International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE	PREVENTION AND MANAGEMENT OF SYSTEMIC COMPLICATIONS ARISING FROM POOR ORAL HYGIENE: CURRENT STATE OF KNOWLEDGE
DOI	https://doi.org/10.31435/ijitss.2(50).2026.5114
RECEIVED	23 February 2026
ACCEPTED	05 April 2026
PUBLISHED	14 April 2026
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

PREVENTION AND MANAGEMENT OF SYSTEMIC COMPLICATIONS ARISING FROM POOR ORAL HYGIENE: CURRENT STATE OF KNOWLEDGE

Katarzyna Nycz
Students' Scientific Club, Department of Neurosurgery, Faculty of Medical Sciences in Katowice, Medical University of Silesia, Katowice, Poland
ORCID ID: 0009-0001-0897-6855

Jędrzej Sztajura *(Corresponding Author, Email: j.sztajura15@wp.pl)*
Students' Scientific Club, Department of Neurosurgery, Faculty of Medical Sciences in Katowice, Medical University of Silesia, Katowice, Poland
ORCID ID: 0009-0009-8975-731X

ABSTRACT

Introduction: Oral health is increasingly recognized as a critical component of overall health and well-being. Poor oral hygiene, particularly dental caries and periodontal disease, has been linked not only to local oral conditions but also to systemic complications, including cardiovascular disease, type 2 diabetes mellitus, respiratory infections, adverse pregnancy outcomes, and neurodegenerative disorders. This review aims to provide a comprehensive overview of the prevention and management of systemic complications arising from poor oral hygiene, summarizing current evidence on biological mechanisms, disease associations, and preventive strategies.
Materials and Methods: A comprehensive literature search was conducted using PubMed, Scopus, Web of Science, and Google Scholar to identify studies investigating the relationship between oral hygiene, periodontal disease, and systemic health outcomes. Keywords included: oral hygiene, periodontal disease, systemic diseases, cardiovascular disease, diabetes, adverse pregnancy outcomes, respiratory disease, neurodegenerative disease, and oral-systemic link.
Conclusion: Evidence indicates that oral health is essential for maintaining physical, mental, and social well-being. Preventive dental care, including early detection and management of caries, periodontal disease, and malocclusion, reduces disease progression, systemic complications, and psychosocial burden. Equitable access to dental services is critical, as limited access exacerbates health disparities and worsens outcomes. Integrating oral healthcare within comprehensive health systems and emphasizing prevention can improve individual and population-level health and quality of life.

KEYWORDS

Oral Hygiene, Periodontal Disease, Cardiovascular Disease, Diabetes, Preventive Dentistry, Health Promotion

CITATION

Katarzyna Nycz, Jędrzej Sztajura. (2026) Prevention and Management of Systemic Complications Arising from Poor Oral Hygiene: Current State of Knowledge. *International Journal of Innovative Technologies in Social Science*. 2(50). doi: 10.31435/ijitss.2(50).2026.5114

Introduction

Oral health is increasingly recognized as an integral component of overall health and well-being rather than an isolated aspect of medical care, as numerous studies have demonstrated its close association not only with oral conditions but also with systemic diseases [1].

The oral cavity represents a complex biological and ecological system that functions as a critical interface between the external environment and the systemic circulation. Nevertheless, oral diseases, particularly dental caries and periodontal disease, remain among the most prevalent chronic conditions worldwide, affecting individuals across all age groups and socioeconomic backgrounds [1,2,3].

Despite their high global burden, these conditions are still frequently underestimated in healthcare systems and public health policies, where oral health is often addressed separately from general medical care. This separation contributes to delayed diagnosis, insufficient prevention, and missed opportunities to mitigate broader health consequences associated with poor oral hygiene.

Over the past decades, growing evidence has demonstrated that inadequate oral hygiene is not merely a local problem confined to the oral cavity but a significant risk factor for a wide range of systemic diseases [4]. Chronic oral infections and periodontal inflammation have been associated with cardiovascular diseases, metabolic disorders such as type 2 diabetes mellitus, adverse pregnancy outcomes, respiratory diseases, and neurodegenerative conditions [5].

These associations are supported by epidemiological data, clinical studies, and mechanistic research highlighting the role of chronic low-grade systemic inflammation, bacteremia, and immune dysregulation originating from oral pathology [6,7].

Periodontal disease is a chronic inflammatory condition characterized by dysbiosis of the oral microbiome and destruction of the supporting structures of the teeth. Pathogenic microorganisms and their virulence factors, including lipopolysaccharides, can enter the systemic circulation through ulcerated periodontal tissues, triggering endothelial dysfunction and systemic inflammatory responses. This persistent inflammatory burden has been proposed as a key mechanism linking poor oral hygiene to the development and progression of systemic diseases [4,8].

Despite the accumulating body of evidence, oral health is still frequently excluded from integrated models of healthcare delivery and preventive medicine. Stomatology and general medicine often operate in parallel rather than in collaboration, resulting in missed opportunities for early detection, prevention, and comprehensive management of systemic complications related to oral diseases. This fragmentation is further exacerbated by socioeconomic disparities, limited access to dental care, and insufficient public awareness of the systemic consequences of poor oral hygiene, all of which contribute to health inequities on a population level [9].

From a public health perspective, improving oral hygiene and access to preventive dental care represents a potentially cost-effective strategy to reduce the burden of chronic systemic diseases. Preventive interventions targeting oral health may not only improve quality of life but also contribute to broader health outcomes by mitigating systemic inflammation and reducing the incidence of associated complications. Consequently, the integration of oral health into general health promotion strategies aligns closely with contemporary approaches to health equity and interdisciplinary care [9].

The aim of this review is to provide a comprehensive and interdisciplinary overview of the prevention and management of systemic complications arising from poor oral hygiene. We summarize current knowledge on the biological mechanisms linking oral and systemic health, review the evidence connecting poor oral hygiene with major systemic diseases and discuss preventive and therapeutic strategies at both the individual and public health levels. By highlighting the central role of oral health in overall well-being, this review seeks to support a more integrated approach to healthcare that bridges dentistry, medicine, and public health.

Materials and methods

A comprehensive literature search was conducted to identify studies examining the relationship between oral hygiene, periodontal disease, and systemic health outcomes. The search was performed using PubMed, Scopus, Web of Science, and Google Scholar. Keywords included: “oral hygiene”, “periodontal disease”, “systemic diseases”, “cardiovascular disease”, “diabetes mellitus”, “adverse pregnancy outcomes”, “respiratory disease”, “neurodegenerative disease”, “inflammation”, and “oral-systemic link”. Additional relevant publications were identified through a manual review of the reference lists of selected articles.

