



THE RELATIONSHIP BETWEEN PERIODONTAL DISEASES AND SYSTEMIC HEALTH CONDITIONS

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Abstract: Periodontal diseases are chronic inflammatory conditions that affect the gingiva, periodontal ligament, cementum, and alveolar bone and may contribute to the total inflammatory burden of the body.

Background: Epidemiological and mechanistic research has linked periodontitis with diabetes, atherosclerotic cardiovascular disease, respiratory disease, adverse pregnancy outcomes, rheumatoid arthritis, chronic kidney disease, and other systemic conditions. Shared risk factors and residual confounding make causal interpretation difficult.

Objective: To critically analyse the biological pathways, strength of association, intervention evidence, and practical implications of the relationship between periodontal and systemic diseases.

Materials and methods: Narrative synthesis of systematic reviews, consensus reports, cohort studies, randomised trials, and international guidance published from 2012 to 2026. Evidence was classified as association, mechanistic plausibility, or improvement in systemic outcomes after periodontal intervention.

Results: The most consistent bidirectional relationship is between periodontitis and diabetes. Non-surgical periodontal therapy may produce a modest short-term improvement in HbA1c, but it does not replace medical treatment. Associations with cardiovascular, respiratory, pregnancy-related, and immune-inflammatory outcomes are clinically relevant but do not prove that periodontal treatment prevents major systemic events. Conclusion: Dental and primary-care professionals should use shared risk assessment, screening, referral, and supportive periodontal care while avoiding exaggerated causal claims.

Keywords: periodontitis, systemic inflammation, diabetes, cardiovascular disease, respiratory disease, integrated care

INTRODUCTION

Periodontal diseases are common inflammatory disorders affecting the tissues that support the teeth. Gingivitis is usually limited to the gingiva, occurs without attachment loss, and may be reversible when biofilm control is restored. Periodontitis involves dysbiotic subgingival biofilm, a susceptible host response, clinical attachment loss, and alveolar bone destruction. The condition is not biologically isolated from the rest of the body. The ulcerated epithelium of periodontal pockets can permit microorganisms, microbial products, and inflammatory mediators to enter the circulation during daily activities such as chewing and toothbrushing [1–3].

During the past several decades, periodontitis has been studied in relation to diabetes, atherosclerotic cardiovascular disease, chronic obstructive pulmonary disease, pneumonia, adverse pregnancy outcomes, rheumatoid arthritis, chronic kidney disease, and neurodegenerative disorders. The 2023 EFP–WONCA Europe consensus recognised independent associations with cardiovascular disease, diabetes, COPD, obstructive sleep apnoea, and selected respiratory outcomes and recommended closer cooperation between dental and family-medicine teams [1]. Association, however, is not synonymous with causation. Age, smoking, obesity, diet, socioeconomic conditions, medication use, health literacy, and access to care can influence both oral and systemic disease.

The purpose of this review is to distinguish robust evidence from plausible but unproven claims, explain the biological and epidemiological links between periodontal inflammation and systemic health, and propose a practical screening and referral pathway. The clinical message must remain balanced: periodontal treatment is unquestionably indicated to protect oral function and quality of life, but it should not be promoted as a guaranteed stand-alone method for preventing myocardial infarction, stroke, premature birth, or other major systemic outcomes.



Figure 1. Clinical appearance of gingival recession associated with periodontal tissue damage

Source: Wikimedia Commons, “Recession parodontale.jpg”, author Damdent, CC BY-SA/GFDL;
https://commons.wikimedia.org/wiki/File:Recession_parodontale.jpg

MATERIALS AND METHODS

For this narrative review, systematic reviews, consensus reports, clinical trials, and relevant observational studies published from 2012 to 2026 were identified in PubMed, the Cochrane Library, Journal of Clinical Periodontology, Nature Communications, European Journal of General Practice, and World Health Organization resources. Search concepts included “periodontitis systemic disease”, “periodontitis diabetes”, “periodontitis cardiovascular disease”, “periodontal treatment HbA1c”, “periodontitis respiratory disease”, and “periodontitis pregnancy outcomes”.



Evidence was interpreted at three levels: first, association—the two conditions occur together more often than expected; second, mechanistic plausibility—microbial, inflammatory, vascular, metabolic, or immune pathways could connect them; and third, intervention evidence—treating periodontitis improves a clinically meaningful systemic outcome. Cross-sectional studies with limited control of confounding were assigned less weight than prospective cohorts, randomised trials, and well-conducted meta-analyses. Conclusions were grouped by clinical relevance without converting statistical association into unsupported causality.

