THE EFFECTS OF E-CIGARETTES AND TOBACCO PRODUCTS ON PERIODONTAL TISSUES: A MODERN SCIENTIFIC ANALYSIS

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Abstract: Periodontal diseases are considered one of the leading causes of tooth loss worldwide. Their development is influenced by a number of microbiological, immunological, genetic, and environmental factors. In recent years, the widespread use of electronic cigarettes (ECs) and other electronic nicotine delivery systems (ENDS) has increased scientific interest in their effects on the oral cavity and periodontal tissues. While the adverse effects of conventional tobacco smoking on periodontal diseases have been extensively studied, the biological effects of electronic cigarette aerosols represent a relatively new area of research. Recent studies indicate that electronic cigarette aerosols may increase oxidative stress, enhance the expression of inflammatory mediators, alter the composition of the oral microbiota, and exert cytotoxic effects on periodontal ligament fibroblasts and alveolar bone cells. This article analyzes contemporary scientific evidence concerning the effects of tobacco products and electronic cigarettes on periodontal tissues.

Keywords: electronic cigarette, vaping, tobacco, periodontitis, periodontal tissues, nicotine, oxidative stress, oral microbiota, inflammation.

Introduction Periodontal diseases are chronic inflammatory conditions affecting the soft and hard tissues that support the teeth. Although microbial biofilm is the primary etiological factor, disease severity is also determined by the host immune response, genetic susceptibility, nutrition, systemic diseases, and harmful habits. Among these factors, tobacco smoking is recognized as one of the most important modifiable risk factors for periodontal disease. According to the World Health Organization, tobacco use causes millions of deaths worldwide every year, both directly and indirectly. From a dental perspective, tobacco products increase the risk of gingivitis, periodontitis, peri-implant inflammation, oral mucosal diseases, and oral cancer. Therefore, tobacco use is considered one of the most important modifiable risk factors in modern periodontology. Over the past decade, electronic cigarettes have become increasingly popular as a supposedly “safer alternative” to conventional cigarettes. Unlike traditional cigarettes, electronic cigarettes generate an aerosol by heating a nicotine-containing liquid rather than burning tobacco. This liquid usually contains propylene glycol, glycerin, nicotine, flavoring agents, and various chemical additives. During the heating process, carbonyl compounds such as formaldehyde, acetaldehyde, and acrolein, as well as certain heavy metal particles, may be generated. These substances may exert toxic effects on periodontal cells. Although electronic cigarettes were initially considered safer than conventional cigarettes, recent clinical and experimental studies suggest that this assumption is not always valid. Increased periodontal pocket depth, clinical attachment loss, alterations in bleeding parameters, increased oxidative stress, and elevated levels of inflammatory cytokines have been reported among electronic cigarette users. However, for some parameters, their adverse effects may be less pronounced than those observed in conventional cigarette smokers. In addition, electronic cigarette aerosols may alter the ecological balance of the oral microbiota and contribute to an increased proportion of periodontopathogenic microorganisms. This may promote persistent chronic inflammation in periodontal tissues and reduce the effectiveness of periodontal treatment.

Aim of the Study The aim of this study is to analyze the biological effects of electronic cigarettes and conventional tobacco products on periodontal tissues, their inflammatory mechanisms, microbiological alterations, and findings from recent clinical studies, as well as to assess their significance in periodontal practice. Tobacco smoking is considered one of the most important modifiable risk factors for periodontal diseases. According to epidemiological studies, regular tobacco users have a 2–6-fold higher risk of developing periodontitis than non-smokers, and the disease tends to be more severe and less responsive to treatment [1–3]. Tobacco smoke contains more than 7,000 chemicals, at least 70 of which are known to possess carcinogenic properties. The substances with major biological effects on periodontal tissues include nicotine, carbon monoxide, formaldehyde, acrolein, benzene, nitrosamines, cadmium, lead, chromium, and free radicals. These substances contribute to impaired periodontal microcirculation, oxidative stress, cellular apoptosis, and chronic inflammatory processes [4].

1.1. Effects of Nicotine on Periodontal Cells Nicotine is the principal biologically active component of tobacco products and affects periodontal tissues through several mechanisms. First, nicotine causes peripheral vasoconstriction. As a result, blood flow in gingival tissues decreases, oxygen delivery is impaired, and regenerative processes are slowed [5]. Second, nicotine suppresses the proliferation of periodontal ligament fibroblasts. Fibroblasts are essential cells responsible for collagen synthesis and periodontal ligament repair. Impairment of their function disrupts the regeneration of connective tissue [6]. Third, nicotine inhibits osteoblast differentiation and may enhance osteoclast activity. This may contribute to accelerated alveolar bone resorption [7].

