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Effects of Nonsurgical Periodontal Therapy (NSPT) on Periodontitis in Smoker Patients (A Narrative Review)

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Abstract

Periodontitis is a chronic inflammatory disease that affects the supporting structures of the tooth. An individual's smoking status and daily cigarette consumption directly influences periodontitis by increasing severity. Smoking is associated with increased risk of the initiation and progression of periodontal disease. The foundation of periodontal therapy is Non-Surgical Periodontal Therapy (NSPT), comprised of scaling and Root Surface Debridement (RSD), aimed to reducing inflammation and improving clinical parameters such as Periodontal Probing Depth (PPD), Clinical Attachment Level (CAL), Bleeding On Probing (BOP), and Plaque Index (PI). Nevertheless, the effectiveness of NSPT might be affected by several factors, including smoking. The purpose of this review article is to assess the effects of NSPT on periodontitis patients, emphasizing the comparison of the results between smokers and non-smokers. Smokers have a high risk of periodontal disease recurrence and experience negative effects on the outcomes of NSPT. Compared to nonsmoker periodontitis patients, smokers with periodontitis tend to respond less favorable to NSPT.

Introduction:

Dental plaque refers to the aggregation of the microorganisms on the surface of the teeth having extracellular matrix consisting of polymers derived from the bacteria and saliva(1). Dental biofilm, a collective of microorganisms found to inhabit surfaces within the oral cavity, forms a dense colorless film that persists and can reform, especially in the absence of adequate oral hygiene practices. The presence of dental biofilm is common in a healthy mouth, uncontrolled proliferation can cause oral health problems. Bacteria in the biofilm ferment dietary sugars, resulting in acid production that emulsifies tooth enamel and results in cavities, termed dental caries. Furthermore, uncontrolled biofilm can cause gingivitis, which is the first part of the gingival disease that is characterized by inflammation and gingiva bleeding. Untreated gingivitis can be progressed to periodontitis, a serious gingival infection that can lead to damage around the teeth and is also a risk factor of tooth loss (2). While dental biofilm is recognized as the primary etiologic factor in periodontal diseases, the progression and severity of these conditions can be influenced by various risk factors, including smoking, age, gender, systemic diseases, and oral hygiene practices(3). Smoking is a well-documented risk factor for periodontal disease(4). Among systemic conditions, diabetes mellitus has also been identified as a key modifiable risk factor strongly linked to the progression of periodontal disease(5).Periodontal diseases involve the destruction of the supportive structures around teeth, known as the periodontium. Key indicators for periodontal diseases include BOP, the presence of PPD, CAL and radiographically evaluated alveolar bone loss. Without intervention, it can lead to tooth loss; however, most cases are preventable and treatable (6). Periodontal disease are widespread, affecting populations in both developed and developing countries, accounting for roughly 20-50 percent of the global population(3). The high prevalence of periodontal disease across various age groups, including adolescents, adults, and older individuals, numerous risk factors, including smoking, inadequate oral hygiene, diabetes, medication use, age, hereditary factors, and stress, are associated with the development and progression of periodontal disease(3, 7). The new classification of periodontal health was introduced in the 2017 World Workshop held jointly by the European Federation of Periodontology (EFP) and the American Academy of Periodontology (AAP). This definition considers periodontal health to be achieved in the presence of healthy periodontal tissues and the absence of inflammation, the latter assessed clinically through BOP, CAL and PPD scores (8).This new staging system incorporated a unique stages (I-IV) and grades (A-C) system to describe the speed and intensity of disease evolution. Staging criteria assess the degree of bone loss by radiographic or CAL measurements, while grading criteria utilize the patient's age and the degree of bone loss to estimate the rate of disease progression. A new classification system was developed, incorporating separate stages (I-IV) and grades (A-C), allowing for more detailed assessment of the rate and severity of disease progression. Staging criteria assess bone loss using CAL or radiographic measurements, and grading criteria utilize age of the patient and the severity of bone loss to assess rates of disease progression. This new optimization includes an evaluation of existing periodontal status and assessing the risk factors for future disease progression. BOP, CAL and PPD are used to evaluate periodontal health, and the current state is classified as stable, in remission, or unstable (8, 9). The new classification system incorporates aspects of disease severity: the most severe form is stage I grade A, meaning the patient has had mild bone loss and has a slow rate of progression. On the other hand, Stage IV Grade C is the most severe designation, representing a significant amount of loss of alveolar bone, the apical portion of the root and a high rate of progression(10). The aim of this review was to provide up-to-date evidence on the relationship between smoking and nonsurgical periodontal treatment outcomes with focus

