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A Destructive Connection: A Review of Cigarette Smoking Impact on Periodontal Health

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Abstract

Smoking is a significant global public health concern, leading to many conditions such as cancer, pulmonary diseases, and cardiovascular disorders. This article examines the impact of smoking on human health, highlighting its biochemical and physiological effects. The study highlights the detrimental effects of prolonged tobacco use, considering both active and passive smoking. The study also examines the challenges associated with smoking cessation and the role of nicotine dependence in this process. The research evaluates public health programs and preventative strategies designed to reduce smoking prevalence. The findings emphasize the urgent necessity for comprehensive smoking control policies, awareness programs, and targeted cessation measures to mitigate the adverse health effects of smoking.

Keywords: Tobacco Consumption, Nicotine Dependence, Public Health, Smoking Cessation, Health Hazards, Secondhand Smoke, Preventive Measures.

1. INTRODUCTION

Cigarette smoking and other use of tobacco is one of the world's biggest wellbeing issues that kills and sickens millions of people every year (Brugge, 2018).The World Health Organization has projected a rise in the number of smokers to 1.7 billion by 2025. (Organization, 2024).

Cigarette smoking is predisposed for a number of diseases, varying from the oral cavity to the immune system, cancer, respiratory and cardiovascular diseases (such as ischemia, peripheral heart disease and stroke).(Liccardo *et al.*, 2019) Thousands of individuals die annually due to mouth cancer caused by smoking (Inchingolo *et al.*, 2020).

Smoking is the key danger factor in the occurrence, degree and dangerousness of periodontal diseases(O'g, 2024). A common oral condition is periodontal disease that affects the supporting structures surrounding teeth according to severity can be gingivitis or periodontitis (M. Nazir *et al.*, 2020). Periodontal disease is marked by ongoing inflammation in the mouth caused by bacteria that spread across the body. Eventually, this inflammation destroys the tissues supporting the teeth, hence causing mobility, and tooth loss (Ray, 2023).

After tooth caries, it was considered the next most common cause that led to tooth loss among grownups in rich countries (Wen, Chen, Zhong, Dong, & Wong, 2022). Smoking tobacco is correlated with an increased prevalence and incidence of periodontal disease because of environmental risk factors. (M. A. Nazir, 2017)

Periodontal disease in early stages (gingivitis) would record some symptoms such as (bleeding on probing, pocket formation, edematous, redness) (Heitz-Mayfield, 2024), Advanced stage periodontitis is marked by loss of periodontal ligament and degradation of surrounding supportive components as alveolar bone structures (Stankovic & Minic, 2019). Behavioral variables like smoking affect host reactivity, which interacts with bacterial infection to induce periodontal diseases (M. A. Nazir, 2017). Smoking affects oral health by inducing the formation of more dental plaque and calculus, which are the major etiological causes for gum

disorders (Hinrichs, Thumbigere-Math, Korczeniewska, Diehl, & Ambalavanan, 2024). Some signs indicating tobacco use can be connected to less obvious periodontal inflammation indicators and symptoms are gingival hemorrhage, erythema, and edema, which imply a suppressive effect on the body's response to inflammation. (Klokkevold, Mealey, Hernandez-Kapila, & Ambalavanan, 2024). Studies have shown that deviations in the subgingival bacterial profile are the main cause of smokers' greater periodontitis rates in comparison to non-smokers. Smoking increases chemokines and cytokines, which alters the subgingival flora. Many people agree that the beginning and development of periodontal disease are significantly influenced by both host responses and periodontal bacteria (Jiang, Zhou, Cheng, & Li, 2020). According to earlier research, those who smoke heavily—at least 20 cigarettes per day—are more likely to get periodontal disease(G. Ashwini, 2019). Most studies show that smokers suffer more severe periodontal disease than nonsmokers, marked by increased bones, attachment, and tooth loss, receding gingival tissue, and pocket development (Bunæs, 2017). Several investigations have revealed that smokers react more sharply to operative or nonsurgical periodontal treatments than non-smokers. (Nilamahan, 2019)

This review is noticeable, however, because smoking is a public health concern that affects overall health. It has been an etiological cause of gum disease and results in tooth loss. If needed, proper oral hygiene care, routine check-ups, and smoking cessation are not carried out.