1.2. Increased Oxidative Stress Tobacco smoke significantly increases the production of reactive oxygen species (ROS) in periodontal tissues. ROS may lead to lipid peroxidation of cell membranes, DNA damage, mitochondrial dysfunction, protein oxidation, and cellular apoptosis. Under normal physiological conditions, the antioxidant defense system, including superoxide dismutase, catalase, and glutathione peroxidase, neutralizes ROS. However, exposure to tobacco smoke weakens antioxidant defenses and promotes the development of oxidative stress [8].

1.3. Impairment of the Immune Response Tobacco products adversely affect multiple components of both innate and adaptive immunity. Smokers may exhibit impaired neutrophil chemotaxis, reduced phagocytic activity, suppressed macrophage function, decreased T-lymphocyte activity, and reduced levels of secretory immunoglobulin A (IgA). As a result, periodontopathogenic bacteria may persist for longer periods, contributing to the development of chronic inflammation [9]. Although electronic cigarettes do not produce combustion products characteristic of conventional tobacco smoking, their aerosols are not biologically harmless. The main components of e-cigarette liquids include propylene glycol, glycerin, nicotine (which may be absent in some products), flavoring substances, stabilizers, and preservatives. During heating, these components may undergo thermal decomposition and generate formaldehyde, acrolein, acetaldehyde, and other toxic carbonyl compounds [10].

2.1. Cytotoxic Effects on Periodontal Ligament Fibroblasts In vitro studies have shown that electronic cigarette aerosols may significantly reduce the viability of periodontal ligament fibroblasts. The main observed changes include decreased cellular proliferation, impaired migration, reduced collagen synthesis, mitochondrial dysfunction, and increased apoptosis. These changes may potentially delay periodontal tissue regeneration [11].

2.2. Expression of Inflammatory Cytokines Exposure to electronic cigarette aerosols has been associated with increased levels or expression of several inflammatory mediators, including interleukin-1β (IL-1β), interleukin-6 (IL-6), interleukin-8 (IL-8), tumor necrosis factor-alpha (TNF-α),

and prostaglandin E_2. These mediators may prolong inflammatory processes and contribute to the progression of periodontal tissue destruction [12].

2.3. Increased Matrix Metalloproteinase Activity Matrix metalloproteinases (MMPs) play an important role in the pathogenesis of periodontitis. Increased levels or activity of MMP-8, MMP-9, and MMP-13 have been reported in studies involving electronic cigarette exposure [13]. These enzymes contribute to the degradation of collagen fibers and may promote destruction of the periodontal ligament.

2.4. Effects on Alveolar Bone Experimental studies suggest that electronic cigarette aerosols may suppress osteoblast differentiation and increase osteoclast activity. Consequently, they may contribute to decreased bone mineral density, increased alveolar bone resorption, potentially reduced implant success, and delayed regeneration following periodontal treatment [14].

2.5. Alterations in the Oral Microbiota The composition of the oral microbiota in electronic cigarette users differs from that of non-smokers. Periodontopathogenic microorganisms such as Porphyromonas gingivalis, Treponema denticola, Tannerella forsythia, Fusobacterium nucleatum, and Prevotella intermedia have been reported in association with periodontal disease. These microorganisms include members of the “red complex” and other periodontopathogenic groups and play an important role in the development and progression of periodontal diseases [15]. Recent systematic reviews and meta-analyses have demonstrated significant differences in periodontal parameters among conventional cigarette smokers, electronic cigarette users, and non-smokers. According to a large meta-analysis published in 2025, conventional cigarette smokers showed the highest levels of periodontal parameters such as probing depth, clinical attachment loss, and marginal bone loss, whereas electronic cigarette users generally demonstrated intermediate values and non-smokers the lowest levels [16, 17].

3.1. Differences in Clinical Periodontal Parameters Differences among the groups have been observed in several major clinical parameters, including:

- Gingival bleeding on probing (BOP);
- Probing depth (PD);
- Clinical attachment loss (CAL);
- Plaque index (PI);
- Marginal bone loss (MBL).

According to systematic reviews:

Conventional cigarette smokers: highest CAL and MBL values.

Electronic cigarette users: moderate levels of inflammation and alterations in biofilm composition.

Non-smokers: minimal periodontal destruction [13].

A 2025 meta-analysis found that electronic cigarette use was associated with an increased risk of periodontitis; however, the level of periodontal damage was statistically lower than that observed among conventional cigarette smokers [16].

