



The impact of oral microbiota on the development of atherosclerosis: an integrative review

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ABSTRACT

Introduction: Atherosclerosis is a chronic inflammatory disease of the arterial wall and the leading cause of cardiovascular mortality worldwide. The oral microbiota, the second largest microbial community of the human body, has been increasingly implicated in systemic inflammatory processes that may influence cardiovascular health.

Objectives: To analyse the impact of the oral microbiota on the development of atherosclerosis, exploring the mechanisms by which oral dysbiosis may contribute to systemic inflammation and to the progression of cardiovascular disease.

Methodology: An integrative literature review was conducted following the PRISMA recommendations, using the PubMed, SciELO and ScienceDirect databases. The search strategy was based on the PECO model, combining the terms "oral microbiota", "atherosclerosis", "cardiovascular disease" and "systemic inflammation". Studies published between 2015 and 2025 were considered, and pre-defined inclusion and exclusion criteria were applied.

Results: Eighteen articles were included. The evidence showed a consistent association between alterations in the oral microbiota and atherosclerosis. Bacteria associated with periodontal disease can enter the bloodstream and contribute to systemic inflammation; oral microorganisms have been identified within atherosclerotic plaques; and chronic inflammation related to oral dysbiosis favours endothelial dysfunction and lesion progression. Shared risk factors - smoking, diabetes and poor oral hygiene - were also relevant.

Conclusion: The evidence suggests a potential relationship between oral microbiota dysbiosis and atherosclerosis, reinforcing oral health as an integral part of systemic health. Effective prevention and control of oral infections may contribute to reducing cardiovascular risk and support integrated approaches between dentistry and cardiovascular medicine.

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RESUMO

Introdução: A aterosclerose é uma doença inflamatória crónica da parede arterial e a principal causa de mortalidade cardiovascular a nível mundial. A microbiota oral tem sido cada vez mais associada a processos inflamatórios sistémicos com potencial impacto na saúde cardiovascular.

Objetivos: Analisar, através de uma revisão integrativa, o impacto da microbiota oral no desenvolvimento da aterosclerose, explorando os mecanismos pelos quais a disbiose oral pode contribuir para a inflamação sistémica e para a progressão da doença cardiovascular.

Metodologia: Realizou-se uma revisão integrativa da literatura segundo as recomendações PRISMA. A estratégia de pesquisa baseou-se no modelo PECO. Foram considerados estudos publicados entre 2015 e 2025, aplicando critérios de inclusão e exclusão previamente definidos.

Resultados: Foram incluídos 18 artigos. A evidência científica demonstrou uma associação consistente entre alterações da microbiota oral e a aterosclerose. Bactérias Gram-negativas associadas à doença periodontal podem entrar na circulação e contribuir para a inflamação sistémica, tendo algumas espécies sido identificadas em placas ateroscleróticas. A inflamação crónica associada à disbiose oral parece favorecer a disfunção endotelial e a progressão das lesões. Fatores de risco comuns, como o tabagismo, a diabetes e a higiene oral inadequada, revelaram-se igualmente relevantes.

Conclusões: A evidência sugere uma relação potencial entre a disbiose da microbiota oral e a aterosclerose, reforçando a saúde oral como parte integrante da saúde sistémica. A prevenção e o controlo eficazes das infeções orais, como a doença periodontal, podem contribuir para reduzir o risco cardiovascular e apoiar abordagens integradas entre a medicina dentária e a medicina cardiovascular.

Introduction

Atherosclerosis is a chronic inflammatory disease of the arterial wall and a major cause of disability, loss of productive life-years and death worldwide. Cardiovascular diseases are the leading global cause of death, and this burden is projected to keep rising, reaching approximately 23.6 million deaths by 2030.¹ The disease is characterised by the accumulation of low-density lipoprotein (LDL) in the arterial intima and by the expression of leukocyte adhesion molecules and chemokines by the activated endothelium, which promotes the recruitment of innate and adaptive immune cells and a local inflammatory process. Intensification of this process can lead to local proteolysis, rupture of the atherosclerotic plaque and, ultimately, thrombus formation, causing ischaemia and myocardial infarction.² Atherosclerosis-related cardiovascular disease results from well-established risk factors - arterial hypertension, diabetes mellitus, visceral obesity, dyslipidaemia and age - as well as from unhealthy lifestyle habits such as smoking and an inadequate diet.³

