



## Smoking and its Effects on Periodontium - An Overview

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### Abstract

The increasing use of multiple medications in chronic disease management has raised concerns about drug–drug interactions (DDIs) and their impact on patient safety. This challenge is particularly significant in health conditions such as diabetes, which often associates with comorbidities like increased blood pressure, hyperlipidemia, and heart ailments. Such health conditions and their comorbidities require complex multi-drug regimens or polypharmacy. Drug–Drug Interaction (DDIs) occurs when one drug interferes with the pharmacodynamic or pharmacokinetic functions of the other drug, potentially leading to severe toxicity or treatment failure. Such interactions are a major contributor to hospital admissions, and the associated risks escalate as the number of medications increases. This research work, presents a clinical decision-support system which analyzes DDI of multi-drug combinations. It also calculates the overall DDI risk, identifying the risk-contributing drug and suggesting its safer therapeutic alternative. By converting DDI interaction analysis into clear and actionable guidance, this system helps in selecting safer drug combinations and supports better clinical decision-making.

**Keywords:** Drug–Drug Interaction (DDIs); Graph Neural Network (GNN)

### Introduction

Smoking is one of the most significant modifiable risk factors affecting periodontal health. It is strongly associated with increased, prevalence, severity, and progression of periodontal diseases. Tobacco use alters host immune response, microbial ecology, and healing mechanisms. These changes ultimately lead to destruction of periodontal tissues. Smokers exhibit a higher risk of periodontitis compared to non-smokers. They also demonstrate poorer clinical outcomes following periodontal therapy [1].

### Classification of smokers

#### Based on exposure

- Current smokers – Individuals who currently smoke tobacco (daily or occasionally).
- Former smokers – Individuals who previously smoked but have quit.
- Never smokers – Individuals who have never smoked in their lifetime.

**Based on frequency**

- Light smokers – < 10 cigarettes per day
- Moderate smokers – 10–20 cigarettes per day
- Heavy smokers – > 20 cigarettes per day

**Based on duration**

- Short-term smokers – Smoking for a short duration (few years)
- Long-term smokers – Smoking for many years [11].

**Composition of Tobacco smoke**

Phase	Components	Effects
Particulate phase(Tar)	Nicotine, tar, polycyclic aromatic hydrocarbons(carcinogens)	Tissue deposition, addiction, carcinogenic effects.
Gaseous phase	Carbon monoxide, carbon dioxide, ammonia, hydrogen cyanide.	Impaired oxygen transport and cellular toxicity [6]

**Table a**

Periodontitis is a chronic inflammatory disease affecting the supporting tissues of the teeth, including the gingiva, periodontal ligament, cementum, and alveolar bone. It is initiated by microbial plaque biofilm and characterized by clinical attachment loss, periodontal pocket formation, and alveolar bone destruction, which may ultimately lead to tooth loss if untreated [12].

**Risk factors of periodontitis**

Periodontitis is influenced by several risk factors that affect the host response and disease progression. Tobacco smoking is one of the most important modifiable risk factors, as it alters immune response, promotes pathogenic bacteria, and impairs periodontal healing. Poor oral hygiene and plaque accumulation are major local factors contributing to periodontal tissue destruction. Additionally, systemic conditions and environmental factors can further increase susceptibility to periodontal disease [2].

**Effects of smoking on periodontal disease**

**Prevalence of Gingivitis and periodontitis**

Smokers may exhibit less gingival bleeding even when inflammation is present, largely due to nicotine causing vasoconstriction. They also tend to have decreased gingival crevicular fluid flow. Despite minimal visible symptoms, plaque

buildup can still occur, which may delay the early detection of gingival disease [8]. At the same time, smoking markedly worsens periodontitis. It is associated with deeper periodontal pockets, greater clinical attachment loss, and increased alveolar bone destruction. Smokers also experience greater tooth mobility and a higher rate of tooth loss. Overall, the progression of periodontal disease is typically more rapid in smokers [4].

**Effects on etiology and pathogenesis of periodontal disease**

**Microbiology**

Smoking changes the composition of subgingival microbial flora, promoting greater colonization by periodontal pathogens such as *Porphyromonas gingivalis*. At the same time, beneficial bacteria are reduced, leading to a shift toward a more pathogenic microbial profile. The biofilm in smokers tends to become more mature and virulent. Additionally, smoking fosters an anaerobic environment that supports the growth of harmful bacteria, thereby accelerating the progression of periodontal disease [10].

