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The Impact of Smoking on the Oral and Cardiopulmonary Systems: Bridging Local and Systemic Pathologies

Eduarda Rossi Pinheiro[1], Vitória Rossi Pinheiro[2], Bianca Raquel Marques[3], Renato Felipe Pereira [4], José Augusto Pereira da Cruz [5]

[1]*University Center North of São Paulo (UNORTE), São José do Rio Preto, SP, Brazil.*
[2]*University Center North of São Paulo (UNORTE), São José do Rio Preto, SP, Brazil.*
[3]*University Center North of São Paulo (UNORTE), São José do Rio Preto, SP, Brazil.*
[4]*Department of Basic Sciences, São Paulo State University (UNESP), School of Dentistry, Araçatuba, SP, Brazil.*
[5]*University Center North of São Paulo (UNORTE), São José do Rio Preto, SP, Brazil*

Abstract:
Tobacco smoking remains a leading modifiable risk factor for numerous systemic and oral diseases, with substantial global health consequences. This review highlights the interconnected mechanisms through which smoking influences oral homeostasis, cardiovascular function, and pulmonary health. Nicotine and tobacco-derived toxins disrupt gingival microcirculation and promote oral microbial dysbiosis, facilitating periodontal disease progression. Chronic periodontal inflammation subsequently contributes to systemic inflammatory burden, endothelial dysfunction, and atherosclerosis, thereby elevating cardiovascular risk in smokers. Additionally, smoking-induced alterations in the oral microbiome may extend to the lower respiratory tract, exacerbating chronic pulmonary diseases such as COPD. Understanding the complex oral-pulmonary-cardiovascular axis is critical for developing comprehensive strategies aimed at reducing the burden of smoking-related diseases. This article focuses specifically on the role of smoking in cardiovascular and pulmonary pathologies, emphasizing the need for integrated approaches that address oral health as part of systemic disease management.

Key Word*: Cardiovascular system; Oral disease; Respiratory system; Systemic disease; Smoking.*

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I. Introduction

Tobacco smoking remains one of the most important modifiable risk factors for both oral and systemic diseases worldwide. With approximately 1.3 billion tobacco users half of whom die from tobacco-related causes and more than 8 million deaths each year, the impact of smoking on health is substantial and transcends traditional boundaries [1]. While the deleterious cardiovascular and pulmonary effects of tobacco smoking are well documented, its role in disrupting oral homeostasis ranging from mucosal alterations to periodontal degradation has garnered increasing attention. These early oral manifestations not only compromise local tissues but may also serve as harbingers of systemic pathology [2,3].

Smoking induces a spectrum of biological alterations in oral tissues, mediated primarily by nicotine, carbon monoxide, and polycyclic aromatic hydrocarbons [4]. Nicotine's vasoconstrictive action reduces gingival blood flow and impairs oxygen delivery, while carcinogens disrupt epithelial integrity and modulate cellular apoptosis and fibroblast function. Moreover, smoking reshapes the oral microbiome by fostering colonization of pathogenic species, elevating biofilm virulence, and destabilizing the oral microbial ecosystem [5].

Evidence indicates that smoking is the leading environmental risk factor for periodontal disease [6] tripling the risk of alveolar bone loss, gingival recession, and pocket formation while also promoting mucosal lesions such as leukoplakia and oral cancer. Furthermore, smoking exacerbates aesthetic and functional deterioration through tooth discoloration, halitosis, candidiasis, and increased risk of tooth loss [7].

These local alterations in the oral environment do not remain confined to the mouth; rather, they reflect and contribute to broader systemic processes. Smoking-induced immunosuppression and microbiome dysbiosis can worsen diseases such as diabetes mellitus, osteopenia/osteoporosis, and HIV-associated oral infections [8]. Though the direct mechanisms often involve complex immune-modulating and microbial pathways, they underscore the need to view oral pathologies not in isolation, but as components of multifaceted systemic interactions [2].