Studies published within the last ten years were considered, with a focus on clinical trials, cohort and case-control studies, systematic reviews, and meta-analyses. Articles were included if they provided data on associations between oral health status and systemic conditions, or mechanistic insights into the oral-systemic connection.

Oral microbiome and pathophysiological mechanisms linking oral and systemic health

The oral cavity hosts a highly diverse microbial community, collectively referred to as the oral microbiome, which plays a critical role in maintaining both oral and systemic homeostasis. A balanced microbiome contributes to immune regulation, inhibition of pathogenic colonization, and the maintenance of oral tissue integrity. However, poor oral hygiene can disrupt this balance, leading to dysbiosis characterized by the overgrowth of pathogenic bacteria and fungi, including *Porphyromonas gingivalis*, *Fusobacterium nucleatum*, and *Aggregatibacter actinomycetemcomitans*, but also *Penicillium*, *Rhodotorula*, *Saccharomycetales*, *Streptococcus*, *Veillonella*, *Neisseria*, *Actinomyces*, and *Schizophyllum*. These pathogens produce virulence factors such as lipopolysaccharides, proteases, and toxins, which can damage periodontal tissues and elicit local inflammatory responses [10,11,12].

Chronic periodontal inflammation can exert systemic effects through several interconnected mechanisms. Pathogenic bacteria and their products can enter the bloodstream via ulcerated periodontal pockets, leading to transient bacteremia. This, in turn, promotes systemic inflammation, endothelial dysfunction, and immune system activation, potentially contributing to the onset or progression of cardiovascular disease, diabetes, and other chronic disorders. Moreover, locally produced inflammatory mediators, such as cytokines (e.g., IL-6, TNF-α, CRP), may spill into systemic circulation, amplifying the overall inflammatory load. Dysbiosis and persistent oral inflammation can also impact distant organs through microbiome-mediated pathways, including alterations in gut microbiota and activation of neuroinflammatory processes [7,13].

Importantly, the relationship between oral microbiota and systemic health is bidirectional. Systemic conditions such as diabetes mellitus or immunodeficiency can exacerbate dysbiosis and periodontal disease, creating a self-perpetuating cycle of inflammation and tissue damage. Understanding these mechanisms highlights the oral cavity as a key hub in systemic health and underscores the importance of preventive and therapeutic strategies aimed at maintaining microbial balance and controlling inflammation [14,15,16].

Systemic complications associated with poor oral hygiene

Cardiovascular diseases

Poor oral hygiene and chronic periodontal disease have been consistently associated with an increased risk of cardiovascular diseases, including atherosclerosis, endocarditis, and hypertension. From a mechanistic perspective, the systemic inflammatory burden generated by periodontal infections plays a central role in endothelial dysfunction, an early and critical step in atherogenesis. Pathogenic bacteria, particularly *Porphyromonas gingivalis*, along with their virulence factors, can enter the bloodstream through ulcerated periodontal pockets, triggering vascular inflammation, oxidative stress, and promoting the development of atherosclerotic plaques [17,18,19].

Epidemiological research has revealed a strong link between periodontitis and major cardiovascular events, including myocardial infarction and stroke. Meta-analyses have shown that individuals with severe periodontitis face a higher relative risk of developing coronary artery disease compared to those with healthy periodontal status. Furthermore, periodontal treatment has been associated with reductions in systemic inflammatory markers, such as CRP and IL-6, indicating potential benefits for lowering cardiovascular risk [20,21].

In addition, poor oral health has been implicated in the development of infective endocarditis, particularly among individuals with underlying cardiac conditions. Chronic periodontal infections can induce transient bacteremia, enabling oral pathogens to colonize damaged endocardial surfaces [22]. Periodontitis has also been linked to hypertension, potentially through mechanisms involving systemic inflammation, endothelial dysfunction, and immune dysregulation. Together, these findings highlight the critical role of oral health as a modifiable risk factor in the prevention and management of cardiovascular diseases [17].

Metabolic disorders

A growing body of evidence indicates a strong and bidirectional association between poor oral hygiene, periodontal disease, and metabolic disorders, particularly type 2 diabetes mellitus. Periodontitis is increasingly regarded as both a consequence and a contributing factor of diabetes, with chronic periodontal inflammation aggravating metabolic imbalance. Ongoing infection within periodontal tissues stimulates the production of proinflammatory mediators that disrupt insulin signaling pathways, ultimately leading to poorer glycemic control.

Insulin resistance constitutes a central mechanistic connection between periodontal disease and metabolic dysfunction. The systemic spread of inflammatory cytokines, including IL-6, TNF-α and CRP, originating from inflamed periodontal tissues, can disrupt insulin sensitivity in peripheral tissues such as skeletal muscle and adipose tissue. This proinflammatory environment fosters chronic low-grade systemic inflammation, a defining feature of type 2 diabetes mellitus and metabolic syndrome [14,15].

In contrast, chronic hyperglycemia and the formation of advanced glycation end products in diabetes negatively influence periodontal health by weakening host immune defenses, intensifying oxidative stress, and altering the composition of the oral microbiota. These pathological changes hasten periodontal tissue degradation and amplify inflammatory cascades, ultimately sustaining a vicious cycle that links metabolic imbalance with the progression of periodontal disease [23].

Notably, periodontal treatment has gained recognition as a supportive intervention in metabolic regulation. Evidence from clinical investigations indicates that non-surgical periodontal therapy can result in small yet clinically relevant improvements in glycemic control, reflected by reductions in glycated hemoglobin (HbA1c), along with decreases in systemic inflammatory biomarkers among patients with type 2 diabetes. Collectively, these observations underscore the significance of periodontal care not only for maintaining oral health but also for enhancing metabolic control and potentially mitigating diabetes-related complications [24].

Neurological and neurodegenerative diseases

An increasing body of research indicates that inadequate oral hygiene and chronic periodontal disease may play a role in the onset and progression of neurological and neurodegenerative conditions, such as Alzheimer's and Parkinson's diseases. These links are thought to arise from persistent systemic inflammation, immune dysregulation, and the dissemination of oral pathogens and their toxic metabolites beyond the oral cavity. Given the heightened sensitivity of the central nervous system to inflammatory and microbial challenges, the oral–brain axis has become an important focus of research in neurodegenerative disease [25,26].

In Alzheimer's disease, periodontal pathogens – particularly *Porphyromonas gingivalis* – have been implicated in driving neuroinflammatory processes. Evidence from studies has revealed bacterial DNA, virulence factors, and gingipains within the brains of affected individuals, pointing to a potential direct microbial contribution. These pathogens and their neurotoxic products may reach the central nervous system either via the bloodstream or through neural routes, including the trigeminal and olfactory nerves. Once established in the brain, they can activate microglia, enhance amyloid-β deposition, and aggravate tau pathology, collectively accelerating neurodegenerative progression [25,27,28].