3.2. Inflammatory Mediators and Immune Response Significant differences have also been observed in inflammatory biomarkers, including IL-1β, IL-6, TNF-α, MMP-8, MMP-9, and C-reactive protein (CRP). These markers tend to be highest among conventional cigarette smokers, while electronic cigarette users generally show intermediate increases. This suggests that electronic cigarettes should not be considered completely "safe," although their overall adverse profile may be less severe than that of conventional cigarette smoking [11].

3.3. Oxidative Stress and Cellular Damage Tobacco smoke significantly increases the production of reactive oxygen species (ROS) in periodontal cells. This contributes to lipid peroxidation, DNA damage, mitochondrial dysfunction, and apoptosis. Electronic cigarette aerosols may also generate ROS, although the amount may be lower than that generated by conventional cigarette smoke [15].

3.4. Microbiological Alterations Dysbiosis of the oral microbiota has been observed in both groups, although its extent may differ. Among individuals who use conventional tobacco products, an increased relative abundance of major periodontopathogens such as Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola has been reported. These microorganisms belong to the "red complex," which is considered important in the pathogenesis of periodontitis. Among electronic cigarette users, increased levels of Porphyromonas gingivalis, Prevotella intermedia, and Fusobacterium nucleatum have also been observed, although their abundance is often lower than that found among conventional cigarette smokers. At the same time, a reduction in beneficial commensal microorganisms that contribute to the physiological balance of the oral cavity has been reported in both groups. This represents one of the common pathogenic mechanisms contributing to the development of periodontal dysbiosis [9].

3.5. Clinical Outcomes and Treatment Effectiveness The response to periodontal therapy, particularly scaling and root planing (SRP), also differs among groups:

Non-smokers: best clinical response.

Electronic cigarette users: intermediate response.

Conventional cigarette smokers: poorer response.

Smoking may reduce treatment effectiveness by impairing blood supply and slowing collagen regeneration [16].

Discussion This literature analysis demonstrates that periodontal diseases are complex, multifactorial pathobiological conditions resulting from interactions among microbiological, immunological, genetic, and environmental factors. Tobacco use represents one of the most important modifiable risk factors in this process. It contributes to impaired microcirculation, increased oxidative stress, altered secretion of inflammatory mediators, and impaired immune responses within periodontal tissues. Consequently, destructive changes may develop in the periodontal ligament, alveolar bone, and gingival tissues, leading to a more severe clinical course. Although electronic cigarettes were initially promoted as a safer alternative to conventional tobacco products, recent experimental and clinical studies indicate that they may also adversely affect periodontal health. Nicotine, carbonyl compounds, and other chemical components present in electronic cigarette aerosols may increase oxidative stress in periodontal cells, enhance the expression of inflammatory cytokines, suppress fibroblast proliferation, and disrupt the ecological balance of the oral microbiota. However, based on currently available evidence, the severity of these adverse effects appears to be lower for many clinical parameters than that observed among conventional cigarette smokers. Nevertheless, current scientific evidence does not

support considering electronic cigarettes safe for periodontal tissues. Rather, they should be regarded as one of the factors that may contribute to the development and progression of periodontitis. Therefore, modern periodontology should consider not only individual microorganisms but also the complex interactions among biofilm ecology, host immune responses, and environmental factors when evaluating the biological effects of tobacco and electronic cigarettes.

Conclusion The analysis of the available scientific literature demonstrates that both conventional tobacco products and electronic cigarettes have adverse effects on periodontal tissues. Conventional tobacco products represent one of the most important modifiable risk factors for the development and progression of periodontal diseases. They accelerate periodontal destruction through impaired microcirculation, increased oxidative stress, immune dysfunction, activation of inflammatory mediators, and enhanced alveolar bone resorption. Although electronic cigarettes generally generate fewer toxic combustion-related components than conventional cigarettes, their aerosols may exert cytotoxic effects on periodontal ligament cells and gingival tissues, induce dysbiosis of the oral microbiota, and contribute to persistent inflammatory processes. Current scientific evidence does not provide sufficient grounds for considering electronic cigarettes a safe alternative with regard to periodontal health. Therefore, effective prevention and treatment of periodontal diseases should include reducing or completely eliminating the use of tobacco and nicotine-containing products, improving individual oral hygiene, and identifying modifiable risk factors at an early stage. Future large-scale prospective and randomized clinical studies investigating the long-term effects of electronic cigarettes on periodontal tissues are needed to strengthen the evidence base in this field.

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