1.1 Aim of the review

The goal was to examine and discuss all the current publications that have researched the effects of cigarette smoking on the periodontal tissue, clarifying the impact of smoking cigarettes on periodontal disease severity and clarifying the consequence of smoking on periodontal therapy results.

1.2 Objectives

Summarizing the efficacy of smoking on gum health, comparing the severity of different types of periodontal diseases in cigarette smokers and non-smokers and categorize its influence on periodontal therapy.

2. METHOD

Review and discuss previous research related to the influence of smoking on gingival health, using (gum health, cigarette smoking, periodontitis, gingivitis, periodontal disease, pigmentation, periodontium, GCF, salivary composition) as key terms and six databases were searched including Google, Google Scholar, Journal of Periodontology, Medline Plus, PubMed and National Library of Medicine.

Inclusion criteria were cross-sectional, interventional and longitudinal studies of human and clinical articles, articles focused on tobacco and periodontal disease have defined based on clinical parameters such as plaque index (PI), gingival index (GI), bleeding on probing (BOP), pocket depth (PD), clinical attachment loss (CAL) and susceptibility to the progression of periodontitis in smokers and non-smokers. Exclusion criteria were review articles, old articles, animal studies, case series and letters to the editor.

Based on the goal of this study. First, titles were approximated; articles were either included or excluded depending on their relevance. Of the more than 100 investigations on the link between smoking and periodontal health, about 25 papers examined and compared.

3. Smoking's influence on periodontal tissue

3.1 Biological events and consequences

Though epidemiological studies have demonstrated a close link between smoking and periodontal disease, the mechanisms by which smoking promotes the onset of periodontitis are still not completely understood (Loos & Van Dyke, 2020). Aiming to understand the mechanisms behind smoking's control of gum tissue, in vitro and in vivo studies have looked at the effects of cigarette compounds like nicotine and cotinine on the gum tissue (Kopa-Stojak & Pawliczak, 2024). Reports say nicotine usually negatively affects the proliferation, adhesion, and chemotaxis of periodontal tissue cells. It also makes human gingival fibroblasts generate pro-inflammatory cytokines in a synergistic way with lipopolysaccharide from P. gingivalis and Escherichia coli (Zhu *et al.*, 2022).

Among the toxic chemicals in cigarette smoke is nicotine. A passive smoking model was created to study how cigarette smoke generally affects periodontal tissues (Apatzidou, 2022). In ligature-induced periodontitis, cigarette smoke inhalation (CSI) has been linked to faster loss of periodontal bone. Examining the gingival tissue at periodontitis locations more closely showed exposed mice had greater amounts of matrix metalloproteinase (MMP)-2 than non-exposed animals (Spiropoulou, Zareifopoulos, Bellou, Spiropoulos, & Tsalikis, 2019). Bastos *et al.*, (2017) claim this finding suggests MMP-2 could be one of the elements responsible for the increased tissue destruction observed in smokers' periodontal tissues.

Sansone *et al.*, (2023) claim that these results usually suggest that nicotine seems to be a key molecule for the higher periodontal damage observed in smokers and might be at least mostly accountable for the negative consequences of cigarette smoke in general.

Marques *et al.,* (2021)reported smokers had urine levels of around 1200 ng/mL, plasma and saliva nicotine levels of approximately 100 ng/mL.

Nonsmokers usually have plasma and saliva nicotine levels below 2 ng/mL unless they are passive smokers. (Feng, Cummings, & McIntire, 2018)

Aleksandrowicz *et al.,* (2021)claim that smokers' gingival crevicular fluid had more TNF- α and IL-8 than that of nonsmokers. Conversely, smoking and its byproducts have been connected to lower levels of pro- and anti-inflammatory cytokines as well as W. Zhang *et al.,* (2022) claim that at least for IL-1 β, IL-8, IL-2, and TNF- α, cigarette smoke seems to include potent gene expression and protein production inhibitors.