Furthermore, an increasing body of research points to a link between periodontal infections and Parkinson's disease, potentially driven by shared inflammatory and immunological mechanisms. Chronic inflammation localized in the periodontium may trigger neuroinflammatory responses and oxidative stress, thereby heightening the vulnerability of dopaminergic neurons. It is hypothesized that the systemic rise in cytokines resulting from periodontitis compromises the integrity of the blood–brain barrier, facilitating the translocation of inflammatory agents and microbial byproducts into the central nervous system [25,29,30].

Neuroinflammation serves as a central link between compromised oral health and the development of neurodegenerative disorders. Chronic oral dysbiosis is capable of maintaining a state of low-grade systemic inflammation, which in turn fosters persistent microglial activation and subsequent neuronal degradation. Furthermore, the translocation of oral pathogens and their associated endotoxins may disrupt brain homeostasis by altering gut microbiota and activating neuroimmune pathways. Consequently, these insights highlight oral hygiene and the clinical management of periodontal disease as vital, modifiable interventions to decrease neuroinflammatory stress and potentially slow the progression of neurodegenerative pathologies [25,31,32].

Respiratory diseases

There is growing recognition that inadequate oral hygiene and persistent periodontal infections are major risk factors for respiratory diseases, most notably aspiration pneumonia and chronic obstructive pulmonary disease (COPD). The mouth acts as a primary sanctuary for pathogens that may be inhaled into the lower airways, a risk that is particularly acute among elderly or institutionalized individuals. Such aspiration of oral flora can trigger acute lung infections, worsen the severity of chronic respiratory ailments, and contribute to higher rates of morbidity and mortality [33,34].

Four possible mechanisms have been described to explain the biological plausibility of an association between oral conditions and nosocomial respiratory infections:

1. **Direct aspiration of oral pathogens into the lungs.** Periodontal organisms such as *Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitans* have been implicated in aspiration pneumonia. Moreover, dental biofilms can be colonized by pulmonary pathogens, reinforcing the idea that the oral cavity may serve as a reservoir for microbes responsible for respiratory infections in high-risk patients.
2. **Enzymatic remodeling of the respiratory mucosa.** A second, more complex pathway involves the biochemical modification of the respiratory tract's mucosal surfaces by salivary enzymes. In the presence of active periodontal disease, there is a marked increase in the concentration of proteolytic enzymes, such as mannosidase, fucosidase, and sialidase. These enzymes, secreted by both the oral microbiota and infiltrating polymorphonuclear leukocytes, physically alter the mucosal epithelium. By hydrolytically removing surface fibronectin, these enzymes strip away a crucial protective layer, thereby exposing specific receptors that respiratory pathogens use for attachment. Consequently, poor oral hygiene directly correlates with heightened enzymatic activity, creating a landscape that is far more receptive to bacterial colonization and subsequent infection.
3. **Degradation of protective salivary barriers.** In addition to modifying the epithelium, periodontopathic bacteria actively dismantle the host's first line of defense: the protective salivary film. Hydrolytic enzymes, particularly those produced by *P. gingivalis*, are capable of degrading salivary mucins and other molecules that typically inhibit bacterial adhesion. When these protective films are compromised, the respiratory mucosa loses its "non-stick" properties, allowing pathogens to anchor themselves more effectively to the mucosal receptors. This enzymatic degradation means that individuals with chronic oral inflammation possess a significantly weakened barrier against the infiltration of respiratory invaders.
4. **Systemic and local cytokine-mediated vulnerability.** Finally, the persistent inflammatory state characteristic of untreated periodontal disease leads to a chronic release of pro-inflammatory cytokines and other biologically active mediators. These molecules originate from both the diseased periodontal tissues and peripheral mononuclear cells. Once released, they can induce structural changes in the respiratory epithelium and upregulate the expression of adhesion receptors. This systemic and local "priming" of the lungs not only encourages the colonization of pathogenic bacteria but also exacerbates the host's inflammatory response, significantly increasing the overall risk and severity of respiratory infections.

Collectively, these mechanisms illustrate the complex interplay between oral health and respiratory disease and underscore the importance of rigorous oral hygiene and targeted periodontal care, particularly in hospitalized and geriatric populations, as a preventive strategy to reduce respiratory complications [35].

Consistent epidemiological data demonstrate a correlation between periodontal infections and an elevated incidence of aspiration pneumonia, with the highest risk observed in the elderly and those suffering from dysphagia. For individuals with COPD, persistent oral inflammation and microbial imbalances are tied to more frequent and severe flare-ups. This relationship is likely facilitated by the systemic spread of pro-inflammatory markers and the direct migration of oral pathogens into the respiratory system [36].

Moreover, individuals in clinical settings, particularly those within intensive care units or under prolonged mechanical ventilation, face a significantly elevated risk of respiratory ailments linked to inadequate oral care. Research demonstrates that rigorous oral hygiene protocols, such as mechanical plaque debridement and the application of antimicrobial rinses, effectively lower the frequency of ventilator-associated pneumonia (VAP) and enhance recovery rates. Such evidence highlights the necessity of prioritizing oral health as a fundamental preventive measure to safeguard the respiratory systems of high-risk patients [37].

Pregnancy outcomes

Accumulating evidence indicates that poor oral hygiene and periodontal disease are associated with adverse pregnancy outcomes, most notably preterm birth and low birth weight. Pregnancy is characterized by profound hormonal and immunological changes that can exacerbate gingival inflammation and periodontal tissue breakdown, thereby increasing susceptibility to oral infections. In turn, untreated periodontal disease may act as a chronic inflammatory burden capable of influencing maternal–fetal health [38.39].

During pregnancy, the maternal body undergoes dramatic hormonal fluctuations – particularly the marked increase in estrogen and progesterone levels – which significantly alter the vascular response and immune environment of the gingival tissues. These systemic changes often exacerbate pre-existing gingival inflammation, leading to a condition commonly referred to as pregnancy gingivitis, and accelerating the

breakdown of periodontal support structures. Consequently, the pregnant patient becomes uniquely susceptible to opportunistic oral infections [40,41].

Preterm birth and low birth weight have been consistently linked to maternal periodontal disease in epidemiological studies. Women with moderate to severe periodontitis appear to have a higher risk of delivering prematurely or giving birth to infants with reduced birth weight compared to periodontally healthy counterparts. These outcomes carry significant clinical implications, as both preterm birth and low birth weight are major contributors to neonatal morbidity, long-term developmental complications, and infant mortality [39,42,43].