Building upon this connection between oral and systemic health, the pathophysiological mechanisms induced by smoking extend even further. Smoking catalyzes endothelial dysfunction, inflammation, hypercoagulability, insulin resistance, and dyslipidemia—all pivotal to atherothrombosis and vascular disease [9]. In parallel, smoking-induced oral dysbiosis and chronic periodontal inflammation may act as reservoirs of pathogenic bacteria, which can be aspirated into the lower respiratory tract, potentially contributing to the development or exacerbation of chronic respiratory diseases such as COPD and emphysema [10]. Considering that periodontal inflammation may contribute to systemic inflammatory load, our review investigates the hypothesis that oral disease acts as both a marker and mediator of cardiopulmonary risk in smokers

II. Materials and methods

This study is a narrative literature review based on the selection and critical analysis of scientific articles addressing the effects of smoking on oral health and its systemic repercussions, particularly concerning cardiovascular and pulmonary diseases. The search for publications was conducted in recognized electronic databases, including PubMed, Scielo, LILACS, Embase, and Google Scholar.

The search strategy employed specific descriptors such as “smoking,” “tobacco,” “oral health,” “periodontal disease,” “cardiovascular diseases,” “pulmonary diseases,” “systemic effects,” and “oral manifestations.” Inclusion criteria comprised full-text articles available in open access, published in English, and directly addressing the relationship between smoking, oral diseases, and their connections with systemic pathologies.

Each selected article underwent a detailed analysis of its objectives, methodology, results, and discussion to ensure a critical appraisal of the available scientific evidence. This systematic and analytical approach allowed for the development of an up-to-date and comprehensive review on the proposed topic.

III. Results and Discussion

Mechanisms of Action of Tobacco on Oral Tissues

Tobacco smoke introduces a host of biologically active compounds such as nicotine, carbon monoxide, and polycyclic aromatic hydrocarbons that disrupt oral tissue integrity and function. Nicotine induces vasoconstriction, reducing gingival blood flow and impairing oxygen and nutrient delivery to periodontal tissues [11]. Chronic exposure leads to microvascular dysfunction characterized by increased capillary tortuosity, decreased perfusion, and reduced angiogenic response, which persists even after smoking cessation [3]. Acute effects include initial transient hyperemia, but repeated insults result in long-term ischemia and impaired healing capacity [13].

In addition to vascular changes, nicotine and other smoke toxins directly impair host immune defenses. Nicotine decreases leukocyte chemotaxis, phagocytic ability, and antibody production, which diminishes both innate and adaptive immune responses within the gingiva [14]. Smoke exposure also elevates systemic and local inflammatory markers, such as sICAM 1, CRP, and acute phase proteins, contributing to a persistent yet blunted inflammatory profile that suppresses normal tissue repair while promoting chronic pathology [15,16].

A growing body of evidence highlights how smoking alters the oral microbiome—a shift marked by a reduction in microbial diversity and enrichment of pathogenic species including *Streptococcus, Porphyromonas, Prevotella*, and *Veillonella* [17, 18]. This dysbiosis fosters biofilm virulence, increases tissue inflammation, and creates a microenvironment conducive to periodontal disease. The oral ecosystem in smokers converges toward pathogen dominance and impaired resilience, setting the stage for more aggressive microbial-driven tissue destruction [17].

From the cellular perspective, tobacco constituent exposure disrupts epithelial and connective tissue cell functions. Nicotine and polycyclic hydrocarbons induce apoptosis in gingival fibroblasts and epithelial cells, suppress fibroblast proliferation, and hinder extracellular matrix synthesis. They also impair osteoblastic activity and angiogenesis crucial processes for periodontal regeneration and bone maintenance [3,19]. These combined vascular, immune, microbial, and cellular dysfunctions underlie the structural and functional decline characteristic of periodontal tissue in smokers.

Key Oral Pathologies Associated with Tobacco Use

Tobacco use is strongly associated with the development and progression of several oral diseases, with periodontitis being one of the most extensively studied [2]. Clinical evidence shows that smokers have greater alveolar bone loss, deeper periodontal pockets, and a higher prevalence of gingival recession compared to non-smokers [20]. The vasoconstrictive action of nicotine, reduced gingival blood flow, and suppressed inflammatory response contribute to masking clinical signs of inflammation, which can delay diagnosis of periodontal disease [3].