Research on the impact of smoking on gene expression indicates that while smokers had higher levels of IL-6, IL-1ra, and INF- γ than nonsmokers, they had lower levels of IL-1β, IL-8, IL-10, TNF-α, MMP-8, and OPG. Although smokers' 1β: IL-1ra ratio was similar to that of the healthy group, their RANKL: OPG and IL-6: IL-10 ratios in sites with periodontitis were greater than those of nonsmokers (Falfán-Valencia *et al.,* 2020). F. Liu *et al.,* (2024) claim that smoking might raise levels of pro-inflammatory cytokines like IL-6 and INF- γ and reduce degrees of anti-inflammatory and anti-resorptive factors like IL-10 and OPG, accordingly, which could also affect bone deterioration in periodontal disease.

3.2 The Effect of Smoking on Causes and Pathogenesis of Periodontal Disease

The greater frequency and degree of periodontal damage connected to smoking suggests that the host-bacterial interactions usually seen in chronic periodontitis are altered, hence more destroying the periodontal tissue (Preshaw, Chambrone, Holliday, & Ambalavanan, 2024).This discrepancy between the bacterial effect and host response could result from alterations in the subgingival biofilm's structure (such as a rise in the number and their virulence of pathogenic organisms), alterations in the host's reaction to the bacterial challenge, or a combination of the two (Abdulkareem *et al.,* 2023).

Table 1: Smoking's effect on the causes and the pathophysiology of periodontal diseases.(Yixin Zhang, He, He, Huang, & Li, 2019)

Etiologic Factor	Effects of Smoking
Microbiology	Increased microbiome complexity and periodontal pathogen colonization of periodontal pockets.
Immune– inflammatory reaction	Alteration in phagocytosis, oxidative burst, and chemotaxis of neutrophils.
	Gingival crevicular fluid levels of tumor necrosis factor-α and prostaglandin E2 rise.
	Increase the amounts of neutrophil collagenase and elastase in the crevicular fluid of the gingiva.
	Monocyte reaction to lipopolysaccharide with a rise in prostaglandin E2 synthesis.
Physiology	Gingival blood vessels are deteriorating, and inflammation increases.
	Upon probing, gingival crevicular fluid shows reddening and decreased flow; this is coupled with an inflammatory rise.
	Lowering in subgingival temperature.
	Increase the amount of time required to recuperate after receiving local anesthetic.

3.2.1 Microbiology

Several research have investigated the potential alterations in the subgingival microbiota due by smoking. Earlier studies tended to show little difference between smokers and nonsmokers. For example, (Mínguez *et al.,* 2019) took plaque samples from deep pockets (i.e., ≥6 mm) in 142 patients and found no differences in the counts of Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, and Prevotella intermedia.

A comparable investigation of individuals utilizing immunoassay revealed no significant difference in the prevalence of A. actinomycetemcomitans, P. gingivalis, P. intermedia, and Eikenella corrodens between smokers and nonsmokers (Zhong *et al.,* 2020). Conversely, other research has revealed disparities in the microbial composition of subgingival biofilm between smokers and nonsmokers (Jiang *et al.,* 2020). Research including 798 participants with varied smoking histories revealed that smokers had markedly elevated levels of Tannerella forsythia and were 2.3 times more likely to possess T. forsythia compared to nonsmokers and former smokers (Preshaw *et al.,* 2024).

This was accompanied by elevated levels of T. forsythia, A. actinomycetemcomitans, and P. gingivalis in the pockets of the smoking group following therapy, in contrast to the nonsmoking group (Nociti Jr, Casati, & Duarte, 2015). The findings that smokers did not benefit from mechanical treatment as well as nonsmokers were of interest.

Numerous discrepancies in microbiologic study findings arise from methodological factors, including bacterial counts versus proportions or prevalence, the number of sampled sites and selected pocket depths, sampling techniques, the subject's disease status, and the

techniques used of bacterial identification and data analysis (Azzawi & Abdul-Rahman, 2018).