on comparing treatment responses in smokers and nonsmokers with periodontitis.

Microbiological effect of smoking

Periodontitis starts when dental plaque accumulates and is colonized by pathogenic bacteria, including *Porphyromonas gingivalis (P.gingivalis), Tannerella forsythia(T.forsythia)*, and *Treponema denticola (T.denticola)*. Smoking has been shown to modify the subgingival microbiome and induce a dysbiotic environment that promotes the outgrowth of periodontal pathogens. Smokers have also been found to harbor higher levels of pathogenic bacteria including *P. gingivalis* and *Aggregatibacter actinomycetemcomitans(A.A)* compared to nonsmokers. These pathogens produce virulence factors that evade the host immune response and exacerbate inflammation(11).

Immunological effect of smoking

During a bacterial infection, the pathogens stimulate the host immune system, leading to the recruitment of immune cells (neutrophils, macrophages, and lymphocytes) within the periodontal tissues. In general, the immune response is identified by the secretion of pro-inflammatory cytokines, including interleukin-1β (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), which enhance tissue destruction and bone resorption(12). A balance between pro-inflammatory and anti-inflammatory responses is vital in influencing disease trajectory. In healthy individuals, the two arms of the immune system work together to traditionally regulate bacterial incursions and bring inflammation to resolution. However, in individuals with periodontitis, this balance is altered, resulting in persistent inflammation and tissue destruction. Periodontitis is influenced by multiple factors such as genetic variables and environmental triggers (such as smoking) that affect immune responses(12, 13).

Smoking has a strong effect on the immune system and aggravates the inflammatory process in periodontitis. Tobacco smoke contains high levels of nicotine, carbon monoxide, and reactive oxygen species (ROS), which interfere with the function of the immune system, including neutrophils, macrophages, and lymphocytes. For instance, smoking has been shown to decrease the phagocytic function of neutrophils and macrophages, thereby impairing the clearance of periodontal pathogens. Additionally, smoking alters the production of cytokines, favoring a pro-inflammatory state that perpetuates tissue destruction(13).

In addition, smoking alters the adaptive immune response by shifting the ratios of T helper (Th) cell subsets. During periodontitis, smoking stimulates a Th2-dominated response that plays a less significant part in controlling bacterial infections than a Th1 response. This immune dysregulation contributes to the persistence of periodontal inflammation and the progression of tissue destruction(14). The effect of smoking on immune system explained in Figure 1.

The effect of smoking on periodontium

Smoking significantly affects the periodontium, comprising the supportive tissues of the teeth. Studies show that smoking independently increases the risk of initiating, worsening, and extending periodontal disease(15). It diminishes the likelihood of successful treatment and influences the development and progression of periodontitis(16, 17). The effects of smoking on periodontium were first documented in 1947, relating necrotizing ulcerative gingivitis to tobacco use(18). In 2017, the acknowledgment of smoking's detrimental effect on periodontal tissues became explicit with the incorporation of this risk factor as a "grade modifier" in the new periodontitis classification scheme. This recognition was prompted by its increase the risk of periodontitis progression by accelerates periodontal tissue destruction, reduced healing response by impairs blood flow, immune response and fibroblast function (19).