In addition to periodontitis, tobacco use is closely linked to oral mucosal lesions such as leukoplakia, a white lesion considered potentially malignant. Smokers have a significantly higher risk of developing oral

leukoplakia, especially in areas of direct contact with smoke, such as the buccal mucosa and floor of the mouth [21]. Studies also show a higher malignant transformation rate of these lesions into squamous cell carcinoma among chronic smokers [22].

Another common condition among smokers is oral candidiasis. The altered oral environment caused by smoking including reduced pH, impaired local immunity, and microbiome disruption favors colonization by Candida species, especially Candida albicans. This is further exacerbated by tobacco-induced xerostomia, which compromises the protective functions of saliva. Immunocompromised individuals, such as those with HIV, are at even greater risk when they smoke [23,24].

Lastly, smoking is associated with both aesthetic and functional changes, such as halitosis, extrinsic dental staining, delayed wound healing after oral surgery, and a higher rate of dental implant failure [25]. Studies indicate that tobacco products impair osseointegration and increase the risk of peri-implantitis, making the restorative prognosis less favorable in smokers [26].

Interactions between Smoking, Systemic Diseases, and Oral Manifestations: Focus on Cardiovascular and Pulmonary Diseases

Smoking exerts a significant influence not only on oral health but also on various systemic conditions that present with oral manifestations. Among the most relevant are diabetes mellitus, HIV infection, cardiovascular diseases, and chronic respiratory diseases, all potentially modulated or exacerbated by tobacco exposure [27]. The interaction between these pathologies and smoking is bidirectional: smoking can worsen the prognosis of systemic disease while simultaneously amplifying its oral repercussions [3]. In this article, we will focus on relationship between smoking and its impact on cardiovascular and pulmonary diseases.

Cardiovascular Diseases: The Role of Inflammation and Endothelial Dysfunction

Smoking is one of the main modifiable risk factors for the development of cardiovascular diseases such as atherosclerosis, coronary artery disease, and stroke. Chronic exposure to cigarette smoke triggers a systemic inflammatory process characterized by endothelial cell activation, increased pro-inflammatory cytokines (such as IL-6 and TNF-α), elevated oxidative stress markers, and promotion of endothelial dysfunction. These mechanisms contribute to the accumulation of atheromatous plaques and favor a hypercoagulable state, increasing the risk of thrombotic events [28].

These systemic alterations are closely linked to chronic periodontal inflammation, which shares similar mediators, including prostaglandins, matrix metalloproteinases, and reactive oxygen species [20]. Periodontitis may therefore not only reflect but also contribute to an increased systemic inflammatory burden, establishing a feedback loop between oral health and cardiovascular risk [29]. Multiple studies associate severe periodontitis in smokers with a higher incidence of cardiovascular events, suggesting that the oral cavity can serve as a reservoir of inflammation with measurable systemic impact [29,30,31].

Beyond inflammatory effects, smoking also directly affects vascular function by reducing the bioavailability of nitric oxide, a potent vasodilator and regulator of vascular homeostasis [28]. The decrease in nitric oxide contributes to vasoconstriction, increased blood pressure, and enhanced adhesion of inflammatory cells to the endothelium, accelerating the atherosclerotic process [32]. In the oral context, this vascular dysfunction can impair gingival microcirculation, compromising local immune responses and tissue repair capacity, which favors periodontitis progression in smokers [3]. Therefore, the impact of smoking on the cardiovascular system and oral health is interconnected not only through systemic inflammation but also through localized vascular dysfunction.

Pulmonary Diseases: Relationship with the Oral Microbiome and Mucosal Immunity

Smoking is the primary risk factor for chronic pulmonary diseases such as Chronic Obstructive Pulmonary Disease (COPD), chronic bronchitis, and emphysema [33]. Cigarette smoke compromises the immune barriers of the airways, reduces mucociliary clearance, promotes alterations in innate and adaptive immune responses, and generates an imbalance in the respiratory microbiota. Recent studies indicate that this imbalance may partly originate from colonization by pathogenic oral microorganisms migrating to the lungs, a process facilitated by saliva aspiration or disruption of oral barriers [34,35].