The biological plausibility of this association is supported by a dual pathway involving both inflammatory and microbiological mechanisms. Untreated periodontal disease is not merely a localized condition; rather, it functions as a persistent reservoir of chronic systemic inflammation. This sustained "inflammatory burden" can trigger a systemic dissemination of pro-inflammatory mediators, including prostaglandin E2 (PGE2), TNF-α, and various interleukins (IL-1, IL-6). These molecules are known biological regulators of labor onset and fetal growth; when present at elevated levels, they may prematurely activate uterine contractions or impair placental function. In addition to this biochemical signaling, a direct microbiological pathway has been proposed. Oral pathogens and their associated endotoxins can translocate hematogenously from the gingival tissues directly to the placenta and amniotic fluid. Once these microbes infiltrate the maternal–fetal unit, they trigger localized inflammatory responses that further compromise the intrauterine environment. Collectively, these systemic and direct effects can dictate the timing of labor and undermine fetal development. Consequently, the maintenance of periodontal health is increasingly viewed as an indispensable component of comprehensive prenatal care, essential for mitigating preventable risks to both mother and child [44,45,46,47].

Taken together, these insights emphasize that maintaining superior oral health is a critical determinant of a successful pregnancy. Implementing routine periodontal screenings and ensuring access to specialized dental care throughout gestation could serve as vital preventive measures, effectively limiting systemic inflammatory exposure. Such evidence necessitates a paradigm shift toward a more integrated healthcare model, where dental and obstetric professionals work in close coordination to minimize the risk of adverse birth outcomes and safeguard maternal–fetal well-being [47,48]. The major systemic complications associated with poor oral hygiene are summarized in Table 1.

Table 1. Major systemic complications associated with poor oral hygiene.

Mechanistic pathway	Description	Associated systemic outcomes
Chronic low-grade systemic inflammation	Persistent release of pro-inflammatory cytokines from periodontal tissues	CVD, diabetes, neurodegeneration, adverse pregnancy outcomes
Transient bacteremia	Entry of oral pathogens into bloodstream through ulcerated periodontal pockets	Endocarditis, atherosclerosis, placental infection
Endothelial dysfunction	Inflammatory and oxidative vascular damage	Hypertension, atherosclerosis
Insulin signaling disruption	Cytokine-mediated interference with glucose metabolism	Type 2 diabetes mellitus
Neuroinflammatory activation	Microglial stimulation, amyloid-β deposition, tau pathology	Alzheimer's disease
Aspiration of oral pathogens	Inhalation of dental biofilm bacteria into lungs	Pneumonia, COPD exacerbations
Enzymatic mucosal degradation	Proteolytic enzymes alter respiratory epithelium and salivary protection	Respiratory infections

Prevention of systemic complications through oral health management

Individual-level prevention

Individual-level prevention is a cornerstone of periodontal care, enabling patients to directly influence disease progression through targeted oral hygiene practices and lifestyle modifications. Evidence indicates that personalized preventive measures can effectively reduce gingival inflammation, limit periodontal tissue destruction, and contribute to long-term stabilization of periodontal status. Emphasizing patient engagement in daily preventive behaviors is therefore essential, not only for maintaining oral health but also for mitigating systemic consequences associated with chronic periodontal disease [49].

Daily oral hygiene practices, including regular toothbrushing with fluoride toothpaste and appropriate interdental cleaning, are consistently promoted as effective preventive measures. However, the adoption and maintenance of these behaviors are not determined solely by individual choice or awareness. Socioeconomic circumstances influence access to essential oral hygiene products, and in many low- and middle-income countries the affordability of fluoride toothpaste remains limited. As a result, disparities in the ability to perform recommended hygiene practices contribute to persistent inequalities in oral health outcomes [49].

Patient education is therefore essential to support effective self-care and informed decision making. Improving individuals' understanding of oral disease risk and preventive behaviors is a key objective of chairside counseling and public health messaging. Nevertheless, the impact of patient education is often constrained by environments saturated with marketing products that are detrimental to health. Extensive promotion of sugar-rich foods and beverages, particularly to children, shapes preferences and consumption patterns early in life, reducing the effectiveness of individual-level advice and reinforcing unhealthy behaviors [49].

Dietary factors play a particularly important role in the prevention of oral diseases, with free sugar consumption representing a major modifiable risk factor. Global increases in sugar availability, driven by economic development and the expansion of multinational food and beverage corporations, have contributed to rising rates of dental care. Nutritional counseling at the individual level can help raise awareness of the relationship between diet and oral health and encourage healthier choices. However, such counseling operates within commercial environments in which sugary products are widely available, aggressively marketed, and increasingly affordable. Consequently, even well-informed individuals may face significant challenges in translating knowledge into sustained behavioral change [49].

Professional dental care

Professional dental care is crucial for the prevention and management of periodontal diseases, primarily through timely diagnosis, appropriate intervention, and continuous monitoring. Traditional periodontal assessment relies on clinical and radiographic evaluations, which remain the standard for determining disease extent and guiding treatment. While these methods effectively manage established disease, they are based on retrospective indicators of tissue damage and provide limited insight into current disease activity. This restricts the ability to assess real-time response to therapy, identify patients at higher risk of progression, and optimize the use of emerging targeted interventions aimed at reducing disease activity and preventing further tissue loss [49,50].

Periodontal treatment strategies encompass both non-surgical and surgical approaches. Non-surgical therapy, including scaling and root planning, adjunctive local or systemic pharmacological interventions, and reinforcement of oral hygiene practices, forms the foundation of periodontal care in most patients. Surgical interventions, such as flap procedures, regenerative therapy, and pocket reduction, are indicated when non-surgical management alone is insufficient, particularly in cases with deep periodontal pockets, significant attachment loss, or complex anatomical considerations. Following these protocols allows clinicians to stabilize disease, promote tissue regeneration where possible, and reduce the microbial and inflammatory burden within the periodontium [49,50].

The release of inflammatory mediators from periodontal tissues represents a biologically plausible mechanism linking periodontitis with chronic systemic diseases. Effective periodontal therapy, by reducing microbial burden and local inflammation, can substantially modulate the body’s systemic inflammatory response. Although epidemiological evidence supports this association, the lack of standardization in disease definitions, study designs, and treatment protocols limits the ability to draw definitive conclusions. Advancing this field requires precise disease classification, clearly defined and reproducible intervention protocols, rigorous statistical analysis, and adequately powered clinical trials. Only through such improvements can the extent to which periodontal therapy reduces systemic inflammatory markers and translates into meaningful, clinically significant systemic health benefits be reliably determined [49,50,51].

