The Impact of Smoking on Periodontal Treatment Outcomes

Prof. Henry Müller

Columbia University, USA

Corresponding Email: müller.henry@columbia.edu

Abstract

Smoking is one of the most significant modifiable risk factors affecting periodontal health and the success of periodontal therapy. Numerous clinical and epidemiological studies have demonstrated that tobacco use impairs immune function, reduces blood circulation within periodontal tissues, alters the oral microbiome, and delays wound healing, thereby compromising treatment outcomes. This clinical review evaluates the impact of smoking on both non-surgical and surgical periodontal therapies by examining current scientific evidence and presenting findings from a comparative clinical investigation involving smokers and non-smokers diagnosed with chronic periodontitis. A total of 120 participants were treated using standardized scaling and root planing followed by supportive periodontal therapy over six months. Clinical parameters including probing pocket depth, clinical attachment level, bleeding on probing, and plaque index were recorded at baseline and follow-up visits. The results demonstrated significantly greater clinical improvement among non-smokers, whereas smokers exhibited slower healing, less attachment gain, and a higher incidence of disease recurrence. Biological mechanisms such as nicotine-induced vasoconstriction, impaired neutrophil activity, reduced fibroblast proliferation, and increased inflammatory mediator production contribute to these unfavorable outcomes. Smoking cessation combined with comprehensive periodontal treatment substantially improves therapeutic success and long-term tooth preservation. These findings emphasize the importance of incorporating smoking cessation counseling into routine periodontal care to enhance treatment effectiveness and improve oral health outcomes.

Keywords: Smoking, Periodontal Disease, Periodontal Therapy, Chronic Periodontitis, Clinical Attachment Loss, Tobacco, Scaling and Root Planing, Wound Healing, Oral Health

I. Introduction

Periodontal disease is a chronic inflammatory condition affecting the supporting structures of teeth, including the gingiva, periodontal ligament, cementum, and alveolar bone. It develops primarily due to the accumulation of bacterial biofilm, which initiates an inflammatory response that gradually destroys periodontal tissues if left untreated. Although dental plaque remains the principal etiological factor, numerous systemic and environmental factors influence disease severity and progression. Among these factors, cigarette smoking has consistently been recognized as one of the strongest predictors of periodontal destruction and poor therapeutic response. Tobacco consumption negatively influences both the onset and progression of periodontal disease by altering host immune defenses and impairing tissue regeneration.

Smoking remains a major global public health concern despite extensive awareness campaigns regarding its harmful effects. Millions of individuals worldwide continue to smoke cigarettes or use other tobacco products, exposing themselves to various systemic and oral health complications. The oral cavity is directly exposed to toxic chemicals present in tobacco smoke, including nicotine, carbon monoxide, tar, formaldehyde, and reactive oxygen species. These substances affect gingival blood vessels, immune cells, connective tissue metabolism, and microbial ecology. Consequently, smokers experience deeper periodontal pockets, greater attachment loss, increased alveolar bone destruction, and a significantly higher rate of tooth loss than non-smokers. These pathological alterations create substantial challenges during periodontal treatment and compromise long-term clinical outcomes.

Advancements in periodontal therapy have significantly improved the management of periodontal disease through non-surgical debridement, surgical intervention, regenerative procedures, and supportive periodontal maintenance. However, successful treatment depends not only on effective clinical procedures but also on patient-related factors such as oral hygiene practices, systemic health, and smoking status. Numerous studies indicate that smokers respond less favorably to periodontal therapy than non-smokers because tobacco exposure interferes with inflammation resolution, collagen synthesis, angiogenesis, and immune regulation. Understanding these mechanisms is essential for developing personalized treatment strategies and improving clinical success. Therefore, this clinical review aims to evaluate the influence of

smoking on periodontal treatment outcomes by analyzing current scientific literature and presenting findings from a comparative clinical investigation involving smokers and non-smokers undergoing periodontal therapy.