RESULTS

1. Biological mechanisms linking periodontal and systemic disease

The ulcerated surface of a periodontal pocket may serve as a portal for transient bacteraemia during chewing, toothbrushing, or professional instrumentation. *Porphyromonas gingivalis*, *Aggregatibacter actinomycetemcomitans*, other organisms, and their lipopolysaccharides can activate innate and adaptive immune responses in vascular endothelium and distant tissues. Detection of microbial DNA or antigens outside the mouth does not prove that the organism initiated the systemic disease; it may have reached tissue already altered by inflammation or vascular injury. Mechanistic findings must therefore be interpreted as plausibility rather than definitive causal proof.

A second pathway is an increase in the total systemic inflammatory burden. Periodontitis is associated with increased signalling involving IL-1 β , IL-6, TNF- α , prostaglandins, C-reactive protein, and oxidative stress. These pathways may influence insulin signalling, endothelial function, lipid metabolism, coagulation, and immune regulation. A third pathway consists of shared determinants: smoking, uncontrolled hyperglycaemia, obesity, stress, inadequate sleep, low physical activity, poor diet, and socioeconomic disadvantage raise the risk of both periodontitis and many non-communicable diseases [2,3]. Genetic and immune phenotypes may also amplify inflammatory responses in several organ systems.

Table 1. Relationships between periodontitis and major systemic conditions

Systemic condition	Nature of the evidence	Plausible link	Practical implication
Diabetes mellitus	Strong, consistent bidirectional association; intervention evidence for modest short-term HbA1c improvement	Hyperglycaemia, AGE–RAGE signalling, oxidative stress, impaired healing, and systemic inflammation	Assess periodontal status in diabetes; refer severe unexplained periodontitis or hyperglycaemic symptoms for testing



Systemic condition	Nature of the evidence	Plausible link	Practical implication
Atherosclerotic cardiovascular disease	Consistent association and biological plausibility; insufficient proof that treatment prevents major events	Bacteraemia, endothelial activation, inflammation, thrombosis, and shared risk factors	Record blood pressure, smoking, obesity, and family history; refer high-risk patients
Respiratory disease and pneumonia	Association stronger in frail, dependent, hospitalised, or aspiration-prone populations	Oral reservoir of respiratory pathogens and microaspiration	Strengthen oral hygiene and professional care in vulnerable groups
Adverse pregnancy outcomes	Observational association; intervention trials inconsistent	Inflammatory mediators, bacteraemia, and common social or behavioural determinants	Provide necessary periodontal care and hygiene support without promising prevention of preterm birth
Rheumatoid arthritis and other inflammatory conditions	Association and shared pathways; causality remains uncertain	Chronic inflammation, smoking, immune dysregulation, and citrullination	Treat periodontal disease, document systemic symptoms, and communicate with medical teams
Kidney and cognitive conditions	Associations have been reported; certainty is low to moderate	Chronic inflammation, vascular injury, and shared risk factors	May justify screening awareness; no causal conclusion should be drawn

2. Bidirectional relationship with diabetes mellitus

The relationship between diabetes and periodontitis is the best characterised. Persistent hyperglycaemia increases the formation of advanced glycation end-products, which interact with RAGE receptors and promote inflammation, oxidative stress, impaired collagen turnover,



microvascular dysfunction, and delayed wound healing. Neutrophil function and resolution of inflammation may also be altered. Consequently, periodontal destruction may progress more rapidly and response to treatment may be less favourable in patients with poorly controlled diabetes [4].

In the opposite direction, a large burden of inflamed periodontal tissue may worsen insulin resistance. A Cochrane review found that periodontal therapy in people with diabetes was associated with an average HbA1c reduction of approximately 0.4 percentage points at three to four months, with a smaller effect at six months [5]. This is a useful adjunctive benefit, not a replacement for medication, nutrition, physical activity, or medical follow-up. Dental referral for glucose or HbA1c testing is reasonable when severe periodontitis is accompanied by polyuria, polydipsia, unexplained weight loss, recurrent infection, or delayed healing. Medical teams should likewise ask patients with diabetes about gingival bleeding, tooth mobility, and the timing of their last dental examination.