Management strategies for systemic complications related to oral diseases

Periodontal treatment as a systemic intervention

Periodontal treatment is crucial not only for preserving oral health but also for mitigating systemic risks associated with chronic diseases. Evidence demonstrates that periodontitis contributes to systemic inflammation, which can exacerbate conditions such as type 2 diabetes mellitus, cardiovascular diseases, adverse pregnancy outcomes, and pneumonia. In diabetic patients, periodontal therapy reduces local and systemic inflammatory mediators, contributing to improved glycemic control and potentially lowering the risk of diabetes-related complications. In cardiovascular disease, periodontal treatment decreases systemic markers of inflammation, including C-reactive protein, fibrinogen, interleukin-6, and tumor necrosis factor-α, improves endothelial function, lowers blood pressure, and favorably modulates lipid profiles, highlighting its potential role in reducing cardiovascular morbidity and improving vascular health. Similarly, in the context of adverse pregnancy outcomes, timely periodontal intervention may help prevent premature birth and low birth weight by limiting the hematogenous dissemination of oral pathogens and inflammatory mediators. In high-risk populations, such as the elderly or hospitalized patients, maintaining periodontal health can reduce oral microbial reservoirs that contribute to pneumonia, further demonstrating the systemic impact of periodontal care. Collectively, these findings emphasize that periodontal therapy functions not merely as a local treatment but as a systemic intervention capable of modulating inflammatory burden, improving biochemical markers associated with chronic diseases, and supporting overall systemic health. Early diagnosis, individualized treatment planning, and rigorous periodontal management are therefore essential to maximize both oral and general health outcomes [50].

Pharmacological and adjunctive approaches

Adjunctive pharmacological strategies are used to complement conventional periodontal therapy by directly targeting pathogenic biofilms and modulating local inflammatory responses [52].

Systemic antibiotics, such as amoxicillin combined with metronidazole, metronidazole alone, azithromycin, or doxycycline, have been shown to provide modest improvements in probing depth and clinical attachment, particularly in patients with deep pockets or aggressive periodontitis. Nevertheless, the benefits are variable, and routine use is limited due to concerns about antimicrobial resistance, adverse effects, and inconsistent clinical relevance. Accordingly, systemic antibiotics are generally reserved for severe cases, specific microbial profiles, or medically compromised patients, and should be prescribed based on individualized risk assessment and microbiological testing [52,53].

Hydrogel-based drug delivery systems offer a localized alternative by providing sustained, site-specific release of antimicrobial and anti-inflammatory agents directly within periodontal pockets. These injectable, stimuli-responsive hydrogels can maintain effective drug concentrations, for example metronidazole or minocycline, at the diseased site, reducing subgingival pathogen load and controlling inflammation while minimizing systemic exposure [52,53].

Multifunctional designs allow co-delivery of antibacterial and anti-inflammatory compounds, supporting microbial control, modulating host responses, and promoting tissue regeneration. Such approaches align with personalized periodontal therapy, aiming to optimize the local microenvironment, limit tissue destruction, and reduce potential systemic effects of chronic periodontal infection [52,53].

Public health perspective and health equity

Socioeconomic status (SES), including income, education, race, ethnicity, and housing conditions, is strongly associated with disparities in oral disease burden. Socially patterned lifestyle and environmental factors, such as diet quality, tobacco and alcohol use, antibiotic exposure, and physical activity, significantly influence the composition and function of the oral microbiome. Chronic social disadvantage and structural discrimination may further alter microbial profiles through sustained inflammatory and stress-related mechanisms. Importantly, most oral microbiome research has been conducted in socioeconomically privileged, predominantly Euro-centric populations, limiting generalizability. Expanding research to more diverse populations is essential to better understand the interplay between social determinants, microbial ecology, and oral health outcomes [54].

The shift from treatment-focused to prevention-oriented oral microbiome research highlights the need for population-level interventions aimed at maintaining microbial homeostasis. Public health strategies promoting balanced nutrition, reduction of tobacco and alcohol use, physical activity, responsible antibiotic use, and water fluoridation are central to sustaining a symbiotic oral microbiota and reducing dysbiosis-

associated disease risk. Emerging microbiome-modulating approaches, including oral microbiome transplantation and targeted prebiotic or probiotic therapies, may serve as adjunctive strategies, particularly in underserved settings [54].

Population-based oral health education remains fundamental to shaping behaviors that directly affect oral microbial ecology. Oral hygiene practices, dietary habits, and regular preventive dental care are closely linked to microbial diversity and stability. Targeted educational initiatives for vulnerable groups - such as older adults, individuals with chronic diseases, and socioeconomically disadvantaged populations - may support the maintenance of a health-associated microbiome and contribute to reducing oral health inequalities at the population level [54]. Prevention and management strategies for systemic complications related to poor oral hygiene are summarized in Table 2.

Table 2. Prevention and management strategies for systemic complications related to poor oral hygiene.

Level of intervention	Key strategies	Mechanism of action	Potential systemic impact
Individual-level prevention	Daily toothbrushing with fluoride toothpaste, interdental cleaning, dietary control (reduced free sugar intake), lifestyle modification	Reduction of biofilm accumulation, decreased gingival inflammation, improved microbial balance	Lower systemic inflammatory burden; reduced risk of CVD, diabetes progression, adverse pregnancy outcomes
Patient education	Chairside counseling, public health messaging, risk awareness programs	Improved health literacy, behavioral modification	Long-term disease stabilization; reduced chronic inflammation and associated systemic risks
Professional dental care	Periodontal screening, scaling and root planing, surgical therapy when indicated, continuous monitoring	Reduction of microbial load and local inflammatory mediators	Modulation of systemic inflammatory markers (CRP, IL-6, TNF-α); improved glycemic and cardiovascular parameters
Periodontal therapy as systemic intervention	Early diagnosis, individualized treatment planning, maintenance therapy	Decreased systemic cytokine levels, improved endothelial function, better metabolic control	Improved HbA1c, reduced cardiovascular morbidity, lower risk of preterm birth and pneumonia
Pharmacological adjuncts	Systemic antibiotics (selected cases), local drug delivery (hydrogels, minocycline, metronidazole), host-modulating agents	Targeted antimicrobial and anti-inflammatory effects; modulation of host response	Reduced inflammatory burden with minimized systemic exposure
Public health strategies	Water fluoridation, nutrition policies, tobacco/alcohol control, access to dental services	Maintenance of oral microbiome homeostasis; reduction of disease incidence	Decreased health disparities; population-level reduction of systemic disease burden

Future directions

The future of periodontology is increasingly oriented toward precision-based and interdisciplinary approaches that integrate biomaterials science, molecular diagnostics, and digital health technologies. Advances in hydrogel-based drug delivery systems exemplify the transition toward personalized dentistry, enabling the design of injectable, stimulus-responsive biomaterials with controlled release profiles tailored to the specific microbial composition and inflammatory status of individual periodontal pockets. The incorporation of antibacterial, anti-inflammatory, and osteogenic agents within multifunctional hydrogels allows targeted modulation of the local microenvironment, supporting both infection control and tissue regeneration [54].