To address some issues, research collected subgingival biofilm from every tooth except third molars in 272 adult participants, comprising 50 current smokers, 98 past smokers, and 124 nonsmokers (Preshaw *et al.*, 2024). Utilizing checkerboard DNA-DNA hybridization technology to examine 29 distinct subgingival species revealed that participants of the red-orange complex species—including Eikenella nodatum, Fusobacterium nucleatum ss vincentii, P. intermedia, Peptostreptococcus micros, Prevotella nigrescens, T. forsythia, P. gingivalis, and Treponema denticola—exhibited significantly greater prevalence in current smokers compared to nonsmokers and former smokers (Teughels *et al.*, 2024).

The heightened incidence of these periodontal pathogens resulted from higher colonization of shallow locations (pocket depth ≤4 mm), with no significant differences comparing smokers, former smokers, and nonsmokers in pockets measuring 4 mm or larger (Sreekumar, 2019). Furthermore, these pathogenic bacteria exhibited greater prevalence in the maxilla compared to the mandible. The studies indicate that smokers exhibit a higher degree of colonization by periodontal pathogens compared to nonsmokers or former smokers, thereby elevating the risk of periodontal disease development (PARTHASARATHY, Jeevitha, & DHARMAN, 2021).

Recent investigations have reached same findings, utilizing the latest-generation sequencing analysis of smokers health periodontium (Hagenfeld *et al.*, 2020). Exhibiting a markedly diverse, pathogen-dense, commensal-deficient, anaerobic microbiome that closely resembles the microbiome found in individuals with advanced periodontitis rather than that of periodontally healthy nonsmokers, and which is predisposed to the future onset of periodontitis contingent upon suitable ecological and environmental alterations (Rafla'a, Jabu, Altaee, & Shwalia, 2019).

3.2.2 Immune-Inflammatory Reactions

The host's immunological response to biofilm formation is fundamentally protective. In periodontal health, there is a balance between the bacterial challenge created by the biofilm and the immune-inflammatory reactions in the gingival tissues, resulting in no loss of periodontal support (Rösing *et al.*, 2019). In contrast, periodontitis is linked to a disruption in the host–bacterial equilibrium, which may be triggered by modifications in the bacterial composition of the subgingival biofilm, alterations in host responses, environmental changes, or a combination thereof (Teughels *et al.*, 2024). Smoking significantly influences the immune-inflammatory response, leading to an escalation in the degree and severity of periodontal damage (Chahal, Chhina, Chhabra, & Chahal, 2017).

The harmful consequences of smoking seem to stem from changes in the immune-inflammatory reaction to bacterial exposure. Neutrophils are crucial to the host's response to bacterial challenges, and changes in their quantity or functionality can lead to localized and systemic infections (Magán-Fernández *et al.*, 2020). The essential tasks of neutrophils encompass chemotaxis (targeted movement from the circulation to the infection site), phagocytosis (the engulfment of foreign particles like bacteria), and elimination by oxidative and nonoxidative processes (Arora & Singh, 2024). Neutrophils derived from the blood vessels in the peripheral zone, oral cavity, or saliva of smokers, or exposed in vitro to entire tobacco smoke or nicotine, exhibit functional modifications in chemotaxis, phagocytosis, and the oxidative burst (N. Ashwini, 2016). In vitro investigations on the impact of tobacco-related items on neutrophils have demonstrated adverse effects on cellular motility and the oxidative burst (White *et al.*, 2018).

Furthermore, the levels of immunoglobulin G2 antibodies, crucial for phagocytosis and bacterial eradication, have been found to be diminished in smokers (Shahoumi, Saleh, & Meghil, 2023). In comparison to nonsmokers with periodontitis, smokers appear to possess less defense against periodontal microorganisms (Moon, Lee, & Lee, 2015). In contrast, increased concentrations of tumor necrosis factor–α have been seen in the gingival crevicular fluid of smokers (Abduljabbar, Akram, Vohra, Warnakulasuriya, & Javed, 2018), along with raised levels of prostaglandin E2, neutrophil elastase, and matrix metalloproteinase-8 (Ghassib, Chen, Zhu, & Wang, 2019). In vitro studies indicate that nicotine exposure enhances the release of prostaglandin E2 by monocytes in reaction to lipopolysaccharide (Han *et al.*, 2021). The observations indicate that smoking modifies neutrophil responses to bacterial challenges, resulting in elevated production of tissue-destructive enzymes and subsequent periodontal tissue loss (VL *et al.*, 2022).

