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The Impact of Conventional and Alternative Nicotine Delivery Systems on the Pathogenesis of Periodontal Diseases and Periodontal Treatment Outcomes – A Literature Review

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Abstract
Introduction: Periodontal diseases constitute a significant public health concern, and their development and progression are strongly influenced by environmental factors, including nicotine exposure. In recent years, the popularity of alternative nicotine delivery systems, such as electronic cigarettes and heated tobacco products, has increased considerably, necessitating an evaluation of their impact on periodontal tissues.
Aim: The aim of this review was to compare the effects of conventional tobacco smoking and alternative nicotine delivery systems on the pathogenesis of periodontal diseases and on the outcomes of periodontal treatment.
Material and Methods: A narrative literature review covering the years 2020–2025 was conducted using the PubMed, Scopus, and Google Scholar databases. Clinical studies, systematic reviews, and meta-analyses investigating the effects of nicotine on periodontal tissues and periodontal treatment outcomes were included.
Results: Conventional tobacco smoking exerts a strong multidirectional effect on periodontal tissues, leading to microcirculatory disturbances, impairment of the immune response, and exacerbation of inflammatory processes, resulting in disease progression and poorer treatment outcomes. Although alternative nicotine delivery systems eliminate the combustion process, they also demonstrate adverse biological effects, including oxidative stress, microbial dysbiosis, and chronic low-grade inflammation. Their clinical impact appears to be less pronounced, yet still significant.
Conclusions: No form of nicotine delivery is neutral with respect to periodontal health. Patients using both conventional and alternative nicotine products should be regarded as a group at increased risk for periodontal disease. Complete nicotine abstinence remains the most effective strategy for improving periodontal treatment prognosis.

Keywords: periodontitis, smoking, e-cigarettes, heated tobacco products, nicotine delivery systems

1. Introduction

Periodontal diseases are chronic inflammatory conditions with a complex etiopathogenesis, leading to the destruction of the tooth-supporting tissues, including the periodontal ligament and the alveolar bone (Al-Aali et al. 2020). According to data from the Global Burden of Disease Study, severe periodontitis affects approximately 10–15% of the adult population, corresponding to nearly 700–800 million people worldwide, which places it among the most common chronic diseases (Holliday et al. 2021). Its development results from an imbalance between bacterial biofilm and the host immune response, modulated by numerous local and systemic factors. Among these, tobacco smoking is of particular importance and is considered one of the most significant modifiable risk factors influencing the initiation, progression, and treatment effectiveness of periodontitis (Calciolari et al. 2022, de Molon et al. 2026).

Overall, various forms of periodontal diseases affect a much larger proportion of the population, exceeding 50% of adults. Current analyses of the global burden of periodontal diseases indicate that their epidemiological significance is likely to persist in the coming decades, particularly due to population ageing and inequalities in access to dental prevention and treatment (Hu et al. 2021). The prevalence of periodontal diseases increases with age, reaching the highest values in groups over 60 years of age, which is associated with the cumulative exposure to risk factors and the chronic, often asymptomatic course of the disease. Socioeconomic factors are also of considerable importance, as higher incidence and more severe disease progression are observed in populations with lower economic status and limited access to dental care. Moreover, periodontal diseases show significant geographical variation, reflecting differences in lifestyle, prevention, and the prevalence of risk factors such as tobacco smoking and diabetes.

In recent years, significant changes have been observed in patterns of nicotine consumption, associated with the growing popularity of alternative nicotine delivery systems, including electronic nicotine delivery systems (ENDS), such as electronic cigarettes, and tobacco heating systems (THS) (Ebersole et al. 2017, Ganesan et al. 2020). These products are often perceived as less harmful than conventional cigarettes, mainly because they eliminate the combustion process. The literature also emphasizes that the increasing prevalence of ENDS and THS use among dental patients requires these products to be considered as a distinct form of environmental exposure, whose potential relevance for oral health remains the subject of ongoing research (Camoni et al. 2023). Nevertheless, they deliver nicotine and other biologically active substances whose effects on oral tissues have not yet been clearly determined.