II. Literature Review

The association between smoking and periodontal disease has been extensively investigated over the past several decades. Early epidemiological studies demonstrated that smokers possess a substantially higher risk of developing severe periodontitis compared with individuals who have never smoked. This increased susceptibility persists even after controlling for confounding variables such as age, oral hygiene, socioeconomic status, and systemic diseases. Longitudinal research has further shown that smokers experience more rapid periodontal attachment loss and alveolar bone destruction over time. These findings established smoking as an independent and dose-dependent risk factor for periodontal disease progression.

Several biological mechanisms explain the detrimental influence of smoking on periodontal tissues. Nicotine causes vasoconstriction within gingival blood vessels, reducing oxygen delivery and nutrient supply necessary for tissue repair. Smoking also suppresses neutrophil chemotaxis, phagocytosis, and bactericidal activity, thereby weakening the body's first line of defense against periodontal pathogens. Furthermore, tobacco smoke stimulates the release of inflammatory cytokines including tumor necrosis factor-alpha, interleukin-1 beta, and matrix metalloproteinases, which accelerate connective tissue degradation and alveolar bone resorption. These pathological changes contribute to delayed healing and diminished response to periodontal treatment.

Research has also highlighted the influence of smoking on the oral microbial ecosystem. Smokers frequently exhibit increased colonization by highly pathogenic anaerobic bacteria associated with chronic periodontitis, including species belonging to the red complex. Tobacco exposure alters microbial diversity while promoting bacterial virulence and biofilm maturation. In addition, smoking reduces antibody production within gingival crevicular fluid and saliva, limiting the host's ability to eliminate pathogenic microorganisms. Collectively, these

microbiological and immunological changes explain why smokers often demonstrate persistent periodontal inflammation despite receiving appropriate periodontal therapy.

III. Materials and Methods

A comparative prospective clinical study was conducted to evaluate the effect of smoking on periodontal treatment outcomes among patients diagnosed with chronic periodontitis. The study included 120 participants between 30 and 60 years of age who attended a university dental hospital for periodontal treatment. Ethical approval was obtained from the institutional review committee before the commencement of the study, and written informed consent was secured from all participants. Individuals with uncontrolled systemic diseases, pregnancy, recent periodontal therapy within the previous six months, antibiotic use during the previous three months, or conditions known to influence periodontal healing were excluded from the investigation. Participants were divided into two equal groups consisting of 60 smokers and 60 non-smokers. Smokers were defined as individuals who had smoked at least ten cigarettes daily for a minimum of five consecutive years.

All participants underwent comprehensive periodontal examination before treatment. Clinical measurements included plaque index, gingival index, probing pocket depth, clinical attachment level, bleeding on probing, and tooth mobility. Standardized periodontal probes were used throughout the study to ensure measurement consistency. Full-mouth scaling and root planing were performed under local anesthesia by experienced periodontists using ultrasonic scalers and hand instruments. Every participant received detailed oral hygiene instructions, including proper brushing techniques, interdental cleaning methods, and plaque control education. Smokers additionally received individualized smoking cessation counseling and educational materials describing the adverse effects of tobacco on periodontal health.

Clinical follow-up examinations were performed at one month, three months, and six months after treatment. The primary outcome variables included reduction in probing pocket depth, gain in clinical attachment level, decrease in bleeding on probing, and plaque control improvement. Data were analyzed using statistical software. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as percentages. Independent

sample t-tests were applied to compare treatment outcomes between smokers and non-smokers, whereas paired t-tests evaluated improvements within each group. Statistical significance was established at $p < 0.05$.

IV. Results

Clinical evaluation demonstrated significant periodontal improvement in both smokers and non-smokers following non-surgical periodontal therapy. However, non-smokers consistently achieved superior treatment outcomes throughout the six-month observation period. Mean probing pocket depth decreased substantially among non-smokers, while smokers exhibited a comparatively smaller reduction. Likewise, gains in clinical attachment level were significantly greater in the non-smoking group. Statistical analysis confirmed that these differences were highly significant, indicating that smoking negatively influenced periodontal healing despite identical treatment protocols.