Smoking stands out as the most significant modifiable risk factor for periodontal disease due to its impact both locally and systemically on the periodontium. It influences bacterial plaque formation and

the inflammatory response, facilitating the progression to periodontitis. The harmful components of tobacco, including their vasoconstrictive and osteoclastic properties, contribute to tissue destruction and inflammation within the periodontium(20, 21).

Smoking interferes with the normal process of wound healing due to vasoconstrictive nature of tobacco compounds diminishes blood flow to the periodontium, thereby impeding the supply of oxygen and nutrients to the tissues, resulting in delayed recovery and heightened vulnerability to infections The Additionally, Smoking disrupts the immune response, causing dysregulated inflammation and decreased defense against bacterial pathogens (20, 22).

Smoking has been exposed to have a significant effect on alveolar bone loss, with research indicating that tobacco use, including cigarette and water pipe smoking, is associated with an increased risk of periodontal vertical bone loss. Studies have shown a significant association between cigarette smoking and the occurrence and severity of vertical bone defects, with a 2 to 3-fold increase in relative risk (23). Osteoclast cells and the health of the bone, and smoking has a significant effect. Clinical data shows that cigarette smoke exposure stimulates osteoclasts described in vitro and increases bone resorption. Moreover, smoking is also known to negatively affect both osteoblast differentiation and activity by influencing bone mineralization as well as the expression of genes related to the markers of osteoblast differentiation. Chronic exposure to smoke increases the development of osteoclasts and bone resorption gene expression (24).

Cigarette smoke is laden with harmful chemicals that impede the body's natural healing mechanisms. Smoking decreases blood flow by generating carbon monoxide, thereby restricting the transportation of oxygen and nutrients to the wound, which disrupts the usual healing process. Additionally, nicotine diminishes the production of immunoglobulin, a crucial antibody vital in combating infections, and lowers saliva production, resulting in a dry mouth that elevates the risk of bacterial accumulation and infections(25).

Nonsurgical periodontal therapy (NSPT)

The periodontal treatment aimed to maintain a healthy and fully functional periodontium, thus ensuring the preservation of natural teeth. This treatment approach involves motivating and instructing patients on oral hygiene practices, as well as mechanically removing plaque and deposits from both supra- and sub-gingival areas, addressing factors that contribute to plaque retention (such as overhangs), and modifying risk behaviors (such as discontinuing smoking). The treatment of periodontal diseases should primarily focus on mechanical debridement, utilizing either manual or ultrasonic methods, to remove calculus, and biofilm from both supra- and sub-gingival areas (26). The effectiveness of various adjunctive treatment modalities has been rigorously clinically evaluated and researched (27-29).

Smoking and non-surgical periodontal treatment

The efficacy of NSPT can be influenced by smoking status. In nonsmokers, NSPT is highly effective in reducing local and systemic inflammation and restoring immune homeostasis. Through the elimination of bacterial biofilm and calculus, the microbial load is reduced, resulting in a decrease in pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α in the gingival crevicular fluid (GCF)(30). This reduction in inflammation is accompanied by a decrease in immune cell infiltration, particularly neutrophils and macrophages, into the periodontal tissues(30).

NSPT also encourages a shift in the balance between pro-inflammatory and anti-inflammatory mediators. For instance, IL-10 levels, which are considered an anti-inflammatory cytokine, increase after NSPT, which helps with the resolution of inflammation and tissue healing(31). Furthermore, NSPT has been shown to enhance neutrophil and macrophage function, a phenomenon associated with improved pathogen clearance and inflammatory resolution(32).

Though the presence of the colony in nonsmokers was greater than that in smokers, the effects of NSPT on the immune system in smokers appear to be compromised. Smoking decreases the function of immune cells (neutrophils, macrophages, and lymphocytes) which exhibit less reactivity against periodontal pathogens(13). For example, smokers exhibit reduced phagocytic activity of neutrophils and macrophages, which compromises the clearance of bacteria and resolution of inflammation following NSPT(13).