Available data indicate that both tobacco smoke and aerosol generated by ENDS may affect the oral microbiome, modulate the immune response, and influence inflammatory and regenerative processes within the periodontium. However, findings regarding alternative forms of nicotine delivery remain heterogeneous, and their interpretation is complicated by methodological differences, short observation periods, and the limited number of long-term studies. In the context of the growing number of users of these products and their potential impact on oral health, a critical summary of the current state of knowledge is particularly important. Analysis of the available literature may contribute to a better understanding of the mechanisms through which nicotine delivered by different routes affects periodontal tissues and to the assessment of its clinical significance, both in relation to disease development and periodontal treatment outcomes (Leite et al. 2019, Global burden of diseases study 2019).

The aim of this literature review is to provide a synthetic evaluation of current scientific reports concerning the effects of conventional tobacco smoking and alternative nicotine delivery systems on periodontal tissues, with particular emphasis on pathophysiological mechanisms, microbiological changes, and clinical implications.

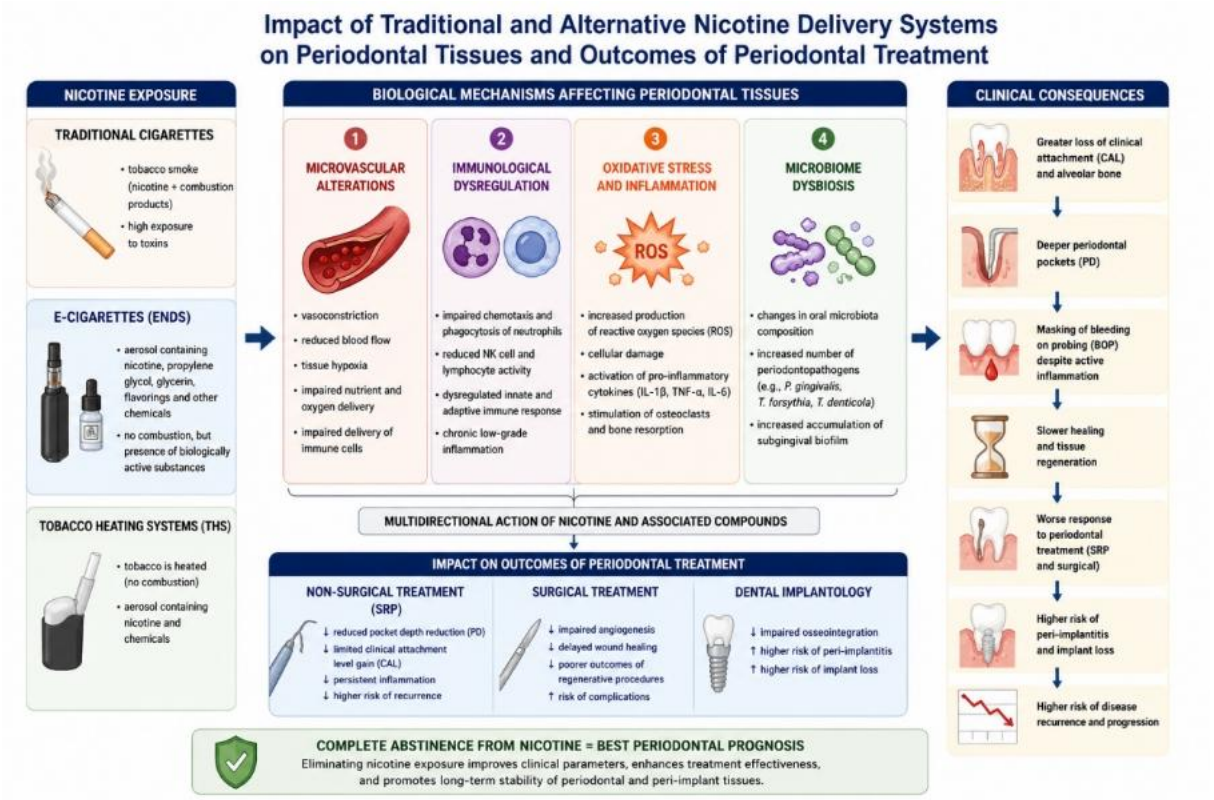

Figure 1. Impact of Traditional and Alternative Nicotine Delivery Systems on Periodontal Tissues and Outcomes of Periodontal Treatment.