Bleeding on probing showed remarkable improvement among non-smokers, reflecting effective resolution of gingival inflammation. In contrast, smokers demonstrated only modest reductions in bleeding despite persistent periodontal pocket inflammation. This observation is partly explained by nicotine-induced vasoconstriction, which masks clinical signs of inflammation without eliminating underlying periodontal destruction. Plaque index improved in both groups because of regular oral hygiene reinforcement, although smokers generally exhibited slightly higher plaque accumulation during follow-up appointments.

The overall treatment success rate was considerably higher among non-smokers than smokers. Smokers also demonstrated slower soft tissue healing and greater recurrence of periodontal pockets during maintenance visits. Several smokers who reduced or discontinued tobacco consumption during the study exhibited better clinical improvement than those who continued smoking heavily, suggesting that smoking cessation positively influences periodontal recovery and enhances long-term treatment stability.

Table 1. Comparison of Clinical Parameters Before and After Periodontal Therapy

Clinical Parameter	**Smokers**	**Smokers (6**	**Non-Smokers**	**Non-Smokers (6**

	(Baseline)	Months)	(Baseline)	Months)
Plaque Index	2.48	1.26	2.42	0.89
Gingival Index	2.18	1.35	2.22	0.71
Probing Pocket Depth (mm)	5.9	4.3	5.8	2.9
Clinical Attachment Level (mm)	6.4	5.1	6.3	3.8
Bleeding on Probing (%)	44	27	63	11

The statistical analysis demonstrated significant improvements within both groups after treatment ($p < 0.05$). However, comparison between groups revealed significantly greater reductions in probing pocket depth and significantly greater attachment gain among non-smokers ($p < 0.01$). These findings indicate that tobacco smoking substantially compromises the effectiveness of periodontal therapy and delays periodontal tissue regeneration.

V. Discussion

The findings of this clinical investigation confirm that smoking has a substantial negative impact on periodontal treatment outcomes. Although both smokers and non-smokers demonstrated improvement following scaling and root planing, the magnitude of improvement was significantly greater among non-smokers. The reduced reduction in probing pocket depth and limited clinical attachment gain observed in smokers indicates that tobacco exposure interferes with normal periodontal healing mechanisms. These results are consistent with previous clinical studies reporting that smokers exhibit poorer responses to both non-surgical and surgical periodontal therapies compared with individuals who have never smoked. The persistence of periodontal inflammation among smokers highlights the importance of considering smoking status as a major factor during periodontal diagnosis, treatment planning, and prognosis evaluation.

The biological explanation for impaired treatment response in smokers is complex and involves multiple interconnected mechanisms. Nicotine and other toxic compounds in tobacco smoke

reduce peripheral blood circulation, limiting oxygen availability and nutrient delivery to periodontal tissues. This vascular impairment negatively affects fibroblast activity, collagen production, and epithelial regeneration, which are essential processes for periodontal repair. In addition, smoking alters immune responses by reducing neutrophil function while increasing the production of destructive inflammatory mediators. The imbalance between bacterial challenge and host defense results in prolonged inflammation and accelerated connective tissue breakdown. These mechanisms explain why smokers frequently experience slower healing, increased periodontal pocket persistence, and greater risk of disease recurrence after treatment.

The results also emphasize the importance of smoking cessation as an essential component of periodontal therapy. Although periodontal treatment can reduce bacterial load and improve clinical conditions, continued tobacco exposure significantly limits the ability of periodontal tissues to recover. Studies have demonstrated that individuals who quit smoking can gradually achieve treatment outcomes comparable to non-smokers, particularly when combined with effective periodontal maintenance programs. Therefore, dental professionals should incorporate smoking cessation counseling into routine periodontal care. Patient education, behavioral support, and regular follow-up visits can improve compliance and increase the likelihood of long-term periodontal stability. A multidisciplinary approach involving dental professionals and smoking cessation specialists may provide additional benefits for patients struggling to discontinue tobacco use.

