

# AN OVERVIEW ON THE EFFECT OF CIGARETTES SMOKING ON GINGIVAE: LITERATURE REVIEW

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## ABSTRACT

**Background:** Diseases affecting the periodontal tissues (e.g. gingival tissue) contribute to a spectrum of multifaceted pathological entities that present with countless clinical features. Local and systemic factors contribute greatly to the risk of developing periodontal disease.

**Objectives:** The importance of smoking in the development of the periodontal disease, specifically gingival disease.

**Methodology:** We conducted the literature search within the PubMed database using the keywords: “periodontal disease” and “smoking” and “periodontitis” and “gingivitis” and “nicotine” with dates from 1990 to 2020.

**Review:** Smoking has been shown to adversely affect all types of periodontal therapy. Additionally, almost all (90%) of cases with refractory periodontitis are from patients who smoke. Many studies have indicated a great reduction in probing depths and bleeding on probing in nonsmokers, compared to smokers.

**Conclusion:** Dentists are the safety gate and need to be vigilant about the adverse effects of smoking on the health and need to identify potential patients and offer prompt management.

**Key words:** periodontal disease; smoking; periodontitis; gingivitis; nicotine.

## Introduction

Diseases affecting the periodontal tissues (e.g. gingival tissue) contribute to a spectrum of multifaceted pathological entities that present with countless clinical features<sup>1</sup>. Local and systemic factors contribute greatly to the risk of developing periodontal disease. Examples of systemic factors include (1) hormonal imbalance, (2) diabetes, and (3) tobacco smoking<sup>2</sup>. Literature suggests that smoking plays a significant role in the etiology of several periodontal diseases<sup>3-6</sup>. One of the well-studied periodontal diseases that is directly affected by heavy smoking is acute necrotizing ulcerative gingivitis (ANUG)<sup>7</sup>. Data shows that the presentation of gingival disease presents more intensely in the young<sup>1</sup>. Severe gingival inflammation and a higher risk of plaque development have been observed in smokers of the school-age population. A study reported that a higher prevalence of periodontal pockets and bone loss was observed in smokers<sup>8</sup>. The effects of long-term smoking were studied in monozygotic twins and results indicate that smokers had a lower tendency to gingival bleeding<sup>9</sup>. Thus, strong evidence supports the significant role of smoking on

the course of periodontal disease and its influence on the soft and bony component of periodontal tissue<sup>10</sup>. Furthermore, prolonged smoking has been suggested to be an integral part of the etiology of refractory periodontitis. Smokers were at higher risk compared to nonsmokers to experience periodontal breakdown<sup>9,11,12</sup>. Smoking adversely influences regenerative therapy such as osseous grafting, guided tissue regeneration. The importance of smoking in the development of the periodontal disease, specifically gingival disease. Therefore, in this paper, we will review the proper literature discussing the role of smoking in the development of the periodontal disease.

## Methodology:

We conducted the literature search within the PubMed database using the keywords: “periodontal disease” and “smoking” and “periodontitis” and “gingivitis” and “nicotine” with dates from 1990 to 2020. We also used the Google Scholar database for additional literature searches. After reading the abstracts, we manually selected the

relevant papers for this review. In regards to the inclusion criteria, the articles were selected based on the inclusion of one of the following topics; “periodontal disease” and “smoking” and “periodontitis” and “gingivitis” and “nicotine”. Exclusion criteria were all other articles that did not have one of these topics as their primary endpoint.

## Review:

### Biological effects

Despite that the association between periodontal disorders and smoking is well-studied and understood through, any epidemiological experiments, the underlying mechanisms of smoking-induced pathogenesis is not<sup>13</sup>. Table 1 shows the molecules thought to be involved in the pathogenesis of smoking-mediated cellular damage. Studies report that smokers have a significantly higher risk of scoring high on the plaque index couple that with a lower bleeding tendency<sup>9</sup>. Studies that investigate the microbiological element of smoking-induced periodontal disease have revealed that a higher prevalence of bacterial species related to periodontal disease is present in smokers, such as *Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitans*<sup>14,15</sup>.

A recent report that utilized real-time polymerase chain reaction (PCR) was able to establish a positive relationship between the degree of smoking and the amount of bacteria growth<sup>16</sup>. The effect of smoking on experimental gingivitis has also been extensively studied. A recent report demonstrated that the amount of gingival bleeding sites, exudate, and redness were significantly lower in smokers<sup>17</sup>. Other reports showed that the vascular reaction intensity after plaque-induced gingivitis was less than half of that in nonsmokers. A large number of studies have considered smoking to be a risk factor for periodontitis. However, many of those studies do not address the pathological mechanisms involved<sup>9,12</sup>.

### Periodontal management

Smoking has been shown to adversely affect all types of periodontal therapy. Additionally, almost all (90%) of cases with refractory periodontitis are from patients who smoke. Many studies have indicated a great reduction in probing depths and bleeding on probing in nonsmokers, compared to smokers. Also, a higher gain of clinical attachment after surgical management has been observed. However, it has been shown that the use of local and systemic antimicrobial therapy could greatly enhance the management outcomes and overall tissue regeneration in smokers<sup>9,10</sup>.

### Smoking cessation

Stopping smoking has been shown to greatly improve periodontal clinically. This effect is also observed in ex-smokers with no relation to the duration of their smoking<sup>18</sup>. A prompt reversal of gingival inflammation usually occurs within the first weeks after cessation. Then, gingival tissues' thickness starts to decrease gradually with steady reversal of the fibrotic appearance<sup>18</sup>. Within a year, the gingiva regains a normal appearance. Although the health benefits that accompany smoke cessation both on oral and systemic health, many patients fail to achieve it. As patients start to be deprived of nicotine, they start experiencing very distressing symptoms such as mental and physical fatigue, anxiety, inability to concentrate, anger, and an irresistible. Therefore, many patients end up relapsing and fail to maintain sufficient caseation<sup>16</sup>. As a result, multifactorial approaches that take into account the physical, physiologic, and psychological aspects of smoking cessation are developed and implemented<sup>19</sup>. They include psychotherapy, educational leaflets, nicotine chewing gum, and other anti-smoking products. Such modalities could be used to maintain long-term absence<sup>10</sup>. The dental healthcare provider has a vital role in educating patients about the harmful effects of tobacco on periodontal health. They also can recognize early signs of pre-carcinomatous and carcinomatous lesions and suggesting therapeutic approaches. Such efforts need to be continued, despite the negative reactions from the patient<sup>1,13</sup>.

## Conclusion:

Many clinical and epidemiologic reports suggest the supposition that smoking is an imperative risk factor in the occurrence of periodontal diseases. Compared with nonsmokers, smokers show unsatisfactory healing after periodontal interventions. Despite the harmful local and systemic effects of tobacco smoking on periodontal health are well-established, more research is still needed to improve our knowledge of this entire process. Dentists are the safety gate and need to be vigilant about the adverse effects of smoking on the health and need to identify potential patients and offer prompt management.

Interleukins	Non-interleukins
Interleukin-1 $\beta$	Interferon- $\gamma$
Interleukin-1ra	Tumour Necrosis Factor - $\alpha$
Interleukin-6	Metalloproteinases-2 and -8
Interleukin-8	Receptor activator of NF- $\kappa$ B ligand
Interleukin-10	Osteoprotegerin

**Table 1.** Molecules are thought to be involved in the pathogenesis of smoking-mediated cellular damage.

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