2. Materials and Methods

A review of the scientific literature available in the PubMed, Scopus, and Google Scholar databases was conducted. The search strategy included publications identified using the following keywords: *periodontitis, smoking, e-cigarettes, heated tobacco products,* and *nicotine delivery systems*. Articles published primarily between 2020 and 2025 were included, with particular emphasis on systematic reviews, meta-analyses, and clinical studies.
Studies investigating the effects of nicotine on periodontal tissues and periodontal treatment outcomes were included in the analysis. Single case reports and studies of low methodological quality were excluded.

3. The Impact of Conventional and Alternative Nicotine Delivery Systems on Periodontal Tissues

3.1. Conventional Smoking – Pathogenetic Mechanisms and Clinical Presentation

Conventional tobacco smoking is one of the most extensively documented risk factors for periodontal diseases, influencing their development and progression through multiple

biological mechanisms. A key effect of nicotine is the induction of vasoconstriction within periodontal tissues, leading to reduced perfusion, chronic hypoxia, and impaired cellular metabolism. Consequently, regenerative processes are disrupted and tissue healing capacity is diminished (Leite et al. 2019, Calciolari et al. 2022).
Simultaneously, significant dysfunction of the immune system is observed. Smokers exhibit impaired chemotaxis, phagocytosis, and neutrophil activity, resulting in a reduced ability to eliminate pathogens present within the subgingival biofilm. In addition, both innate and adaptive immune responses become compromised, promoting a chronic inflammatory process while simultaneously masking its clinical manifestations (Al-Aali et al. 2020).
An important component of the pathogenic process is the increased production of pro-inflammatory cytokines, such as IL-1β and TNF-α, which stimulate osteoclastic activity and accelerate periodontal tissue destruction. Tobacco smoking also enhances oxidative stress, leading to cellular damage and further intensification of inflammatory and degenerative processes (Global burden of diseases study 2019, Calciolari et al. 2022).
From a clinical perspective, smokers often demonstrate the paradoxical phenomenon of reduced gingival bleeding despite advanced periodontal destruction. This finding is primarily attributable to the vasoconstrictive effect of nicotine rather than to lower disease activity. Greater clinical attachment loss, deeper periodontal pockets, and more rapid disease progression are also characteristic findings. Furthermore, smoking significantly worsens periodontal treatment outcomes, both surgical and non-surgical, due to impaired healing and persistent immune dysfunction (Charde et al. 2024).

3.2. Alternative Nicotine Delivery Systems – E-Cigarettes and Heated Tobacco Systems

Alternative nicotine delivery systems, such as electronic nicotine delivery systems (ENDS) and heated tobacco systems (THS), were introduced as potentially less harmful forms of nicotine exposure, primarily due to the elimination of the combustion process. Nevertheless, their biological effects on oral tissues are not neutral. Studies indicate that aerosols generated by e-cigarettes may induce oxidative stress, enhance inflammatory responses, and alter the composition of the oral microbiome (Global burden of diseases study 2019, Ganesan et al. 2020, Apatzidou 2022).
Microbiological imbalance with a shift toward periopathogenic bacterial species has been observed, which may contribute to the development and progression of periodontal diseases. Additionally, alterations in immune cell function have been reported, including impairment of protective inflammatory responses and disturbances in the production of immunological mediators. As a result, despite reduced exposure to combustion-derived toxins, mechanisms promoting chronic low-grade inflammation remain active (Karaaslan et al. 2020, Apatzidou 2022).
From a clinical standpoint, e-cigarette users may present with signs of gingival inflammation, increased dental plaque accumulation, and potential deterioration of periodontal parameters, although generally to a lesser extent than conventional smokers. Preliminary data also suggest a possible partial impact on periodontal treatment response; however, currently available studies are limited by methodological constraints and require further validation.

Heated tobacco systems may demonstrate a somewhat more favorable profile compared with conventional cigarette smoking in selected cases; however, they still deliver nicotine and other potentially biologically active substances, precluding their classification as safe for periodontal tissues.

3.3. Comparison of Effects and Clinical Consequences

Both conventional and alternative nicotine delivery systems exert adverse effects on periodontal tissues; however, they differ in the intensity and scope of the underlying pathophysiological mechanisms. Conventional smoking is associated with more severe vascular, immunological, and structural damage, leading to more rapid disease progression and poorer treatment outcomes.

Although alternative systems eliminate the combustion process, they continue to induce oxidative stress, microbiome dysbiosis, and alterations in immune responses, indicating that they cannot be regarded as biologically neutral alternatives.