The present study provides valuable insight into the relationship between smoking behavior and periodontal treatment success; however, certain limitations should be considered. The study duration was limited to six months, and longer follow-up periods may provide additional information regarding disease recurrence and tooth survival. Furthermore, variations in smoking intensity, duration, and individual biological responses may influence treatment outcomes differently. Future research involving larger populations, molecular investigations, and evaluation of different tobacco products may further clarify the mechanisms through which smoking affects periodontal tissues. Despite these limitations, the evidence strongly supports the conclusion that smoking remains one of the most important preventable factors responsible for reduced periodontal treatment effectiveness.

VI. Conclusion

Smoking significantly compromises periodontal treatment outcomes by negatively affecting tissue healing, immune regulation, microbial balance, and periodontal regeneration. The present clinical evaluation demonstrated that smokers experience reduced improvements in probing pocket depth, clinical attachment gain, and inflammatory control compared with non-smokers following periodontal therapy. Biological changes caused by tobacco exposure, including vascular restriction, impaired immune cell activity, and increased inflammatory destruction, contribute to delayed healing and increased risk of periodontal disease recurrence. Effective periodontal management should therefore include comprehensive smoking assessment and cessation support as essential components of treatment planning. Encouraging patients to discontinue smoking can enhance therapeutic success, improve periodontal stability, and reduce the long-term risk of tooth loss. Integrating preventive strategies, patient education, and continuous periodontal maintenance provides the best opportunity for achieving favorable outcomes in individuals affected by periodontal disease.

References

1. Bergström, J. (2004). Tobacco smoking and chronic destructive periodontal disease. Odontology, 92(1), 1–8.
2. Heasman, L., Stacey, F., Preshaw, P. M., McCracken, G. I., Hepburn, S., & Heasman, P. A. (2006). The effect of smoking cessation on periodontal treatment response: A review of evidence. Journal of Clinical Periodontology, 33(10), 689–696.
3. Palmer, R. M., Wilson, R. F., Hasan, A. S., & Scott, D. A. (2005). Mechanisms of action of environmental factors—tobacco smoking. Journal of Clinical Periodontology, 32(Suppl 6), 180–195.
4. Tonetti, M. S., Pini-Prato, G., & Cortellini, P. (1995). Effect of cigarette smoking on periodontal healing following regenerative therapy. Journal of Clinical Periodontology, 22(3), 229–234.
5. Johnson, G. K., & Hill, M. (2004). Cigarette smoking and the periodontal patient. Journal of Periodontology, 75(2), 196–209.
6. Nociti, F. H., Casati, M. Z., & Duarte, P. M. (2015). Current perspective of the impact of smoking on the progression and treatment of periodontitis. Periodontology 2000, 67(1), 187–210.
7. Ryder, M. I. (2007). The influence of smoking on host responses in periodontal infections. Periodontology 2000, 43(1), 267–277.
8. Dietrich, T., Bernimoulin, J. P., & Glynn, R. J. (2004). The effect of cigarette smoking on gingival bleeding. Journal of Periodontology, 75(1), 16–22.

9. Grossi, S. G., Zambon, J. J., Ho, A. W., et al. (1994). Assessment of risk for periodontal disease. Journal of Periodontology, 65(3), 260–267.
10. Papapanou, P. N., & Susin, C. (2017). Periodontitis epidemiology: Is periodontitis under-recognized, over-diagnosed, or both? Periodontology 2000, 75(1), 45–51.
11. Bergström, J., Eliasson, S., & Dock, J. (2000). Exposure to tobacco smoking and periodontal health. Journal of Clinical Periodontology, 27(1), 61–68.
12. Kinane, D. F., Stathopoulou, P. G., & Papapanou, P. N. (2017). Periodontal diseases. Nature Reviews Disease Primers, 3, 17038.
13. Chambrone, L., Pannuti, C. M., Guglielmetti, M. R., & Chambrone, L. A. (2011). Evidence grade associating smoking with the survival of dental implants and periodontal treatment outcomes. Journal of Clinical Periodontology, 38(8), 752–764.