**Immunology**

Smoking compromises neutrophil function by impairing chemotaxis, adhesion, and phagocytosis, which weakens the body’s ability to clear periodontal pathogens. It is also associated with elevated levels of pro-inflammatory cytokines such as IL-1β and TNF-α in periodontal tissues. In addition, smoking alters T-lymphocyte activity and disrupts normal immune regulation. It further enhances the activity of tissue-degrading enzymes like matrix metalloproteinases (MMPs), resulting in increased destruction of connective tissue [3].

**Physiology**

Smoking induces vasoconstriction in the gingival blood vessels, leading to reduced blood supply to periodontal tissues and masking typical inflammatory signs like bleeding. It also impairs fibroblast growth and function, causing decreased collagen production along with increased collagen degradation. In addition, smoking elevates oxidative stress and free radical generation. Together, these changes delay wound healing and hinder the repair and regeneration of periodontal tissues [11].

**Effects on response to periodontal therapy**

**Non-surgical therapy**

Smokers tend to respond less favorably to non-surgical periodontal therapy. They exhibit less reductions in probing

pocket depth and limited improvement in clinical attachment levels. Inflammation may continue even after treatment, resulting in generally poorer overall outcomes [7].

### Surgical therapy and implants

Smoking delays wound healing following periodontal surgery. Nicotine interferes with angiogenesis and bone metabolism [9]. Bone regeneration is reduced in smokers with higher prevalence of periimplantitis and implant failures.

### Maintenance therapy

Smokers show increased plaque accumulation during the maintenance phase. Recurrence of periodontal pockets with further attachment loss is observed. Compliance with oral hygiene measures may be poor and hence frequent maintenance visits are required to maintain the periodontal health [5].

### Recurrent therapy

Smoking elevates the likelihood of periodontal disease recurrence. Individuals who smoke often need repeated periodontal interventions, and the condition may continue to progress despite treatment. Overall, smoking sustains an environment that favors ongoing periodontal destruction [4].

### Cessation of smoking

Smoking cessation is an essential component in the management and prevention of periodontal diseases. Tobacco smoking negatively affects periodontal tissues by altering the host immune response, impairing healing, and increasing the severity of periodontal destruction. Therefore, cessation of smoking is strongly recommended for improving periodontal health and enhancing treatment outcomes [11].

### Benefits of smoking cessation

Stopping tobacco use leads to significant improvements in both systemic and oral health. In periodontal tissues, smoking cessation reduces inflammation, improves gingival blood flow, and enhances the host immune response. Former smokers often show better periodontal healing and a more favorable response to periodontal therapy compared to current smokers. Additionally, cessation decreases the risk of disease progression and tooth loss associated with periodontitis [12].

### Approaches to smoking cessation

Effective smoking cessation generally involves behavioral counseling, pharmacological support, and continuous follow-up. Dental professionals play a vital role in motivating patients to quit smoking by educating them about the harmful effects of tobacco on oral tissues [11].

#### Behavioral counseling

Behavioral interventions aim to modify the patient's habits and psychological dependence on tobacco. Dentists can provide brief interventions during dental visits by advising patients about the risks of smoking and encouraging them to quit. Structured approaches such as motivational interviewing and support programs help patients develop strategies to avoid smoking triggers and maintain abstinence [11].

#### Pharmacologic therapy

Pharmacological agents can help reduce withdrawal symptoms and nicotine dependence. Commonly used therapies include nicotine replacement therapy (NRT) in the form of patches, gums, lozenges, inhalers, or nasal sprays. These products supply controlled doses of nicotine without the harmful components of tobacco smoke. Other medications such as bupropion and varenicline may also be prescribed to aid smoking cessation [12].

#### The 5 A's model for smoking cessation

Dental practitioners often use the 5 A's approach to help patients quit smoking:

- **Ask:** Identify and document the patient's smoking status.
- **Advise:** Clearly recommend quitting tobacco use.
- **Assess:** Determine the patient's willingness to quit.
- **Assist:** Provide counseling, educational materials, and pharmacologic therapy if needed.
- **Arrange:** Schedule follow-up visits to support cessation efforts [11].

#### Impact of smoking cessation on periodontal therapy

Patients who quit smoking demonstrate improved clinical outcomes after periodontal therapy compared to those who continue smoking. There is reduced pocket depth, improved attachment levels, and better tissue healing in former smokers.

Long-term cessation also lowers the risk of recurrence of periodontal disease [12].

### Conclusion

Smoking has a significant negative impact on periodontal health. It alters microbial composition, immune response, and tissue physiology. It increases disease severity and accelerates disease progression. It also compromises treatment outcomes. Smoking cessation is essential for improving periodontal health and hence it should be an integral part of periodontal therapy [5].

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