Smoking-induced oral dysbiosis—characterized by an increase in periodontopathogenic species and a decrease in commensal microorganisms—may therefore play a role in exacerbating respiratory diseases [8]. Additionally, patients with COPD often present concomitant periodontal disease, with evidence suggesting that periodontitis severity correlates with the degree of pulmonary impairment [36]. These observations support the hypothesis of an "oral-pulmonary" axis, wherein inflammation and dysfunctional oral microbiota directly impact lung health.

Beyond microbiological and immunological alterations, smoking promotes structural remodeling of the airways, such as bronchial wall thickening and alveolar destruction, which impair gas exchange and favor

emphysema progression [37]. These changes are exacerbated by chronic inflammatory responses generated by interactions between altered microbiota and compromised immune defenses [38]. In the oral cavity, the persistent presence of pathogens may serve as a continuous source of antigens and endotoxins, amplifying both systemic and local inflammation, and potentially contributing to pulmonary function deterioration [33]. Therefore, the clinical management of smokers with pulmonary disease should consider oral health as an important component for controlling and preventing respiratory complications.

IV. Conclusion

Tobacco smoking exerts a profound and multifaceted impact on both oral and systemic health, intricately linking oral pathologies with cardiovascular and pulmonary diseases. The chronic inflammatory and immune-modulatory effects induced by smoking create a vicious cycle wherein oral dysbiosis and periodontal inflammation not only reflect but also amplify systemic endothelial dysfunction and respiratory impairment. This bidirectional relationship underscores the importance of integrating oral health into the broader clinical management of smoking-related cardiopulmonary conditions. Future research and clinical interventions should focus on this oral-systemic axis to better prevent and mitigate the significant morbidity and mortality associated with tobacco use.