Smoking further shifts the immune response toward a Th2-dominant profile, which is less effective at controlling bacterial infections than a Th1 profile(14). The immune dysregulation aids in the continuation of inflammation and enhanced clinical outcome after NSPT in smokers. Research showed smaller probing depth and clinical attachment loss in smokers compared with nonsmokers following NSPT(33). Moreover, smoking alters the composition of the subgingival microbiome, creating a dysbiotic environment that favors the growth of periodontal pathogens. This dysbiosis further exacerbates immune dysregulation and inflammation, reducing the effectiveness of NSPT(11).

While many studies have shown reduced probing depth reduction and clinical attachment level gains in smokers compared to non-smokers after scaling and root surface debridement (34-36). Others have reported similar outcomes between the two groups (37, 38). Consequently, D'Aiuto et al(39), It was suggested that periodontal issues like interproximal lesions, the number of affected posterior teeth, deep probing depth, and baseline tooth mobility may have a stronger influence on clinical outcomes than smoking status. These conflicting findings could be attributed to methodological differences among studies, including the use of mean full-mouth measurements, variations in follow-up durations (about one year), and inconsistencies in reporting power calculations (34, 35, 40-43).

The latest study tracked Stage III and IV periodontitis patients, classified as current or never smokers, who received NSPT and were observed over a six-month period. The study incorporated biochemical and microbiological analyses alongside clinical periodontal assessments. Findings revealed that both smokers and non-smokers exhibited similar clinical responses to NSPT. However, Gram-negative bacteria repopulated more rapidly in smokers, which could indicate an increased risk of disease recurrence(44).

A contemporary randomized controlled clinical trial with and without adjunctive photodynamic therapy assessed the response of present smokers and non-smokers with unstable periodontitis to scaling and root surface debridement(43), the investigators found that current smokers had poorer outcomes following nonsurgical periodontal treatment, whereas non-smokers responded similarly to mechanical periodontal therapy. Alternative study relating electronic cigarette, non-smokers, and cigarette smokers users described the highest levels of gingival inflammation in cigarette smokers(42, 45). Patient-related factors, including compliance and home care quality, also play a significant role in clinical outcomes after periodontal therapy (46, 47). Additionally, a dose-response link typically exists between the quantity of cigarettes smoked and the adverse health effects of smoking, including periodontitis. A study of nonsmokers, current smokers, and passive smokers found that tobacco has a dose-dependent negative impact on periodontal health (40). The researchers discovered that smokers had a higher prevalence of *T. denticola* and suppressed the inflammation process, which may increase their susceptibility to periodontitis. Furthermore, not all studies include pack-year or other information about smokers. The ability to chemically confirm smoking status is an important factor in studies on the effects of smoking on healthy periodontal tissue; many studies group participants based on self-reported smoking habits, which are not always reliable. Chemical validation, which typically involves measuring cotinine levels in saliva, produces more consistent results. Cotinine, a long-lived primary

metabolite of tobacco smoke, is a reliable biomarker for smoking status when detected in fluids like saliva, urine, and blood (48). Salivary cotinine levels greater than 100 ng/mL are typically indicative of active smoking, whereas concentrations of 5-7 ng/mL indicate passive smoking (49).More studies about the effect of smoking on periodontal outcome after therapy explained in Table 1.

Conclusion

The current review revealed a significant difference in periodontal treatment outcomes between smokers and nonsmokers with periodontitis. Smokers generally exhibit poorer responses to NSPT, including reduced reductions in PPD, less CAL gain, and impaired healing compared to nonsmokers. The harmful effects of smoking such as compromised immune response, impaired tissue repair, and increased microbial virulence contribute to these suboptimal outcomes. These findings highlight the importance of smoking cessation as an essential part of periodontal therapy to improve treatment efficacy and long-term periodontal health.