The oral microbiota is the second largest microbial community of the human body, after the gut microbiota; more than 700 microbial species have been identified colonising distinct ecological niches, including saliva, tongue, oral mucosa, palate, dental surfaces, gingiva and subgingival and supragingival plaque. The bacterial biofilms on teeth and gums - commonly known as dental plaque - are recognised as primary aetiological factors in periodontitis, a chronic inflammatory condition characterised by progressive destruction of the tooth-supporting connective tissues.⁴ A growing body of evidence demonstrates an association between periodontal disease and systemic diseases, including diabetes, cardiovascular disease and atherosclerosis. Periodontal disease and cardiovascular disease share common risk factors, such as smoking, obesity and diabetes; given the high prevalence and the substantial financial burden of both conditions, clarifying the biological mechanisms that may explain their relationship has become essential.⁵

This review aims to synthesise, through an integrative approach, the existing scientific knowledge on the association between the oral microbiota and atherosclerotic cardiovascular disease, identifying and analysing the pathophysiological mechanisms that may explain the potential role of oral microorganisms in the development and progression of vascular lesions.

Methodology

This work is an integrative literature review, prepared in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) recommendations.⁶ Three databases were used to select the scientific articles: PubMed, SciELO and ScienceDirect. The MeSH descriptors were used to develop the research question using Boolean operators AND and OR, resulting in the

Boolean phrase: ((oral microbiome) OR (oral microbiota) OR (oral dysbiosis) OR (periodontitis)) AND (atherosclerosis).

A temporal limit of 2015–2025 and the Portuguese and English languages were applied as filters. Inclusion and exclusion criteria were then established. The inclusion criteria included studies in humans (adults) diagnosed with atherosclerosis, coronary artery disease or periodontal disease, *in vivo* or *in vitro* studies, clinical trials, cohort studies, case-control studies, narrative reviews and systematic reviews/meta-analyses. Two reviewers independently assessed the articles and extracted the relevant data to determine their eligibility for inclusion in this integrative review. In cases of uncertainty or disagreement, these were discussed and resolved with the involvement of a third reviewer. The articles were selected according to predefined criteria following the PECO (Population, Exposure, Comparison, Outcome) strategy (Table 1). The resulting research question was: *What is the association between oral microbiota dysbiosis and the progression of atherosclerosis?*

Table 1. PECO search strategy.

Component	Abbrev.	Description
Population	P	Individuals with atherosclerosis
Exposure	E	Presence of oral microbiota dysbiosis
Comparison	C	Individuals without atherosclerosis
Outcome	O	Development of atherosclerosis

Results

After applying the inclusion and exclusion criteria, 18 scientific articles were eligible for analysis (Figure 1).

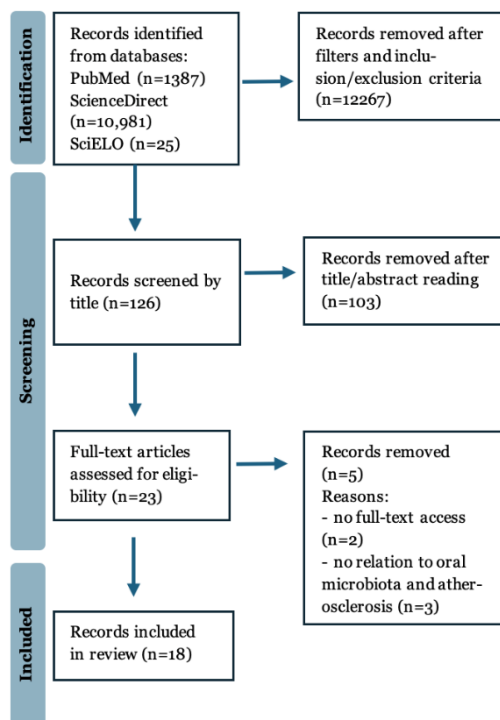


Figure 1. PRISMA-based flow diagram of study selection.

The extracted information was structured into a synthesis table (Table 2), including the authors, year of publication, study design, objective and main results of each investigation.