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References

[1]. WORLD HEALTH ORGANIZATION. Tobacco. Geneva: World Health Organization, 2023. Available at: https://www.who.int/news-room/fact-sheets/detail/tobacco. Accessed on: June 12, 2025.
[2]. Johnson N, Bain C. Tobacco and oral disease. In: British Dental Journal. British Dental Association, London, p. 200–206, 2000.
[3]. SILVA, H. Tobacco use and periodontal disease – the role of microvascular dysfunction. Biology (Basel), v. 10, n. 5, p. 441, 2021.
[4]. Javed F, Rahman I, Romanos GE. Tobacco-product usage as a risk factor for dental implants. In: Periodontology 2000. Wiley, Hoboken, p. 48–56, 2019.
[5]. JOHNSON, G.K.; HILL, M. Cigarette smoking and the periodontal patient. J Periodontol, v. 75, p. 196–209, 2004.
[6]. NETO, J.B.C.; ROSA, E.F.; PANNUTI, C.M.; ROMITO, G.A. Smoking and periodontal tissues: a review. Braz Oral Res, v. 26 (Suppl 1), p. S117–S125, 2012.
[7]. Guiglia R, Musciotto A, Compilato D, Procaccini M, Lo Russo L, Ciavarella D, Lo Muzio L, Cannone V, Pepe I, D'Angelo M, Campisi G. Aging and oral health: effects in hard and soft tissues. In: Current Pharmaceutical Design. Bentham Science Publishers, p. 619–630, 2010.
[8]. Rajasekaran JJ, Krishnamurthy HK, Bosco J, Jayaraman V, Krishna K, Wang T, Bei K. Oral microbiome: a review of its impact on oral and systemic health. In: Microorganisms. MDPI, Basel, p. 1797, 2024.
[9]. MESSNER, B.; BERNHARD, D. Smoking and cardiovascular disease: mechanisms of endothelial dysfunction and early atherogenesis. Arterioscler Thromb Vasc Biol, v. 34, p. 509–515, 2014.
[10]. Bansal M, Khatri M, Taneja V. Potential role of periodontal infection in respiratory diseases – a review. In: J Med Life. Carol Davila University Press, Bucharest, p. 244–248, 2013.
[11]. SHARMA, K.; KAUR, A.; KOONER, K. The impact of smoking on periodontal health: a comprehensive review. Indian J Clin Anat Physiol, v. 11, n. 3, p. 138–145, 2024.
[12]. SILVA, H. Tobacco use and periodontal disease – the role of microvascular dysfunction. Biology (Basel), v. 10, n. 5, p. 441, 2021.
[13]. Qandil R, Sandhu HS, Matthews DC. Tobacco smoking and periodontal diseases. In: Journal of the Canadian Dental Association. Canadian Dental Association, Ottawa, p. 187–195, 1997.
[14]. Qiu F, Liang C-L, Liu H, Zeng Y-Q, Hou S, Huang S, Lai X, Dai Z. Impacts of cigarette smoking on immune responsiveness: up and down or upside down? In: Oncotarget. Impact Journals, New York, p. 268–284, 2016.
[15]. Palmer RM, Scott DA, Meekin TN, Poston RN, Odell EW, Wilson RF. Potential mechanisms of susceptibility to periodontitis in tobacco smokers. In: J Periodontal Res. Blackwell Munksgaard, Copenhagen, p. 363–369, 1999.
[16]. Clark JD III, Wilkinson JD, LeBlanc WG, Dietz NA, Arheart KL, Fleming LE, Lee DJ. Inflammatory markers and secondhand tobacco smoke exposure among U.S. workers. In: American Journal of Industrial Medicine. Wiley, Hoboken, p. 626–632, 2008.
[17]. Mohammed LI, Razali R, Zakaria ZZ, Benslimane FM, Cyprian F, Al-Asmakh M. Smoking induced salivary microbiome dysbiosis and is correlated with lipid biomarkers. In: BMC Oral Health. Springer Nature, London, p. 608, 2024.
[18]. Tamashiro R, Strange L, Schnackenberg K, Santos J, Gadalla H, Zhao L, Li EC, Hill E, Hill B, Sidhu GS, Kirst M, Walker C, Wang GP. Smoking-induced subgingival dysbiosis precedes clinical signs of periodontal disease. In: Scientific Reports. Springer Nature, London, p. 3755, 2023.
[19]. Arredondo J, Hall LL, Ndoye A, Nguyen VT, Chernyavsky AI, Bercovich D, Orr-Urtreger A, Beaudet AL, Grando SA. Central role of fibroblast α3 nicotinic acetylcholine receptor in mediating cutaneous effects of nicotine. In: Laboratory Investigation. Nature Publishing Group, New York, p. 207–225, 2003.
[20]. Sreedevi, M., Ramesh, A., & Dwarakanath, C. Periodontal status in smokers and nonsmokers: a clinical, microbiological, and histopathological study. In: International Journal of Dentistry, Hindawi Publishing Corporation, London, p. 571590, 2012.
[21]. Khanna, D., Shruti, T., Tiwari, M., Sharma, P., Khan, A., Ranjan, S., Balasundaram, P., Khargekar, N., Chaturvedi, P., & Mishra, A. Prevalence of oral potentially malignant lesions, tobacco use, and effect of cessation strategies among solid waste management workers in Northern India: a pre-post intervention study. In: BMC Oral Health, BioMed Central, London, v. 24, p. 1292, 2024.