Parallel progress in the identification of inflammatory biomarkers in saliva and gingival crevicular fluid strengthens the concept of biologically guided therapy, in which measurable molecular parameters may inform diagnosis, risk stratification, and treatment monitoring - not only in periodontitis but also in oral mucosal diseases and the early detection of oral malignancies. Emerging digital technologies and real-time health monitoring systems further complement this paradigm by facilitating continuous assessment of periodontal status and adaptive therapeutic responses. The continued evolution of periodontology will therefore depend on

robust interdisciplinary collaboration among clinicians, microbiologists, bioengineers, and data scientists to translate innovative biomaterials and biomarker-driven strategies into safe, effective, and clinically applicable solutions [54].

Conclusions

The available evidence clearly demonstrates that oral health constitutes a fundamental component of overall health rather than an isolated clinical concern. Oral diseases directly impair essential physiological functions, including eating, speaking, and sleeping, and substantially affect psychological well-being, self-esteem, and social participation. Consequently, untreated oral conditions contribute not only to local tissue damage but also to functional limitations, chronic pain, reduced productivity, and diminished quality of life. These effects confirm that maintaining oral health is indispensable for preserving physical, mental, and social well-being at both individual and population levels.

Preventive dental care is therefore a critical determinant of general health. Early detection and management of caries, periodontal diseases, and malocclusion, combined with routine professional care and reinforcement of oral hygiene practices, significantly reduce disease progression, functional impairment, and associated psychosocial burden. Prevention limits the need for complex and costly interventions, decreases the risk of systemic complications linked to chronic oral inflammation, and improves patient-reported health outcomes. By controlling disease at its earliest stages, preventive strategies protect not only oral structures but also broader health stability and long-term quality of life.

Equitable access to dental services is equally essential. Population-based data consistently show that individuals with limited access to preventive and therapeutic care experience worse oral health outcomes and lower quality of life. Restricted access amplifies health disparities, increases the prevalence of untreated disease, and perpetuates cycles of pain, disability, and social disadvantage. Ensuring universal and timely access to preventive and therapeutic dental care must therefore be recognized as a public health priority. Integrating oral healthcare within comprehensive health systems, with an emphasis on prevention and patient-centered outcomes, is necessary to safeguard both oral and general health and to promote sustainable improvements in population well-being [1,55].

Disclosure

Author's contribution:

All authors contributed to the article.

All authors have read and agreed with the published version of the manuscript.

Conflict of interest: The authors declare no conflict of interest.

Funding statement: No external funding was received to perform this review

REFERENCES

1. Peres, M. A., Macpherson, L. M. D., Weyant, R. J., Daly, B., Venturelli, R., Mathur, M. R., Listl, S., Celeste, R. K., Guarnizo-Herreño, C. C., Kearns, C., Benzian, H., Allison, P., & Watt, R. G. (2019). Oral diseases: A global public health challenge. *The Lancet, 394*(10194), 249–260. https://doi.org/10.1016/S0140-6736(19)31146-8
2. Albandar, J. M. (2011). Underestimation of periodontitis in NHANES surveys. *Journal of Periodontology, 82*(3), 337–341. https://doi.org/10.1902/jop.2011.100638
3. Peng, X., Cheng, L., You, Y., Tang, C., Ren, B., Li, Y., Xu, X., & Zhou, X. (2022). Oral microbiota in human systematic diseases. *International Journal of Oral Science, 14*(1), Article 14. https://doi.org/10.1038/s41368-022-00163-7
4. Tattar, R., Cazvalho da Costa, B. D., & Neves, V. C. M. (2025). The interrelationship between periodontal disease and systemic health. *British Dental Journal, 239*, 103–108.
5. Diehl, D. (2026). Periodontitis and systemic health: An interconnected relationship. In *Advances in Experimental Medicine and Biology* (Vol. 1492, pp. 83–100). https://doi.org/10.1007/978-3-032-03176-1_5
6. Botelho, J., Mascarenhas, P., Viana, J., Proença, L., Orlandi, M., Leira, Y., Chambrone, L., Mendes, J. J., & Machado, V. (2022). An umbrella review of the evidence linking oral health and systemic noncommunicable diseases. *Nature Communications, 13*, Article 7614. https://doi.org/10.1038/s41467-022-35333-6
7. Foroughi, M., Torabinejad, M., Angelov, N., Ojcius, D. M., Parang, K., Ravnan, M., & Lam, J. (2025). Bridging oral and systemic health: Exploring pathogenesis, biomarkers, and diagnostic innovations in periodontal disease. *Infection, 53*, 2277–2302.