The included studies converged on several findings. First, oral commensal and periodontal bacteria—including *Aggregatibacter actinomycetemcomitans*, *Campylobacter rectus*, *Porphyromonas gingivalis*, *Prevotella intermedia*, *Streptococcus mutans* and *Fusobacterium* spp.—were identified directly within atherosclerotic plaques, and the bacterial composition of these plaques showed marked similarities with the oral microbiota.^{7,9} Studies using 16S rRNA sequencing of plaques consistently detected genera typical of the oral cavity (*Streptococcus*, *Veillonella*, *Prevotella*, *Fusobacterium*).² Second, case-control studies of the salivary microbiota showed significant compositional alterations in patients with atherosclerotic

cardiovascular disease, with greater relative abundance of periodontitis-associated bacteria (*Prevotella*, *Fusobacterium*, *Porphyromonas*) and reduction of beneficial species.^{10,16,17} Third, mechanistic reviews described how oral dysbiosis and bacterial products (lipopolysaccharide [LPS], gingipains, extracellular vesicles) enter the circulation, activate Toll-like receptors (TLR2/TLR4) and the NF- κ B and MAPK pathways, and raise pro-inflammatory cytokines (IL-1 β , IL-6, TNF- α), promoting endothelial dysfunction and atherogenesis.^{5,8,11,12,15,19} Finally, experimental infection with *P. gingivalis* aggravated atherosclerosis in animal models by inducing vascular smooth muscle cell apoptosis and repressing macrophage efferocytosis, weakening the fibrous cap.¹³ Reviews of common risk factors and of the oral–gut axis complemented these mechanistic findings.^{3,14,18}

Table 2. Summary of the eighteen included studies.

Author (year)	Title	Study design	Objective	Main findings
Chhibber-Goel et al. (2016)	Linkages between oral commensal bacteria and atherosclerotic plaques in coronary artery disease patients	Meta-analysis	Assess the presence of oral bacteria in atherosclerotic plaques	Oral commensals (<i>A. actinomycetemcomitans</i> , <i>C. rectus</i> , <i>P. gingivalis</i> , <i>P. intermedia</i> , <i>S. mutans</i> , <i>F. necrophorum</i>) found within coronary plaques; plaque composition resembled the oral microbiota, suggesting oral origin and translocation.
Slocum et al., (2016)	Immune dysregulation mediated by the oral microbiome: potential link to chronic inflammation and atherosclerosis	Narrative review	Examine how the oral microbiome induces systemic immune dysregulation	Dysbiosis (<i>P. gingivalis</i> , <i>T. forsythia</i> , <i>F. nucleatum</i>) and LPS trigger systemic immune activation, endothelial dysfunction, LDL oxidation and atherosclerosis progression.
Ziganshina et al., (2016)	Bacterial Communities Associated with Atherosclerotic Plaques from Russian Individuals with Atherosclerosis	Observational study	Characterise bacterial communities in plaques of Russian patients	16S rRNA sequencing showed high diversity (Proteobacteria, Firmicutes, Bacteroidetes, Actinobacteria) with recurrent oral genera (<i>Streptococcus</i> , <i>Veillonella</i> , <i>Prevotella</i> , <i>Fusobacterium</i>).
Pietiäinen et al., (2018)	Mediators between oral and dysbiosis and cardiovascular diseases	Narrative review	Describe mediators linking oral dysbiosis and cardiovascular disease	Oral dysbiosis raises inflammatory mediators (IL-1 β , IL-6, TNF- α); <i>P. gingivalis</i> LPS activates TLRs and NF- κ B, promoting endothelial dysfunction and altered lipoprotein metabolism.
Isoshima et al., (2021)	Microbiome composition comparison in oral and atherosclerotic plaque from patients with and without periodontitis	Observational study	Compare oral and plaque microbiome in patients with/without periodontitis	Greater oral–plaque similarity in periodontitis; <i>Porphyromonas</i> , <i>Fusobacterium</i> , <i>Prevotella</i> and <i>Treponema</i> more frequent; no causal relationship established.
Kato-Kogoe et al., (2022)	Characterization of Salivary Microbiota in Patients with Atherosclerotic Cardiovascular Disease: A Case-Control Study.	Case-control study	Assess salivary microbiota differences in ASCVD vs healthy controls	ASCVD patients showed altered salivary microbiota, with higher <i>Prevotella</i> , <i>Fusobacterium</i> and <i>Porphyromonas</i> and reduced beneficial species; saliva as a potential early marker.
Peng et al., (2022)	Oral microbiota in human systematic diseases	Narrative review	Examine links between oral microbiota and systemic diseases	Oral microbes and products (LPS, proteases) invade the bloodstream in periodontitis; <i>P. gingivalis</i> modulates systemic immunity, contributing to cardiovascular and other diseases.
Zou et al., (2022)	Periodontopathic microbiota and atherosclerosis: Roles of TLR-Mediated inflammation response	Narrative review	Explain how periodontopathogens activate TLR pathways in atherosclerosis	<i>P. gingivalis</i> , <i>T. forsythia</i> and <i>T. denticola</i> activate TLR2/TLR4, raising IL-1 β , TNF- α and IL-6 and promoting endothelial dysfunction and atherogenesis.
Gualtero et al., (2023)	Oral microbiome mediated inflammation, a potential inductor of	Comprehensive review	Analyse how oral dysbiosis induces systemic	Pathogens translocate to the circulation and activate TLR2/4, NF- κ B and MAPK, promoting endothelial