From a clinical perspective, patients using any form of nicotine delivery system should be considered a group at increased risk for periodontal disease. Such individuals require intensified periodontal monitoring, more frequent follow-up visits, and targeted health education. Nevertheless, complete elimination of nicotine exposure remains the key component of management and the most important factor in improving the prognosis of periodontal treatment (Tab. 1).

Table 1. Biological mechanisms associated with traditional cigarette smoking and e-cigarette

Biological mechanism	Traditional cigarettes	E-cigarettes	Clinical effect
Vasoconstriction	Strong	Moderate	Reduced bleeding (masking effect), tissue hypoxia
Oxidative stress	Very high	High	Damage to periodontal tissues
Inflammatory cytokines (IL-1β, TNF-α)	↑↑↑	↑↑	Chronic inflammatory state
Microbiome alterations	Severely disrupted	Disrupted	Increased prevalence of periopathogenic bacteria
Immune response	Impaired	Partially impaired	Faster disease progression
Tissue healing	Significantly impaired	Moderately impaired	Poorer treatment outcomes
Saliva production / pH	Altered	Altered	Promotion of biofilm formation

use and their clinical effects on periodontal tissues.

4. The Impact of Nicotine Exposure on Periodontal Treatment

4.1. Non-Surgical Treatment (Scaling and Root Planing – SRP)

Contemporary periodontal treatment is based on the guidelines of the European Federation of Periodontology, which emphasize the importance of eliminating risk factors, including tobacco smoking (Leite et al. 2018). Conventional tobacco smoking significantly worsens the outcomes of non-surgical periodontal therapy, including scaling and root planing (SRP). Smoking patients demonstrate smaller reductions in periodontal pocket depth (PD) and limited improvement in clinical attachment level (CAL) compared with non-smokers.

The mechanism underlying this phenomenon is multifactorial and primarily involves impairment of the inflammatory response and disturbances in wound healing processes. Through its vasoconstrictive effects, nicotine reduces blood flow within periodontal tissues, leading to diminished delivery of oxygen, nutrients, and immune cells to the treated area. Consequently, reparative processes occur more slowly and with lower efficiency (Sanz et al. 2020).

Additionally, smokers exhibit impaired immune responses, including reduced neutrophil function and decreased ability to eliminate bacterial biofilm. This results in persistent inflammatory activity despite the mechanical removal of subgingival deposits, thereby limiting the effectiveness of SRP therapy (Tab. 2).

Table 2. Key differences in clinical response to non-surgical periodontal treatment in smokers and non-smokers.

Clinical parameter	Non-smokers	Smokers	Clinical significance
Reduction in pocket depth (PD)	Greater	Smaller	Poorer elimination of pathological pockets
Improvement in clinical attachment level (CAL)	Significant	Limited	Weaker connective tissue attachment regeneration
Bleeding on probing (BOP)	More frequent	Reduced	Masking of active inflammation
Inflammatory response	Normal	Weakened	Persistent chronic inflammation
Tissue healing	Effective	Impaired	Prolonged regeneration time
Biofilm elimination	More effective	Less effective	Higher risk of disease recurrence

4.2. Surgical Treatment

Patients who smoke also demonstrate significantly poorer outcomes following surgical treatment of periodontitis. Wound healing after flap surgery and regenerative procedures is impaired, resulting in slower tissue regeneration and a higher risk of postoperative complications.

The principal mechanism responsible for this phenomenon is impaired angiogenesis accompanied by dysfunction of fibroblasts and osteoblasts. Reduced blood flow and chronic hypoxia limit the migration of reparative cells and slow collagen synthesis, directly affecting the quality of the healing process (Leite et al. 2019, Sanz et al. 2020).

As a consequence, smokers exhibit a higher frequency of incomplete tissue regeneration, poorer outcomes of regenerative procedures, and an increased risk of disease recurrence in surgically treated areas.

4.3. Dental Implantology

Tobacco smoking also represents a significant risk factor in dental implantology. Smokers demonstrate an increased risk of complications, including peri-implantitis, impaired osseointegration, and implant loss (Windael et al. 2020, Shabil et al. 2024).

These mechanisms are associated both with microcirculatory disturbances and with the negative effects of nicotine on osteoblast activity and the balance between bone formation and resorption processes. In addition, smoking promotes microbiological dysbiosis within peri-implant tissues, thereby increasing the risk of chronic inflammatory conditions around implants.

