patients about how smoking impacts their gum health. By doing so, dentistry can play a vital role in promoting the overall health and well-being of our youth and future generations. This proactive approach not only addresses periodontal health but also contributes meaningfully to broader public health initiatives.

13. Source of Funding

None.

14. Conflict of Interest

None.

References

1. Silva H. Tobacco Use and Periodontal Disease—The Role of Microvascular Dysfunction. *Biology (Basel)*. 2021;10(5):441.
2. Khan S. Effect of Smoking on Periodontal Health. *Dis Mon*. 2011;57(4):214–7.
3. Dyke TEV. Inflammation and Periodontal Diseases: A Reappraisal. *J Periodontol*. 2008;79(8):1501–2.
4. Darveau RP. Periodontitis: a polymicrobial disruption of host homeostasis. *Nat Rev Microbiol*. 2010;8(7):481–90.
5. Cekici A, Kantarci A, Hasturk H, Dyke TEV. Inflammatory and immune pathways in the pathogenesis of periodontal disease. *Periodontol 2000*. 2014;64(1):57–80.
6. Graves DT, Oates T, Garlet GP. Review of osteoimmunology and the host response in endodontic and periodontal lesions. *J Oral Microbiol*. 2011;3:10.
7. Müller H, Stadermann S, Heinecke A. Longitudinal association between plaque and gingival bleeding in smokers and non-smokers. *J Clin Periodontol*. 2002;29(4):287–94.
8. Trauth JA, Seidler FJ, Ali SF, Slotkin TA. Adolescent nicotine exposure produces immediate and long-term changes in CNS noradrenergic and dopaminergic function. *Brain Res*. 2001;892(2):269–80.
9. Clarke NG, Hirsch RS. Personal risk factors for generalized periodontitis. *J Clin Periodontol*. 1995;22(2):136–45.
10. Bergström J, Floderus-Myrhed B. Co-twin control study of the relationship between smoking and some periodontal disease factors. *Community Dent Oral Epidemiol*. 1983;11(2):113–6.
11. Clarke NG, Shephard BC, Hirsch RS. The effects of intra-arterial epinephrine and nicotine on gingival circulation. *Oral Surg Oral Med Oral Pathol*. 1981;52(6):577–82.
12. Baab DA, Oberg PA. The effect of cigarette smoking on gingival blood flow in humans. *J Clin Periodontol*. 1987;14(7):418–24.
13. Palmer RM, Scott DA, Meekin TN, Odell EW, Wilson RF. Potential mechanisms of susceptibility to periodontitis in tobacco smokers. *J Periodontal Res*. 1999;34(7):363–9.
14. Naderi NJ, Semyari H, Elahinia Z. The impact of smoking on gingiva: a histopathological study. *Iran J Pathol*. 2015;10(3):214–20.
15. Hollenbach KA, Barrett-Connor E, Edelstein SL, Holbrook T. Cigarette smoking and bone mineral density in older men and women. *Am J Public Health*. 1993;83(9):1265–70.
16. Mullally BH, Linden GJ. Molar furcation involvement associated with cigarette smoking in periodontal referrals. *J Clin Periodontol*. 1996;23(7):658–61.
17. Linden GJ, Mullally BH. Cigarette smoking and periodontal destruction in young adults. *J Periodontol*. 1994;65(7):718–23.
18. MacFarlane G, Herzberg MC, Wolff LF, Hardie NA. Refractory periodontitis associated with abnormal polymorphonuclear leukocyte phagocytosis and cigarette smoking. *J Periodontol*. 1992;63(11):908–13.
19. Arno A, Schei O, Lovdal A, Wæhaug J. Alveolar bone loss as a function of tobacco consumption. *J Clin Periodontol*. 1959;17(1):3–10.
20. Borojevic T. Smoking and periodontal disease: a review. *Mater Sociomed*. 2012;24(5):274–6.
21. Jiang Y, Zhou X, Cheng L, Li M. The Impact of Smoking on Subgingival Microflora: From Periodontal Health to Disease. *Front Microbiol*. 2020;11:66.
22. Koivusilta L, Honkala S, Honkala E, Rimpelä A. Toothbrushing as part of the adolescent lifestyle predicts education level. *J Dent Res*. 2003;82(5):361–6.
23. Bergström J, Eliasson S, Dock J. Exposure to tobacco smoking and periodontal health. *J Clin Periodontol*. 2000;27(1):61–8.
24. Amarasena N, Ekanayaka ANI, Herath L, Miyazaki H. Tobacco use and oral hygiene as risk indicators for periodontitis. *Community Dent Oral Epidemiol*. 2002;30(2):115–23.
25. Borojevic T. Smoking and periodontal disease. *Mater Sociomed*. 2012;24(4):274–6.
26. Kocher T, Sawaf H, Fanghänel J, Timm R, Meisel P. Association between bone loss in periodontal disease and polymorphism of N-acetyltransferase (NAT2). *J Clin Periodontol*. 2002;29(1):21–7.
27. Haffajee AD, Socransky SS. Relationship of cigarette smoking to the subgingival microbiota. *J Clin Periodontol*. 2001;28(5):377–88.
28. Page RC. The pathobiology of periodontal diseases may affect systemic diseases: inversion of a paradigm. *Ann Periodontol*. 1998;3(1):108–20.
29. Vartiainen E, Paavola M, Mcalister A, Puska P. Fifteen-year follow-up of smoking prevention effects in the North Karelia youth project. *Am J Public Health*. 1998;88(1):81–5.
30. Preshaw PM, Lauffart B, Zak E, Jeffcoat MK, Barton I, Heasman PA. Progression and treatment of chronic adult periodontitis. *J Periodontol*. 1999;70(10):1209–20.
31. Scabbia A, Trombelli L. Long-term stability of the mucogingival complex following guided tissue regeneration in gingival recession defects. *J Clin Periodontol*. 1998;25(12):1041–6.
32. Suda R, Cao CF, Suzuki M, Hasegawa K, Sasa R. Attachment loss in rural Chinese children over a 3-year period. *Community Dent Oral Epidemiol*. 1999;27(3):216–20.
33. Tomar SL, Asma S. Smoking-attributable periodontitis in the United States: findings from NHANES III. National Health and Nutrition Examination Survey. *J Periodontol*. 2000;71(5):743–51.
34. Tonetti MS, Lang NP, Cortellini P, Suvan JE, Adriaens P, Dubravec D, et al. Enamel matrix proteins in the regenerative therapy of deep