	vascular diseases: a comprehensive review.		inflammation and vascular disease	dysfunction, monocyte adhesion and smooth muscle proliferation.
Huang et al., (2023)	The Roles of Periodontal Bacteria in Atherosclerosis	Narrative review	Analyse the role of periodontal bacteria in vascular inflammation	<i>P. gingivalis</i> , <i>A. actinomycetemcomitans</i> , <i>F. nucleatum</i> and <i>T. forsythia</i> produce virulence factors (LPS, gingipains, vesicles) inducing endothelial dysfunction.
Ricciardi et al., (2023)	Risk Factors and Immunoinflammatory Mechanisms Leading to Atherosclerosis: Focus on the Role of Oral Microbiota Dysbiosis	Narrative review	Review risk factors and immunoinflammatory mechanisms, focusing on oral dysbiosis	Oral dysbiosis contributes via bacteraemia, systemic inflammation, endothelial dysfunction and LDL oxidation (TLR2/4–NF-κB; IL-6, TNF-α, IL-1β); proposed as a cardiovascular risk marker.
Xie et al., (2023)	Oral pathogen aggravates atherosclerosis by inducing smooth muscle cell apoptosis and repressing macrophage efferocytosis	Experimental study	Investigate how an oral pathogen aggravates atherosclerosis	<i>P. gingivalis</i> worsened atherosclerosis in animal models, inducing vascular smooth muscle cell apoptosis and repressing macrophage efferocytosis, weakening the fibrous cap.
Li et al., (2024)	The interaction between oral microbiota and gut microbiota in atherosclerosis	Narrative review	Analyse the oral–gut microbiota interaction in atherosclerosis	Bidirectional oral–gut interaction drives systemic inflammation (TLR4, NF-κB, IL-6, TNF-α); shared phylotypes found in plaques and oral/gut samples.
Wang et al., (2024)	The Oral Microbiota, Microbial Metabolites, and Immuno-Inflammatory Mechanisms in Cardiovascular Disease	Narrative review	Review oral microbiota, metabolites and immuno-inflammatory mechanisms in CVD	Microbial metabolites (LPS, short-chain fatty acids, vesicles, toxins) modulate distant inflammation via TLR2/4 and NF-κB; cytokines promote endothelial dysfunction and atherogenesis.
Wu et al., (2024)	Alterations in the Salivary Microbiome and Metabolism in Patients With Carotid Atherosclerosis From Rural Northeast China	Case-control study	Investigate salivary microbiome and metabolism in carotid atherosclerosis	Salivary microbiota and metabolism significantly altered in carotid atherosclerosis; saliva proposed as a non-invasive screening tool.
Akhi et al., (2025)	Oral Microbiota Linking Humoral Response, Periodontitis and Atherosclerosis	Case-control study	Relate oral microbiota and humoral response to periodontitis and atherosclerosis	Patients showed elevated serum antibodies against periodontal pathogens; significant correlation between oral dysbiosis, specific antibodies and atherosclerosis markers (<i>Filifactor alocis</i> as a candidate link).
Tasouli-Drakou et al., (2025)	Atherosclerosis: A Comprehensive Review of Molecular Factors and Mechanisms	Narrative review	Review molecular factors and mechanisms of atherosclerosis	Atherosclerosis driven by oxLDL-induced chronic inflammation, with endothelial activation, foam cell formation and involvement of NF-κB, MAPK, TLRs, IL-1β, IL-6 and TNF-α.
Xiao et al., (2025)	The oral microbiome and atherosclerosis: current evidence on association, mechanisms, and clinical implications	Narrative review	Synthesise current evidence on the oral microbiome and atherosclerosis	Consistent association between oral dysbiosis (especially periodontitis) and atherosclerosis/cardiovascular events, mediated by bacterial entry and systemic immune activation.

Discussion

Atherosclerosis: epidemiology and pathogenesis.