3.2.3 Physiology

Prior research indicates that specific medical signs of inflammation (e.g., gingival erythema, gingival hemorrhage) are less apparent in smokers compared to nonsmokers (Silva, 2021). This may stem from changes in the vascular reactivity of gingival tissues. No substantial variations in the vascular integrity of healthy gingiva have been noted between smokers and nonsmokers(Rifai, Aoun, & Majzoub, 2020); nevertheless, the microcirculatory response to biofilm formation seems to be modified in smokers relative to nonsmokers. In the presence of inflammation, the flow of gingival crevicular fluid, bleeding upon probing, and the vascularity of gingival tissues are diminished in smokers compared to nonsmokers (Kumar, 2019). Moreover, the oxygen content in healthy gingival tissues is lower in smokers compared to nonsmokers, however this condition is inverted in cases of mild inflammation

(Karatas *et al.*, 2020). Subgingival temperatures decreased in smokers compared to nonsmokers and the recovery from vasoconstriction induced by local anesthetic injection is prolonged in smokers (Quaranta, D'Isidoro, Piattelli, Hui, & Perrotti, 2022). The studies indicate that smokers exhibit substantial modifications in the gingival microvasculature relative to nonsmokers, resulting in decreased blood flow and reduced clinical indicators of inflammation (Silva, 2021). This elucidates the long-documented occurrence of a temporary rise in gingival hemorrhage upon smoking cessation; patients should be cautioned of this phenomenon (Ravella, 2023).

3.3 Smoking and gingivitis

Gingivitis is characterized as inflammation of the gum that does not interfere with alveolar bone stability and junctional epithelial attachment integrity (Peruzzo *et al.*, 2016). Gingival bleeding is justified by an objective sign often associated with periodontal diseases, especially gingivitis, often associated with the accumulation of bacterial plaque; other aspects also contribute to an rise in gingival bleeding (SHIN, JONES, & JOHN, 2021).

Controlled clinical trials have shown that, in human specimens of experimental gingivitis, the inflammatory response to plaque buildup is less in smokers relative to nonsmokers (Buduneli & Scott, 2018). Numerous cross-sectional studies suggest that smokers may exhibit reduced gingival inflammation at a specific plaque level compared to non-smokers (Rösing *et al.*, 2019). A study by the WHO Eastern Mediterranean Regional Office revealed that current frequent smokers exhibited more severe gingivitis compared to non-smokers (model 1 beta = 0.54; 95% CI: -0.04 to 1.12)(Moeis, Hartono, Nurhasana, Satrya, & Dartanto, 2024).

Gingivitis Caused by parasites Trichomonas tenax and Entamoeba gingivalis and show infected with E. gingivalis highly ratio when comparative infected with T. tenax (Rafla'a *et al.*, 2019). The rate of GCF flow at rest is produced less in the smokers. The increase in GCF during experimental gingivitis is perhaps less in smokers (Molnár, 2020).

Other studies have shown variations in the oxygen saturation of hemoglobin between smokers and nonsmokers, suggesting functional deficiencies in the microvasculature of those who smoke (Srivastava *et al.*, 2024).