Conflicts of Interest:

The authors declare that there are no conflicts of interest related to this study

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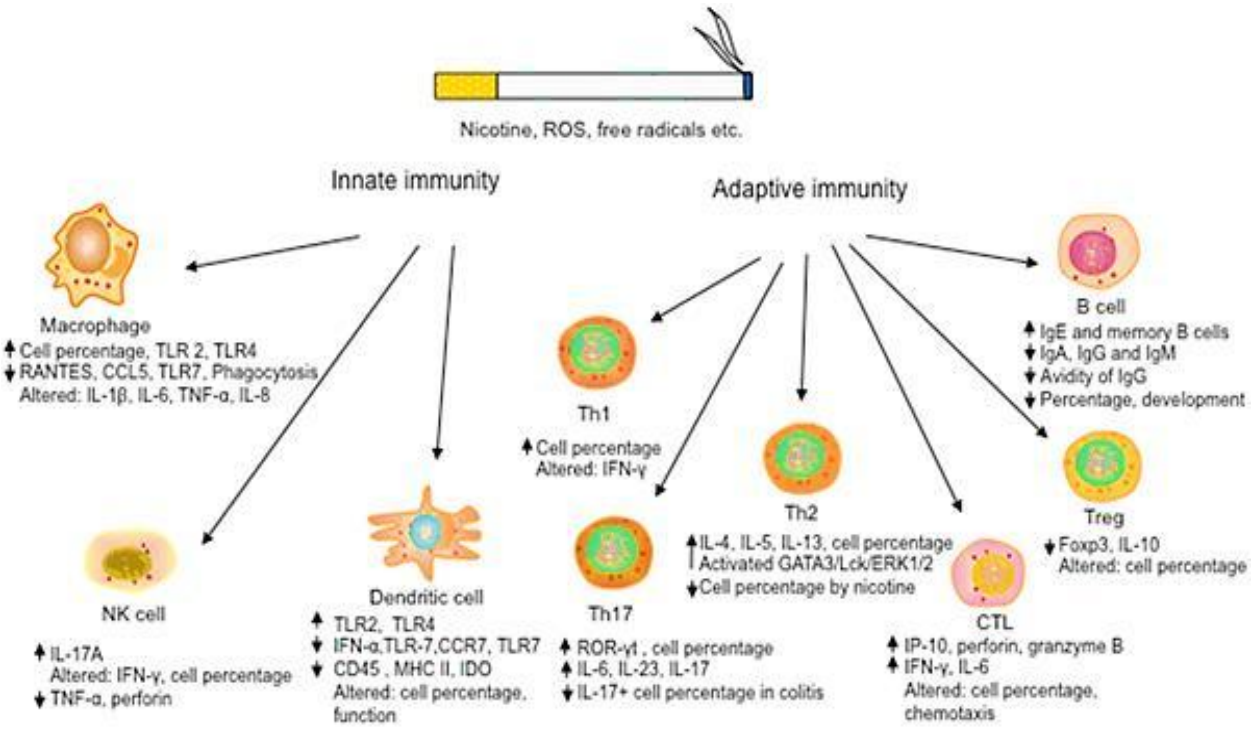

Figure 1: Effects of smoking on the development and function of both innate and adaptive immune cells(50).

Table1 : Overview of studies comparing periodontal treatment outcomes in smokers and nonsmokers with periodontitis

Title	Author, Year.	Type of the study	conclusion
The effect of smoking on bleeding on probing after nonsurgical periodontal therapy: a quasi-experimental study	Ardais R et al.,2014.	A quasi-experimental study	Post-treatment analysis revealed significantly poorer periodontal outcomes (PD reduction and CAL gain) in smokers versus non-smokers after mechanical debridement
Clinical Effects of Nd:YAG Laser Applications During Nonsurgical Periodontal Treatment in Smoking and Nonsmoking Patients with Chronic Periodontitis	Eltas A et al.,2012.	Clinical trial	Although NSPT enhanced periodontal health in both groups, nonsmokers demonstrated superior treatment responses relative to smokers.
Comparison of treatment response patterns following scaling and root planing in smokers and non-smokers with untreated adult periodontitis	Jin L et al.,2000.	Clinical trial	The study demonstrated reduced probing depth reduction and clinical attachment level gains in smokers compared to non-smokers after scaling and root planning.
Outcomes of non-surgical periodontal treatment by dental hygienists in training: impact of site- and patient-level factors	Preshaw P et al.,2013.	Retrospective study	The study reported better periodontal outcomes in non smokers compared to smokers
Does smoking affect gingival crevicular fluid LL-37 levels following non-surgical periodontal treatment in chronic periodontitis?	Türkoğlu O et al.,2016.	Clinical trial	The study reported similar outcomes between the two groups