Consequently, implant survival rates are lower in smokers, while the risk of infectious and inflammatory complications is significantly higher compared with non-smokers.

4.4. Alternative Nicotine Delivery Systems and Periodontal Treatment Outcomes

Data regarding the effects of alternative nicotine delivery systems, such as e-cigarettes and heated tobacco systems, on periodontal treatment outcomes remain limited and inconclusive.
Available studies suggest that e-cigarette users may exhibit a poorer treatment response compared with non-smokers; however, this effect appears to be less pronounced than in conventional smokers. Moderate impairment of clinical parameters, such as periodontal pocket depth and inflammatory indices, has been observed, which may indicate a partially preserved yet abnormal healing response (Donos et al. 2020, Karaaslan et al. 2020).
The mechanisms underlying this phenomenon primarily involve oxidative stress, alterations in the oral microbiome, and dysfunction of immune cells. Although long-term evidence remains insufficient, current data suggest that alternative nicotine delivery systems are not biologically neutral and may adversely affect the course and outcomes of periodontal treatment (Tab. 3).

Table 3. Detailed comparison of clinical parameters after SRP treatment in e-cigarette users versus non-smokers.

Clinical parameter	Non-smokers	E-cigarette users	Clinical significance
Reduction in pocket depth (PD)	Greater	Moderate	Partially effective pocket elimination
Improvement in clinical attachment level (CAL)	Significant	Limited	Possible impairment of attachment regeneration
Bleeding on probing (BOP)	Physiological	Often increased	Absence of inflammation masking effect
Inflammatory response	Normal	Chronic, low-grade	Persistent inflammation
Tissue healing	Effective	Partially impaired	Possible delayed regeneration
Biofilm elimination	More effective	Less effective	Increased risk of pathogen persistence

5. Discussion

The present literature review confirms that nicotine exposure, regardless of the mode of delivery, constitutes a significant factor affecting both the pathogenesis of periodontal diseases and the effectiveness of periodontal treatment (Calciolari et al. 2022, de Molon et al. 2026). The strongest evidence concerns conventional tobacco smoking, which, through complex biological mechanisms, leads to poorer therapeutic outcomes and accelerated destruction of periodontal tissues.

In the context of non-surgical therapy, findings from prospective studies are particularly relevant, demonstrating that the degree of exposure to tobacco smoke directly correlates with the effectiveness of scaling and root planing (SRP) therapy (Sanz et al. 2020). Smokers have been shown to achieve significantly smaller reductions in periodontal pocket depth and more limited clinical attachment gain compared with non-smokers, with this effect demonstrating a dose-dependent relationship. Similarly, more recent analyses indicate that smoking significantly reduces the likelihood of periodontal pocket closure following non-surgical treatment, which directly affects the long-term stability of therapeutic outcomes.

The mechanisms responsible for these phenomena are consistent with previous observations regarding the effects of nicotine on microcirculation and immune responses. Tissue hypoxia, impaired angiogenesis, and dysfunction of immune cells lead to incomplete resolution of inflammation and maintenance of an environment conducive to disease progression. It is noteworthy that similar associations have also been observed in other areas of dentistry, where smoking significantly delays tissue healing following restorative and endodontic treatment.

An important aspect analyzed in recent studies is the attempt to compensate for the negative effects of smoking through adjunctive therapies. Meta-analyses indicate that the use of additional therapeutic approaches, such as local antibiotic therapy, statins, or photodynamic therapy, may partially improve clinical parameters (PD, CAL) compared with SRP alone. Nevertheless, even with adjunctive treatment modalities, therapeutic outcomes in smokers remain inferior to those observed in individuals not exposed to nicotine, emphasizing the fundamental importance of eliminating the risk factor itself.

With regard to surgical treatment and implantology, the literature consistently demonstrates impaired healing and regenerative processes in smoking patients. Reduced angiogenesis, decreased fibroblast activity, and disturbances in bone metabolism translate into lower efficacy of regenerative procedures and increased risk of complications, including peri-implantitis and implant loss. These findings are particularly significant in the context of the growing number of implant procedures performed in populations burdened with multiple risk factors.