Results of a study show that plaque accumulation was 48 percent in smokers and 55 percent in non-smokers after 28 days. While another analysis of clinical parameters disagreed with this outcome, which was conducted on 20 healthy students, 10 of whom were daily smokers, the rate of plaque formation was comparable in both classes (Jose, 2018)

Table 2: Impact of smoking on gingivitis and periodontics (Leite, Nascimento, Scheutz, & Lopez, 2018)

Periodontal Disease	Effect of Smoking
Gingivitis	↓ Gingival inflammation and bleeding on probing.
Periodontitis	↑ Prevalence and severity of periodontal destruction
	↑ Pocket depth, attachment loss, and bone loss.
	↑ Rate of periodontal destruction
	↑ Prevalence of severe periodontitis
	↑ Tooth loss
	↑ Prevalence with increased number of cigarettes smoked per day
	↓ Prevalence and severity with smoking cessation

Changes in the vascular composition of periodontal tissues, resulting from nicotine-induced vasoconstriction in the gingiva of smokers and pronounced gingival keratinization, have been consistently associated with reduced gingival bleeding and gingival index (GI) in smokers (Ali, Abdullah, & Gaphor, 2015). A study indicate that the prevalence of bleeding sites among smokers (27%) is lower than that among non-smokers (40%) (Tawfik, 2020). There was no consistency of elevated values for smokers or non-smokers concerning Gingival (GR) recession, and no statistically significant alterations were seen in any of the conditions (Gayathri, 2017).

3.4 Smoking and periodontitis

Smoking is a significant risk factor for periodontitis, influencing the prevalence, extent, and severity of the disease. Understanding the effects of smoking on the onset, advancement, and treatment of the disease is crucial (Ziukaite, Slot, Loos, Coucke, & Van der Weijden, 2017).

The predominant form of periodontitis, formerly referred to as adult periodontitis or chronic adult periodontitis, is often characterized as a gradually advancing condition (Kinane, Stathopoulou, & Papapanou, 2017).

The prevalence of chronic periodontitis is comparable in both genders and is associated with aging (Cui *et al.*, 2023). Chronic periodontitis is characterized by inflammation, periodontal pockets, loss of attachment, and bone loss, identified as a "site-specific disease" due to the localized effects of subgingival

plaque deposition, resulting in the formation of pockets and attachment loss. Smoking is a principal and widespread risk factor for chronic periodontitis, with risk assessments suggesting that it is associated with 40% of chronic periodontitis cases (Dommisch *et al.,* 2024)

Microbiological research indicates that bacterial species linked to periodontal disease are more abundant in smokers compared to non-smokers, including Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans, Bacteroides forsythus, Prevotella intermedia, and Fusobacterium nucleatum (Enchiparambil, 2022). The odds ratio for periodontitis among current smokers is estimated to vary from 1.5 to 7.3 compared to nonsmokers, contingent upon the observed severity of periodontitis (Ayub *et al.,* 2023). Older adult smokers are nearly three times more susceptible to severe periodontal disease, with the duration of tobacco use being a critical determinant of tooth loss, coronal root caries, and periodontal disease (Yuqing Zhang, Leveille, & Shi, 2022).

Smoking has been demonstrated to influence the severity of periodontal disease in younger people. Cigarette smoking correlates with heightened severity of generalized aggressive periodontitis in young people, with smokers being 3.8 times more likely to develop periodontitis than nonsmokers (Ehsan, 2025). Longitudinal studies have shown that young people who smoke above 15 cigarettes daily exhibit the greatest risk of tooth loss. Moreover, smokers are about six times more likely than nonsmokers to have persistent connection loss (Alobaidi, Heidari, & Sabbah, 2023). During a decade, bone loss has been documented to occur at double the rate in smokers compared to nonsmokers, and to advance more swiftly even with optimal plaque management (Preshaw *et al.,* 2024).

The incidence of moderate and severe periodontitis, along with the proportion of teeth exhibiting over 5 mm of attachment loss, was highest among current cigarette smokers, while cigar and pipe smokers displayed a disease severity that was intermediate between current cigarette smokers and nonsmokers (Gayathri, 2017). Cigar and pipe smokers experience a higher incidence of tooth loss compared to nonsmokers (Souto, Rovai, Villar, Braga, & Pannuti, 2019). Former smokers have a lower risk of periodontitis compared to current smokers, however a higher risk than nonsmokers, with the risk diminishing as the duration since cessation of smoking increases (Rösing *et al.,* 2019).

This indicates that the adverse effects of smoking on the host may be reversed via smoking cessation, therefore emphasizing that smoking cessation programs should be a fundamental aspect of periodontal education and therapy (Chan, Chan, Chu, & Tsang, 2023).