Effects of smoking on non-surgical periodontal therapy in patients with periodontitis Stage III or IV, and Grade C	Kanmaz B et al.,2020.	Clinical trial	Findings revealed that both smokers and non-smokers exhibited similar clinical responses to NSPT. However, Gram-negative bacteria repopulated more rapidly in smokers, which could indicate an increased risk of disease recurrence.
Effectiveness of scaling and root planning with and without adjunct antimicrobial photodynamic therapy in the treatment of chronic periodontitis among cigarette-smokers and never-smokers.	AlAhmari F et al.,2019.	**A randomized controlled clinical trial**	The study reported smokers had poorer outcomes following nonsurgical periodontal treatment compared to nonsmokers.
Impact of cigarette smoking and vaping on the outcome of full-mouth ultrasonic scaling among patients with gingival inflammation.	ALHarthi SS et al.2019.	A prospective study	The study found that smoking significantly diminishes the therapeutic outcomes of NSPT
Outcomes of Periodontal Therapy in Smokers and Non-smokers with Chronic Periodontitis	Dagmar F. Bunae et al.,2017.	A prospective cohort study	Smokers respond less favorably to non-surgical periodontal therapy compared with non-smokers
The impact of smoking on non-surgical periodontal therapy. A systematic review and meta-analysis	Jennifer Chang et al.,2021.	A systematic review and meta-analysis	Smoking negatively impacts clinical responses to non-surgical periodontal therapy. Smokers with periodontitis have significantly less PD reduction and CAL gain than non-smokers.
The impact of electronic cigarette use on periodontitis and periodontal outcomes. a systematic review and meta-analysis	Muhammed Shabil et al.,2024	A systematic review and meta-analysis	Results indicated that smokers exhibited poorer treatment responses to NSPT compared to non-smokers.

Evaluation of periodontal indices among non-smokers, tobacco, and e-cigarette smokers: a systematic review and network meta-analysis	Paolo Pesce et al., 2022.	A systematic review and meta-analysis	The study demonstrated that smoking compromises NSPT effectiveness.
Smoking negatively impacts the clinical, microbiological, and immunological treatment response of young adults with Grade C periodontitis	Rafaela Videira Clima da Silva et al.,2022.	Clinical trial	The study found that smoking significantly diminishes the therapeutic outcomes of NSPT.
Effects of smoking on healing response to non-surgical periodontal therapy.	Chi Pui Wan et al.,2009	A multilevel modelling analysis	Smokers showed less favorable PPD reduction at deep sites after non-surgical periodontal therapy compared to nonsmokers.
The effects of non-surgical periodontal therapy on oxidant and anti-oxidant status in smokers with chronic periodontitis	Aysun Akpinar et al.,2013.	Clinical trial	The study reported comparable treatment outcomes between the two groups.
Comparison between Smokers and Non-Smokers receiving Non-Surgical Periodontal Therapy in Male Patients at Rehman Medical Institute, Peshawar	Nousheen Daud et al.2024	Clinical trial	Smoking significantly impedes the efficacy of non-surgical periodontal therapy.
Treatment of Chronic Periodontitis with Smoking Cessation Care and Periodontal Surgery in an Elderly Patient: A Case Report Including a 4-year Follow-up	Yamashita and coll.2022	Case report	The study reported reduction in the number of sites with dental plaque in subjects who had quit smoking over a follow-up period of 4-6 weeks

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