In recent years, increasing attention has been directed toward the impact of alternative nicotine delivery systems on periodontal treatment outcomes. Although data in this area remain limited, available studies suggest that e-cigarette users exhibit an intermediate therapeutic response profile—poorer than that of non-smokers, yet potentially more favorable than that observed in conventional smokers. The mechanisms underlying these differences

include reduced exposure to combustion-derived products while maintaining oxidative stress and microbiological disturbances (Ganesan et al. 2020, Karaaslan et al. 2020).
From a public health perspective, studies concerning smoking cessation are of particular importance, as they indicate that elimination of nicotine exposure leads to significant improvement in periodontal parameters and reduction in inflammatory burden. These findings confirm that the adverse effects of smoking are, to a considerable extent, reversible, thereby highlighting the importance of behavioral interventions as an integral component of periodontal therapy.
However, several limitations of the available studies should be acknowledged. Many analyses are based on short-term follow-up and heterogeneous study populations, while the lack of standardization regarding the definition of alternative nicotine product users complicates comparison of results. Furthermore, differences in nicotine exposure intensity and the coexistence of other risk factors, such as systemic diseases, may influence interpretation of findings. Another important limitation is the heterogeneity of device types and duration of use, which restricts the possibility of definitive assessment of their clinical impact (McDonough et al. 2021, Thiem et al. 2023).
Despite the growing popularity of alternative nicotine delivery systems, the current state of knowledge does not support considering them safe from the perspective of periodontal health. This underscores the need for caution when formulating clinical recommendations and reinforces the importance of complete nicotine abstinence as the primary therapeutic goal.
In summary, available evidence clearly indicates that nicotine exposure is a significant modifying factor affecting the course and outcomes of periodontal treatment. Although alternative nicotine delivery systems may exert a potentially less pronounced effect than conventional smoking, they are not biologically neutral. Therefore, elimination of nicotine exposure and individualized treatment planning for patients belonging to high-risk groups remain key components of periodontal management (Ralho et al. 2019, Yang et al. 2020).

6. Conclusions

The review of current literature demonstrates a dynamically evolving profile of risk factors in contemporary periodontology, in which nicotine exposure continues to play a central role. Conventional tobacco smoking remains the strongest modifiable risk factor influencing the development and progression of periodontal diseases, exerting a well-documented multidirectional impact on microcirculation, immune responses, and tissue regenerative capacity.
At the same time, alternative nicotine delivery systems, including electronic cigarettes, despite eliminating some toxic combustion-derived products, are not biologically neutral. Their use is associated with distinct pathophysiological mechanisms, such as disruption of microbiological balance, enhancement of oxidative stress, and persistence of chronic low-grade inflammation, all of which may contribute to periodontal tissue damage.
Available evidence suggests that heated tobacco systems may, in selected cases, represent an element of harm-reduction strategies in patients strongly dependent on nicotine, potentially exerting a more favorable effect on the response to non-surgical treatment compared with

continued conventional smoking. However, it should be emphasized that current evidence in this field remains limited and does not allow for definitive clinical recommendations.
From the perspective of dental practice, patients using alternative nicotine delivery systems should not be classified as non-smokers, but rather as a distinct risk group requiring intensified periodontal supervision, more frequent follow-up visits, and targeted health education. Clinically, this necessitates routine assessment of all forms of nicotine exposure during medical history taking and individualization of periodontal treatment planning.
In light of the available evidence, it should be clearly stated that no form of nicotine delivery is neutral with respect to periodontal tissues. Complete nicotine abstinence remains the most effective strategy for fully preserving the regenerative potential of periodontal tissues and achieving long-term stability of periodontal treatment outcomes. Further well-designed prospective studies with long-term follow-up are necessary to fully evaluate the impact of emerging nicotine technologies on periodontal health.

Disclosure

Author Contributions:

Conceptualization: KG

Methodology: WZ

Formal analysis: WC

Investigation: WP

Data curation: KB

Writing – original draft preparation: KG, WZ, WC

Writing – review and editing: WP, KB, AJ, GC

Supervision: AJ

Project administration: GC

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Declaration of the use of generative AI and AI-assisted technologies in the writing process.

During the preparation of this manuscript, the authors used ChatGPT (OpenAI) exclusively for language editing, grammar correction, and translation support. Following the use of this

tool, the authors carefully reviewed and edited the manuscript and take full responsibility for the final content of the publication.

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