Atherosclerosis is a chronic, systemic and progressive inflammatory disease of medium- and large-caliber arteries, characterised by the progressive accumulation of lipids, inflammatory cells and fibrous tissue in the arterial wall, beginning with endothelial dysfunction.¹⁸ It is the main cause of cardiovascular disease and related vascular disorders—coronary artery disease, peripheral artery disease, stroke and aortic atherosclerosis—collectively designated atherosclerotic cardiovascular disease. Cardiovascular diseases remain a leading cause of death worldwide, responsible for approximately 17.9 million deaths per year; their global prevalence nearly doubled between 1990 and 2019, reaching around 523 million cases.¹⁵ Atherosclerotic cardiovascular disease results from the interaction between modifiable risk factors (hypertension, diabetes, dyslipidaemia, obesity, diet, smoking, alcohol abuse and psychosocial factors) and non-modifiable factors (age, sex, ethnicity, clinical history and genetics).³ Smoking induces oxidative stress and reduces nitric oxide (NO) availability, promoting endothelial dysfunction; elevated LDL cholesterol is an established causal factor; and diabetes mellitus accelerates vascular disease through advanced glycation end-products and increased oxidative stress.^{3,18}

The atherosclerotic plaque forms through the accumulation and retention of LDL in the intima, where it is oxidised (oxLDL), stimulating the release of pro-inflammatory mediators and the recruitment of monocytes that differentiate into macrophages. These phagocytose oxLDL, forming foam cells that release cytokines and reactive oxygen species, perpetuating chronic inflammation.⁷ Progressively, plaques calcify through transformation of vascular smooth muscle cells into osteoblast-like cells mediated by RUNX2; apoptosis of smooth muscle and endothelial cells weakens the fibrous cap, increasing the risk of rupture, platelet activation and thrombus formation.¹⁸ Plaques may also undergo regressive changes, described as an interplay of reduction of necrotic and extracellular debris, restoration of endothelial homeostasis and cessation of smooth muscle proliferation.^{2,20}

Oral microbiota composition and systemic inflammation

The oral microbiota comprises more than 700 microbial species across distinct niches. Under normal conditions the oral bacterial community is relatively stable; biofilms are initially colonised by Gram-positive commensals (notably *Streptococcus viridans* group) and, as they mature, incorporate Gram-negative anaerobes such as *Porphyromonas gingivalis*, *Treponema denticola* and *Tannerella forsythia*, frequently associated with periodontal inflammation.^{4,21} The concept that oral infections may affect distant organs dates back to the focal infection theory; current microbiome research reinforces the relationship between oral

microorganisms and chronic diseases.^{4,22} Oral dysbiosis can favour the proliferation of periodontopathogenic bacteria capable of migrating into the bloodstream, triggering bacteraemia and endotoxaemia and promoting systemic inflammatory responses; bacterial products and virulence factors activate endothelial cells, monocytes, macrophages and platelets, increasing pro-inflammatory cytokines and oxidative stress.^{4,11} The oral microbiota also participates in nitrate/nitrite metabolism and NO production; dysbiosis associated with periodontopathogens may disturb this balance and induce systemic inflammation. Transient bacteraemia can occur not only during dental procedures but also during daily activities such as chewing and tooth-brushing, especially in individuals with periodontitis.^{11,12}

Mechanisms linking the oral microbiota to atherosclerosis

The main biological hypothesis is the passage of bacterial toxins and microbial components into the circulation, inducing a systemic inflammatory response. LPS released during endotoxaemia can favour increased LDL and cardiovascular risk, while microbial DNA and membrane phospholipids may reach vascular and adipose tissues.⁷ Bacteraemia and cytokine release promote endothelial changes and leukocyte migration into the vascular wall, with TLRs playing a central role in pathogen recognition and inflammatory activation.^{1,7} Pro-inflammatory mediators produced in periodontal tissues can reach the circulation and alter lipoprotein metabolism (VLDL, IDL, LDL), favouring lipid uptake and attenuating anti-atherogenic processes.⁸ *P. gingivalis* can disseminate during gingival ulceration or periodontal interventions and promote vascular inflammation, endothelial activation and plaque instability.¹³ Beyond direct bacteraemia, an oral–gut–vascular axis has been proposed: ingested oral pathobionts may induce gut dysbiosis, increase intestinal permeability and promote chronic low-grade metabolic endotoxaemia, with trimethylamine N-oxide as an additional cardiovascular risk metabolite.^{9,19} Importantly, although bacteria have been identified in plaques, it remains uncertain whether they participate directly in pathogenesis or represent contaminants; antibiotic therapy in patients with atherosclerosis has not produced significant clinical improvement, underscoring the need for further investigation.²