intrabony defects. *J Clin Periodontol*. 2002;29(4):317–25.
35. Leite FRM, Nascimento G, Scheutz F, López R. Effect of Smoking on Periodontitis: A Systematic Review and Meta-regression. *Am J Prev Med*. 2018;54(6):831–41.
36. Duarte PM, Nogueira CFP, Silva SM, Pannuti CM, Schey KC, Miranda TS. Impact of Smoking Cessation on Periodontal Tissues. *Int Dent J*. 2022;72(1):31–6.
37. Chang J, Meng HW, Lalla E, Lee C. The impact of smoking on non-surgical periodontal therapy: A systematic review and meta-analysis. *J Clin Periodontol*. 2021;48(1):60–75.
38. Ng T, Huang L, Cao D, Yip YWY, Tsang WM, Yam G, et al. Cigarette smoking hinders human periodontal ligament-derived stem cell proliferation, migration and differentiation potentials. *Sci Rep*. 2015;5(2):7828.
39. Queiroz A, Albuquerque-Souza E, Gasparoni LM, França B, Pelissari C, Trierveiler M, et al. Therapeutic potential of periodontal ligament stem cells. *World J Stem Cells*. 2021;13(6):605–8.

Author biography

Ena Sharma, Reader

Maninder Kaur, Intern

Arshdeep Singh Kooner, Consultant

Cite this article: Sharma E, Kaur M, Kooner AS. The impact of smoking on periodontal health: A comprehensive review. *Indian J Clin Anat Physiol* 2024;11(3):138-145.