Non-smokers exhibited elevated total IL-4 levels and decreased IL-8 levels compared to smokers, but plaque buildup in both cohorts significantly augmented total IL-1b and IL-8 levels (Patel, Amirisetty, Kalakonda, Penumatsa, & Koppolu, 2018). While other investigations did not align with prior findings, IL-4 levels were consistent in the smoking group and diminished in the non-smoker group (Kasnak, Ural, Karagülle, & Basegmez, 2024). In a separate study, smokers were categorized according to WHO guidelines based on daily cigarette consumption, indicating that the average concentration of salivary protein in smokers was lower than that in non-smokers (551,486 µg/mL versus 765,361 µg/mL), with the difference not being statistically significant ($P > 0.05$) (Bachtiar, Gusliana, & Bachtiar, 2021). Subsequent correlation studies demonstrated the negative link between protein content and the extent of smoking. Non-smokers had a much higher quantity of salivary albumin (1.1 mg/100 ml) compared to smokers (0.38 mg/100 ml; $P = 0.0274$) (Narayan, 2017). Salivary levels of calcium, magnesium, and phosphorus among smokers were significantly reduced (Alqahtani *et al.,* 2023). Approximately fifty percent of the examined studies linked smoking to occurrences of periodontitis. Moreover, current smokers exhibited a fourfold increased likelihood of developing periodontitis compared to never smokers, with a dose-response relationship observed between daily cigarette use and periodontitis risk (Bunæs, 2017).

4. The Impact of Nicotine Use on Periodontal Treatment:

4.1 Surgical and Non-surgical Therapy:

The diminished responsiveness of periodontal tissues to nonsurgical treatment seen in current smokers is similarly seen following surgical therapy. Over the span of seven years, the degradation in furcation regions was more pronounced in both heavy and light smokers compared to former smokers and nonsmokers (Kocher, Meisel, Biffar, Völzke, & Holtfreter, 2023). Smoking adversely affects the results of directed tissue regeneration and the surgical management of infrabony lesions with bone grafts (Majzoub *et al.,* 2020). A longitudinal comparative study examining the effects of four distinct treatment modalities (coronal scaling, root planning, enhanced Widman flap surgery, and osseous resection surgery) revealed that smokers—classified as "heavy" (≥20 cigarettes/day) and "light" (≤19 cigarettes/day)—exhibited consistently diminished pocket reduction and reduced clinical attachment gain compared to nonsmokers or former smokers (Bhakta, 2024). At 12 months post-guided tissue regeneration treatment for deep infrabony lesions, smokers exhibited fewer than 50% of the attachment gain seen in nonsmokers (2.1 mm vs to 5.2 mm) (Madi, Smith, Alshehri, Zakaria, & Almas, 2023).

In a subsequent investigation, 73 smokers had a reduced attachment gain compared to nonsmokers (1.2 mm against 3.2 mm), increased gingival recession, and

diminished bone filling of the defect. Likewise, following the application of bone grafts for the management of infrabony defects, smokers exhibited a lesser decrease in probing depths relative to nonsmokers (Madi *et al.*, 2023)

Open flap access surgery, devoid of regenerative or grafting techniques, is a prevalent surgical method employed to reach the root and bone surfaces (Zhao *et al.*, 2021). Six months post-procedure, smokers exhibited a markedly lesser reduction in deep pockets (≥7 mm) compared to nonsmokers (3 mm for smokers versus 4 mm for nonsmokers) and a significantly lower gain in clinical attachment (1.8 mm for smokers compared to 2.8 mm for nonsmokers), despite all patients receiving monthly supportive periodontal therapy for six months (Lan, Bulsara, Pant, & Wallace, 2021)

Table 3: Impact of Smoking on Periodontal Therapy Outcomes (Alexandridi, Tsantila, & Pepelassi, 2018)