[22]. Tan, Y., Wang, Z., Xu, M., Li, B., Huang, Z., Qin, S., Nice, E.C., Tang, J., & Huang, C. Oral squamous cell carcinomas: state of the field and emerging directions. In: International Journal of Oral Science, Springer Nature, London, v. 15, p. 44, 2023.
[23]. Tamai, R., & Kiyoura, Y. Candida infections: the role of saliva in oral health—A narrative review. In: Microorganisms, MDPI, Basel, v. 13, n. 4, p. 717, 2025.
[24]. Heron, S. E., & Elahi, S. HIV infection and compromised mucosal immunity: oral manifestations and systemic inflammation. In: Frontiers in Immunology, Frontiers Media, Lausanne, v. 8, p. 241, 2017.
[25]. Tubaishat, R. S., Malkawi, Z. A., & Albashaireh, Z. S. The influence of different factors on the oral health status of smoking and nonsmoking adults. In: The Journal of Contemporary Dental Practice, Jaypee Brothers Medical Publishers, New Delhi, v. 14, n. 4, p. 731–737, 2013.
[26]. Badenes-Catalán, J., & Pallarés-Sabater, A. Influence of smoking on dental implant osseointegration: A radiofrequency analysis of 194 implants. In: Journal of Oral Implantology, American Academy of Implant Dentistry, New York, v. 47, n. 2, p. 110–117, 2021.
[27]. Gajendra, S., McIntosh, S., & Ghosh, S. Effects of tobacco product use on oral health and the role of oral healthcare providers in cessation: a narrative review. In: Tobacco Induced Diseases, Multidisciplinary Digital Publishing Institute (MDPI), v. 21, p. 12, 2023.
[28]. Jia, X., Zhang, P., Meng, L., Tang, W., & Peng, F. The association between smoking exposure and endothelial function evaluated using flow-mediated dilation values: a meta-analysis. In: BMC Cardiovascular Disorders, BioMed Central, v. 24, p. 292, 2024.
[29]. Petrenya, N., Hopstock, L. A., Holde, G. E., Oscarson, N., & Jönsson, B. Relationship between periodontitis and risk of cardiovascular disease: insights from the Tromsø Study. In: Journal of Periodontology, Wiley, v. 93, p. 1353–1365, 2022.
[30]. Khouja, T., Miller, R. G., Moore, P. A., Orchard, T. J., & Costacou, T. Periodontal disease, smoking, cardiovascular complications and mortality in type 1 diabetes. In: Journal of Diabetes Complications, Elsevier, v. 33, p. 603–609, 2019.
[31]. Schulze-Späte, U., Wurschi, L., van der Vorst, E. P. C., Hölzle, F., Craveiro, R. B., Wolf, M., & Noels, H. Crosstalk between periodontitis and cardiovascular risk. In: Frontiers in Immunology, Frontiers Media, Lausanne, v. 15, p. 1469077, 2024.
[32]. Theofilis, P., Sagris, M., Oikonomou, E., Antonopoulos, A. S., Siasos, G., Tsioufis, C., & Tousoulis, D. Inflammatory mechanisms contributing to endothelial dysfunction. In: Biomedicines, MDPI, Basel, v. 9, p. 781, 2021.
[33]. Sethi, J. M., & Rochester, C. L. Smoking and chronic obstructive pulmonary disease. In: Clinics in Chest Medicine, Elsevier, Philadelphia, v. 21, p. 67–86, 2000.
[34]. Li, R., Li, J., & Zhou, X. Lung microbiome: new insights into the pathogenesis of respiratory diseases. In: Signal Transduction and Targeted Therapy, Springer Nature, London, v. 9, art. 19, 2024.
[35]. Huang, C., & Shi, G. Smoking and microbiome in oral, airway, gut and some systemic diseases. In: Journal of Translational Medicine, BioMed Central, London, v. 17, p. 225, 2019.
[36]. Lin, P., Liu, A., Tsuchiya, Y., Noritake, K., Ohsugi, Y., Toyoshima, K., Tsukahara, Y., Shiba, T., Nitta, H., Aoki, A., Iwata, T., & Katagiri, S. Association between periodontal disease and chronic obstructive pulmonary disease. In: Japanese Dental Science Review, Elsevier, Amsterdam, v. 59, p. 389–402, 2023.
[37]. Lin, H., & Li, H. How does cigarette smoking affect airway remodeling in asthmatics? In: Tobacco Induced Diseases, International Society for the Prevention of Tobacco Induced Diseases, Athens, v. 21, p. 13, 2023.
[38]. Rajasekaran, J. J., Krishnamurthy, H. K., Bosco, J., Jayaraman, V., Krishna, K., Wang, T., & Bei, K. Oral microbiome: a review of its impact on oral and systemic health. In: Microorganisms, MDPI, Basel, v. 12, p. 1797, 2024.