8. Bhuyan, R., Bhuyan, S. K., Mohanty, J. N., Das, S., Juliana, N., & Juliana, I. F. (2022). Periodontitis and its inflammatory changes linked to various systemic diseases: A review of its underlying mechanisms. *Biomedicines, 10*(10), 2659. https://doi.org/10.3390/biomedicines10102659
9. Lee, A., Lomazzi, M., Lee, H., & Bedi, R. (2019). Integrating oral health with public health systems under the framework of the Global Charter for the Public's Health. *International Dental Journal, 69*(3), 167–170. https://doi.org/10.1111/idj.12448
10. Baker, J. L., Mark Welch, J. L., Kauffman, K. M., McLean, J. S., & He, X. (2024). The oral microbiome: Diversity, biogeography and human health. *Nature Reviews Microbiology, 22*(2), 89–104. https://doi.org/10.1038/s41579-023-00963-6
11. Gasmi Benahmed, A., Kumar Mujawdiya, P., Noor, S., & Gasmi, A. (2022). Porphyromonas gingivalis in the development of periodontitis: Impact on dysbiosis and inflammation. *Archives of Razi Institute, 77*(5), 1539–1551. https://doi.org/10.22092/ARI.2021.356596.1875
12. Salah, A. N., Doghish, Y. A., Abbass, S. O., Mansour, R. M., Sayed, G. A., Elshami, N. H., Mageed, S. S. A., Mohammed, O. A., Abulsoud, A. I., Zaki, M. B., Mosalam, E. M., Elrebehy, M. A., Alfarsi, K., & Doghish, A. S. (2025). Microbiota-based therapies in oral health and disorders. *Folia Microbiologica, 70*(6), 1217–1240. https://doi.org/10.1007/s12223-025-01324-x
13. Hajishengallis, G. (2015). Periodontitis: From microbial immune subversion to systemic inflammation. *Nature Reviews Immunology, 15*(1), 30–44. https://doi.org/10.1038/nri3785
14. Ranbhise, J. S., Ju, S., Singh, M. K., Han, S., Akter, S., Ha, J., Choe, W., Kim, S. S., & Kang, I. (2025). Chronic inflammation and glycemic control: Exploring the bidirectional link between periodontitis and diabetes. *Dentistry Journal, 13*(3), 100. https://doi.org/10.3390/dj13030100
15. Păunică, I., Giurgiu, M., Dumitriu, A. S., Păunică, S., Pantea Stoian, A. M., Martu, M. A., & Serafinceanu, C. (2023). The bidirectional relationship between periodontal disease and diabetes mellitus: A review. *Diagnostics, 13*(4), 681. https://doi.org/10.3390/diagnostics13040681
16. Harrandah, A. M. (2025). The oral-gut-systemic axis: Emerging insights into periodontitis, microbiota dysbiosis, and systemic disease interplay. *Diagnostics, 15*(21), 2784. https://doi.org/10.3390/diagnostics15212784
17. Orlandi, M., Masi, S., Lucenteforte, E., Bhowruth, D., Malanima, M. A., Darbar, U., Patel, K., Lim, C., Curra, C., & Shiehfung, T. (2025). Periodontitis treatment and progression of carotid intima-media thickness: A randomized trial. *European Heart Journal*. https://doi.org/10.1093/eurheartj/ehaf555
18. Xie, M., Tang, Q., Yu, S., Sun, J., Mei, F., Zhao, J., & Chen, L. (2020). Porphyromonas gingivalis disrupts vascular endothelial homeostasis in a TLR-NF-κB axis dependent manner. *International Journal of Oral Science, 12*, Article 28. https://doi.org/10.1038/s41368-020-00091-1
19. Ferrara, E., D'Albenzio, A., Bassignani, J., Di Tanna, I., Murmura, G., & Balice, G. (2025). The periodontal-cardiovascular disease association: Molecular mechanisms and clinical implications. *International Journal of Molecular Sciences, 26*(16), 7710. https://doi.org/10.3390/ijms26167710
20. Meng, R., Xu, J., Fan, C., Liao, H., Wu, Z., & Zeng, Q. (2024). Effect of non-surgical periodontal therapy on risk markers of cardiovascular disease: A systematic review and meta-analysis. *BMC Oral Health, 24*, Article 692. https://doi.org/10.1186/s12903-024-0692-0
21. Shrivastava, D., Alanazi, A. R. F., Altimani, Z. M. A., Alsalem, A. M., Alam, M. K., & Srivastava, K. C. (2024). The impact of periodontal therapy on systemic inflammatory markers and cardiovascular disease risk in patients with chronic periodontitis. *Journal of Pharmacy & Bioallied Sciences, 16*(Suppl. 4), S3901–S3903. https://doi.org/10.4103/jpbs.jpbs_1216_24
22. Dioguardi, M., Guerra, C., Laterza, P., Illuzzi, G., Sovereto, D., Laneve, E., Martella, A., Lo Muzio, L., & Ballini, A. (2025). Mapping review of the correlations between periodontitis, dental caries, and endocarditis. *Dentistry Journal, 13*(5), 215. https://doi.org/10.3390/dj13050215
23. Xiang, D. D., Sun, Y. X., Jiao, C., Guo, Y. Q., Fei, Y. X., Ren, B. Q., He, X. T., & Li, X. (2025). Diabetes and periodontitis: The role of a high-glucose microenvironment in periodontal tissue cells and corresponding therapeutic strategies. *Stem Cell Research & Therapy, 16*(1), 366. https://doi.org/10.1186/s13287-025-04441-z
24. Sgolastra, F., Severino, M., Pietropaoli, D., Gatto, R., & Monaco, A. (2013). Effectiveness of periodontal treatment to improve metabolic control in patients with chronic periodontitis and type 2 diabetes: A meta-analysis of randomized clinical trials. *Journal of Periodontology, 84*(7), 958–973. https://doi.org/10.1902/jop.2012.120377
25. Wagaskar, P., Gaikwad, S., Suryawanshi, M., & Patil, A. (2025). The link between oral health and neurodegeneration: A review of periodontitis in Alzheimer's and Parkinson's disease and dementia. *Inflammopharmacology, 33*(9), 5023–5036. https://doi.org/10.1007/s10787-025-01888-7
26. Vo, T. T. T., Tsai, M.-H., Cheng, C.-Y., Wang, Y.-L., Lee, W.-J., & Lee, I.-T. (2025). The oral microbiome–redox–inflammation axis in neurodegeneration: Mechanistic insights and therapeutic perspectives. *Biochemical Pharmacology, 242*(Part 3), 117362.
27. Tang, D., Sun, C., Yang, J., Fan, L., & Wang, Y. (2023). Advances in the study of the pathology and treatment of Alzheimer's disease and its association with periodontitis. *Life, 13*(11), 2203. https://doi.org/10.3390/life13112203