Epidemiological and clinical data

Periodontitis and cardiovascular disease are both slowly progressive chronic diseases, and their temporal sequence remains uncertain. Meta-analyses indicate a significant association: a 24% increase in the relative risk of coronary disease in periodontitis and 34% with advanced tooth loss.^{7,8} The INVEST study showed a linear relationship between the severity of oral infections and carotid plaque, and the ARIC study (a long-term epidemiological study funded by the National Institutes of Health [NIH] that has monitored the cardiovascular and cognitive health of more than 15,000 adults in the United States since 1987) reported that periodontitis was associated with a higher incidence of stroke (relative risk 2.6 for cardioembolic and 2.2 for thrombotic stroke), as well as with heart failure, coronary

disease and hypertension.^{1,15} In a cross-sectional study of 96 individuals, severe periodontitis was significantly more prevalent in patients with carotid atherosclerosis than in controls (93.8% vs 58.3%), with greater abundance of periodontitis-associated bacteria.¹⁶ Analysis of the salivary microbiota of 129 Japanese participants likewise showed compositional alterations in patients with atherosclerotic cardiovascular disease, suggesting that the salivary microbiota could be an early marker of disease.¹⁰ Patients with atherosclerosis also exhibited elevated serum antibodies against periodontal pathogens, with *Filifactor alocis* proposed as a potential microbial link.¹⁷ Periodontal treatment reduces inflammatory markers associated with cardiovascular disease and may lower cardiovascular risk.¹¹

Therapeutic perspectives and the role of the dentist

Microbiota-modulation strategies—dietary interventions, prebiotics/probiotics and microbiota-targeted therapies—are being investigated to restore oral and gut microbial balance and reduce inflammatory mechanisms; diet may favour beneficial bacteria such as *Bifidobacterium*, improving lipid and inflammatory metabolism.¹⁴ A preclinical study showed that an engineered short-chain fatty acid-producing *Escherichia coli* Nissle 1917 strain reduced myocardial lesions in an experimental model.¹⁹ Artificial intelligence and machine-learning models are being explored to identify bacterial signatures associated with atherosclerotic cardiovascular disease, supporting personalised prevention.¹⁹ Dentists occupy a privileged position in the prevention of periodontal disease through early detection, patient education and control of modifiable risk factors, thereby contributing to reducing the systemic inflammatory burden.²³

This integrative review has limitations. The predominance of secondary literature and the scarcity of primary studies on causality may constrain the findings. Many available studies have observational or experimental designs that do not allow a definitive causal relationship to be established between oral dysbiosis and atherosclerosis. Differences in study populations, microbiome analysis methods and periodontal diagnostic criteria also limit the interpretation of results.

Conclusion

This integrative review analysed the relationship between the oral microbiota and the development of atherosclerosis, bringing together the scientific data published over the last decade on the main biological mechanisms and clinical implications. The oral cavity should not be regarded as an isolated system but as an important component of systemic health, capable of influencing inflammatory and metabolic processes. Under balanced conditions the oral microbiota exerts protective functions; however, dysbiosis and periodontitis can favour periodontopathogenic microorganisms and the release of inflammatory mediators that, during transient bacteraemia, may reach the systemic circulation

and contribute to chronic low-grade inflammation, endothelial dysfunction, altered lipid metabolism and progression of the atherosclerotic process.

Several epidemiological and clinical studies have shown a significant association between periodontitis and an increased risk of coronary disease, stroke and other cardiovascular manifestations. Although the direct causal relationship still requires further investigation, the available data reinforce a relevant link between impaired oral health and increased atherosclerotic risk. Periodontal treatment has demonstrated the capacity to reduce certain systemic inflammatory markers, suggesting that early intervention may constitute a complementary strategy for cardiovascular prevention. The development of microbiota-modulation strategies and microbiological biomarkers, together with advances in artificial intelligence, may open new perspectives for personalised cardiovascular risk stratification. Overall, the oral microbiota may play an important role in the pathogenesis of atherosclerosis, underlining the need for a multidisciplinary approach between dentistry and general medicine that values oral health promotion as an integral part of the prevention and control of cardiovascular disease.

The data supporting the results can be found or will be provided by the authors upon request.

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