Therapy	Effect of Smoking
Non-Surgical	Reduction in the clinical reaction to root surface debridement.
	Decrease in probing depth.
	Levels of clinical attachment decrease.
	Deleterious effects of smoking with elevated plaque control levels.
Surgery and Implants	Reduction in probing depth and decrease in clinical attachment levels following access flap surgery.
	Increase furcation deterioration following surgery.
	Decrease in clinical attachment levels, decrease in bone fill, increase in recession, and increase in membrane exposure during directed tissue regeneration.
	Reduction of root coverage following grafting treatments for localized receding gingival.
	Reduction of probing depth following bone graft operations.
	Increased risk of implant failure and peri-implantitis.
Maintenance phase	Supplementing probe depth and attachment loss throughout maintenance treatment.
	Elevate disease return in smokers.
	Heightened necessity for retreatment in smokers.
	Elevate tooth loss among smokers following surgical intervention.

In a study of Chaturvedi (2018)assessed the efficacy of surgical versus nonsurgical therapy for chronic periodontitis treatment. They reported that 12 months post-treatment, surgical therapy yielded a 0.6 mm greater reduction in probing depth and a 0.2 mm greater gain in clinical attachment compared to deep pocket nonsurgical therapy (>6 mm) in pockets measuring 4-6 mm. Additionally, nonsurgical treatment lead to 0.5 mm less clinical attachment loss when compared to surgical therapy, while scaling and root planning produced a 0.4 mm greater clinical attachment gain and a 0.4 mm lesser reduction in probing depth compared to surgical therapy (Y. C. Liu *et al.*, 2025).

4.2 Antimicrobial treatment in smokers

Local antibiotics have demonstrated more effectiveness than systemic antibiotics in smokers with persistent periodontitis (Yusri, Elfana, Elbattawy, & Fawzy El-Sayed, 2021).Treatment of periodontitis with antimicrobial treatment (antimicrobial photodynamic therapy aPDT, and systemic metronidazole MTZ combined with amoxicillin AMX). After 180 days, patients in the MTZ + AMX and aPDT groups had significantly reduced mean probing depths, enhanced clinical attachment level gains, and less bleeding on probing. At 180 days, the MTZ + AMX group had a decrease in the levels of Prevotella nigrescens and Porphyromonas gingivalis, whereas the aPDT group demonstrated a drop solely in Prevotella nigrescens. The use of topical antimicrobials in smokers with chronic periodontitis augmented the efficacy of non-surgical periodontal treatment in improving clinical attachment loss (CAL) and diminishing pocket depth (PD) in locations with PD ≥5 mm prior to surgery (Souza *et al.*, 2020)

The microbiological assessment of these patients indicated that the supplementary application of locally administered doxycycline during scaling and root planing may enhance the eradication of T. forsythensis and P. gingivalis in a larger percentage of areas compared to traditional mechanical treatment (Abu-Ta'a & Bazzar, 2023). These findings offer significant insights for addressing the constraints of periodontal treatment in smokers (Kanmaz, Kanmaz, & Buduneli, 2021)

5. CONCLUSION

According to recent evidence, it can be concluded that cigarette smoking led to more periodontal tissue ruin in both gingivitis and chronic periodontitis in several areas of the oral cavity, and weaker progress can be noticeable in smokers. The results of a variety of studies have recorded the link between high smoking status and raised probing depth was statistically significant.

The association between smoking and BOP, smoking and plaque index was not statistically significant. The fact that smoking is not correlated with

BOP is a sign of decrease of tobacco in the gingival vascular inflammatory reaction. Lack of relationship between smoking and plaque index may be related to age and oral hygiene habits of individuals.

6. AND RECOMMENDATIONS

1. The existing body of research provides very little information on the additional benefits of local and systemic antimicrobials on the decrease of periodontal pocket depth (PPD) and the gain in clinical attachment loss (CAL) following non-surgical periodontal treatment in smokers with chronic periodontitis (CP). The primary advice for future studies is to conduct a multi-center clinical trial that greatly increases the sample size and extends the observation duration beyond 12 months.
2. Clinicians should firmly advise smokers to implement a cessation strategy to prevent the worsening of nicotine-induced periodontal disease.

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