28. Dominy, S. S., Lynch, C., Ermini, F., Benedyk, M., Marczyk, A., Konradi, A., Nguyen, M., Haditsch, U., Raha, D., Griffin, C., Holsinger, L. J., Arastu-Kapur, S., Kaba, S., Lee, A., Ryder, M. I., Potempa, B., Mydel, P., … Potempa, J. (2019). Porphyromonas gingivalis in Alzheimer's disease brains: Evidence for disease causation and treatment with small-molecule inhibitors. *Science Advances, 5*(1), eaau3333. https://doi.org/10.1126/sciadv.aau3333
29. Olsen, I., Kell, D. B., & Pretorius, E. (2020). Is Porphyromonas gingivalis involved in Parkinson's disease? *European Journal of Clinical Microbiology & Infectious Diseases, 39*(11), 2013–2018. https://doi.org/10.1007/s10096-020-03944-2
30. Ciongaru, D. N., Dumitriu, A.-S., Dimitriu, B. A., Paunica, S., Giurgiu, M. C., Mocanu, B. F., Popescu, G. A., & Pituru, S. M. (2024). Correlation between periodontal status and Parkinson's disease: A literature review. *Journal of Mind and Medical Sciences, 11*, 24–32. https://doi.org/10.22543/2392-7674.1492
31. Ashford, M., Ziaei, H., & Rezaei, N. (2026). Impact of oral immunity on neuroinflammation and neurodegenerative diseases. In *Advances in Experimental Medicine and Biology* (Vol. 1492, pp. 255–272).
32. Felicetti, A., Azzolino, D., Piro, P. P., Lopes, G. C. D., Rezaeinezhad, N., Lovero, R., Bocchio-Chiavetto, L., Colella, M., & Passarelli, P. C. (2025). The oral–brain axis in Alzheimer's disease: From microbial dysbiosis to neurodegeneration. *Microorganisms, 13*, 2741. https://doi.org/10.3390/microorganisms13122741
33. Gupta, A., Saleena, L. M., Kannan, P., & Shivachandran, A. (2024). The impact of oral diseases on respiratory health and the influence of respiratory infections on the oral microbiome. *Journal of Dentistry, 148*, 105213.
34. Scannapieco, F. A. (1999). Role of oral bacteria in respiratory infection. *Journal of Periodontology, 70*(7), 793–802. https://doi.org/10.1902/jop.1999.70.7.793
35. Gomes-Filho, I. S., Passos, J. S., & Seixas da Cruz, S. (2010). Respiratory disease and the role of oral bacteria. *Journal of Oral Microbiology, 2*, 5811.
36. Gomes-Filho, I. S., Cruz, S. S. D., Trindade, S. C., Passos-Soares, J. S., Carvalho-Filho, P. C., Figueiredo, A. C. M. G., Lyrio, A. O., Hintz, A. M., Pereira, M. G., & Scannapieco, F. (2020). Periodontitis and respiratory diseases: A systematic review with meta-analysis. *Oral Diseases, 26*(2), 439–446. https://doi.org/10.1111/odi.13228
37. Zhao, T., Wu, X., Zhang, Q., Li, C., Worthington, H. V., & Hua, F. (2020). Oral hygiene care for critically ill patients to prevent ventilator-associated pneumonia. *Cochrane Database of Systematic Reviews, 2020*(12), CD008367. https://doi.org/10.1002/14651858.CD008367.pub4
38. Daalderop, L. A., Wieland, B. V., Tomsin, K., Reyes, L., Kramer, B. W., Vanterpool, S. F., & Been, J. V. (2018). Periodontal disease and pregnancy outcomes: Overview of systematic reviews. *JDR Clinical & Translational Research, 3*(1), 10–27. https://doi.org/10.1177/2380084417731097
39. Bhavsar, N. V., Trivedi, S., Vachhani, K. S., Brahmbhatt, N., Shah, S., Patel, N., Gupta, D., & Periasamy, R. (2023). Association between preterm birth and low birth weight and maternal chronic periodontitis: A hospital-based case-control study. *Dental and Medical Problems, 60*(2), 207–217. https://doi.org/10.17219/dmp/152234
40. Wu, M., Chen, S. W., Su, W. L., Zhu, H. Y., Ouyang, S. Y., Cao, Y. T., & Jiang, S. Y. (2016). Sex hormones enhance gingival inflammation without affecting IL-1β and TNF-α in periodontally healthy women during pregnancy. *Mediators of Inflammation, 2016*, 4897890. https://doi.org/10.1155/2016/4897890
41. Zachariasen, R. D. (1993). The effect of elevated ovarian hormones on periodontal health: Oral contraceptives and pregnancy. *Women & Health, 20*(2), 21–30. https://doi.org/10.1300/J013v20n02_02
42. Offenbacher, S., Katz, V., Fertik, G., Collins, J., Boyd, D., Maynor, G., McKaig, R., & Beck, J. (1996). Periodontal infection as a possible risk factor for preterm low birth weight. *Journal of Periodontology, 67*(10 Suppl.), 1103–1113. https://doi.org/10.1902/jop.1996.67.10s.1103
43. Vergnes, J. N., & Sixou, M. (2007). Preterm low birth weight and maternal periodontal status: A meta-analysis. *American Journal of Obstetrics and Gynecology, 196*(2), 135.e1–135.e7. https://doi.org/10.1016/j.ajog.2006.09.028
44. Dogra, S., Targotra, S., & Kour, T. (2025). Association of periodontal disease with adverse pregnancy outcomes. *European Journal of Cardiovascular Medicine, 15*(9), 448–454.
45. Han, Y. W., Redline, R. W., Li, M., Yin, L., Hill, G. B., & McCormick, T. S. (2004). Fusobacterium nucleatum induces premature and term stillbirths in pregnant mice: Implication of oral bacteria in preterm birth. *Infection and Immunity, 72*(4), 2272–2279. https://doi.org/10.1128/IAI.72.4.2272-2279.2004
46. Vander Haar, E. L., So, J., Gyamfi-Bannerman, C., & Han, Y. W. (2018). Fusobacterium nucleatum and adverse pregnancy outcomes: Epidemiological and mechanistic evidence. *Anaerobe, 50*, 55–59. https://doi.org/10.1016/j.anaerobe.2018.01.008
47. Wen, X., Fu, X., Zhao, C., Yang, L., & Huang, R. (2023). The bidirectional relationship between periodontal disease and pregnancy via the interaction of oral microorganisms, hormone and immune response. *Frontiers in Microbiology, 14*, 1070917. https://doi.org/10.3389/fmicb.2023.1070917
48. Islam, N. A. B., & Haque, A. (2024). Pregnancy-related dental problems: A review. *Heliyon, 10*(3), e24259. https://doi.org/10.1016/j.heliyon.2024.e24259
49. Räisänen, I. T., Heikkinen, A. M., Siren, E., Tervahartiala, T., Gieselmann, D.-R., Van der Schoor, G.-J., Van der Schoor, P., & Sorsa, T. (2018). Point-of-care/chairside aMMP-8 analytics of periodontal diseases' activity and episodic progression. *Diagnostics, 8*, 74. https://doi.org/10.3390/diagnostics8040074

50. Fischer, R. G., Gomes Filho, I. S., Cruz, S. S. D., Oliveira, V. B., Lira-Junior, R., Scannapieco, F. A., & Rego, R. O. (2021). What is the future of periodontal medicine? *Brazilian Oral Research, 35*(Supp. 2), e102. https://doi.org/10.1590/1807-3107bor-2021.vol35.0102
51. Hasan, F., Tandon, A., AlQallaf, H., John, V., Sinha, M., & Gibson, M. P. (2025). Inflammatory association between periodontal disease and systemic health. *Inflammation, 48*(6), 3763–3775. https://doi.org/10.1007/s10753-025-02317-1
52. Heitz-Mayfield, L. J. (2009). Systemic antibiotics in periodontal therapy. *Australian Dental Journal, 54*(Suppl. 1), S96–S101. https://doi.org/10.1111/j.1834-7819.2009.01147.x
53. Pan, Q., Zong, Z., Li, H., Xie, L., Zhu, H., Wu, D., Liu, R., He, B., & Pu, Y. (2025). Hydrogel design and applications for periodontitis therapy: A review. *International Journal of Biological Macromolecules, 284*(Pt. 1), 137893. https://doi.org/10.1016/j.ijbiomac.2024.137893
54. Nath, S., Weyrich, L., Zilm, P., Kapellas, K., & Jamieson, L. M. (2024). Oral microbiome research from a public health perspective and implications for oral health. *Community Dental Health, 41*(1), 75–82. https://doi.org/10.1922/CDH_IADR24Nath08
55. Sischo, L., & Broder, H. L. (2011). Oral health-related quality of life: What, why, how, and future implications. *Journal of Dental Research, 90*(11), 1264–1270. https://doi.org/10.1177/0022034511399918