3. Cardiovascular and respiratory diseases

Severe periodontitis is statistically associated with atherosclerotic cardiovascular disease. Periodontal bacterial components have been detected in atheromatous plaques, and periodontal treatment can improve C-reactive protein levels and selected surrogate measures of endothelial function [6]. These findings support biological plausibility but do not establish that periodontal therapy reduces myocardial infarction or stroke. The appropriate practical response is to recognise common risk factors. A patient with periodontitis should not have hypertension, smoking, obesity, dyslipidaemia, family history, or symptoms of cardiovascular disease ignored, and high-risk findings should trigger referral to primary care.

The oral biofilm can act as a reservoir for potential respiratory pathogens. In frail older adults, dependent patients, those with swallowing impairment, and intubated or hospitalised individuals, microaspiration may contribute to pneumonia risk. The coexistence of COPD and periodontitis is partly explained by smoking and social determinants. Consistent oral hygiene and professional care are therefore rational components of prevention in vulnerable groups, but periodontitis should not be described as the direct cause of every respiratory exacerbation [1,2].



Figure 2. Radiographic appearance of alveolar bone loss in periodontitis

Source: Wikimedia Commons, “Periodontalbone loss.JPG”, author DRosenbach, public domain;
<https://commons.wikimedia.org/wiki/File:Periodontalbone loss.JPG>

4. Pregnancy outcomes and immune-inflammatory diseases

Hormonal changes during pregnancy can intensify the gingival response to biofilm. Observational studies have reported associations between periodontitis and preterm birth, low birth weight, and pre-eclampsia [7]. Nevertheless, intervention trials have not consistently shown that periodontal treatment during pregnancy prevents these outcomes. Necessary periodontal care can be provided safely with appropriate obstetric coordination, and women should receive oral-hygiene support before and during pregnancy. The evidence does not justify promising that periodontal therapy alone will prevent premature birth.

Rheumatoid arthritis and periodontitis share chronic inflammatory pathways, smoking exposure, altered immune regulation, and potential mechanisms involving citrullination. Associations have also been reported with chronic kidney disease and cognitive decline, but the certainty and direction of causality vary considerably. These conditions warrant awareness and multidisciplinary communication, not automatic causal attribution. Clinicians should treat active



periodontal disease according to dental indications and refer systemic warning signs through established medical pathways.

Table 2. Practical screening pathway between dental and primary-care settings

Setting	Finding or question	Recommended action	Avoid
Dental practice	Severe periodontitis with no known diabetes, delayed healing, polyuria, polydipsia, or unexplained weight loss	Refer for fasting glucose and/or HbA1c assessment	Diagnosing diabetes solely from oral findings
Dental practice	Hypertension, smoking, obesity, chest symptoms, or high cardiovascular risk	Measure or confirm risk factors and refer to primary care when indicated	Claiming that periodontal therapy prevents myocardial infarction or stroke
Primary care	Diabetes with gingival bleeding, halitosis, mobility, recession, or long absence from dental care	Recommend comprehensive periodontal assessment	Treating oral symptoms only with antibiotics
Pregnancy care	Bleeding, swelling, pain, or known periodontitis	Encourage dental examination and safe necessary treatment	Delaying all dental care until after delivery
Shared follow-up	Completed periodontal treatment in a high-risk patient	Exchange diagnosis, treatment, medication, risk-factor status, and maintenance plan	Vague communication without a specific clinical question

INTEGRATED CLINICAL MANAGEMENT

Systemic risk assessment in a periodontal patient does not require an extensive laboratory programme in every dental office. It begins with a focused history: diabetes diagnosis and control, blood pressure, smoking, body weight, respiratory disease, pregnancy, medication, cardiovascular events, and recent medical review. Severe periodontitis in a patient with no known diabetes, repeated oral infection, delayed healing, or classic hyperglycaemic symptoms warrants medical



testing. Conversely, primary-care professionals should recommend periodontal assessment when patients with diabetes report bleeding gums, halitosis, recession, tooth mobility, or difficulty chewing.

The treatment plan should separate active therapy from supportive periodontal care. Active treatment includes patient-specific oral-hygiene instruction, control of local retentive factors, supra- and subgingival instrumentation, management of acute infection, and surgery when appropriately indicated. Maintenance intervals are determined by stage, grade, smoking, diabetes control, plaque levels, residual pocketing, and adherence. Periodontal treatment cannot compensate for continued smoking or persistently poor glycaemic control; risk-factor modification and supportive care are integral parts of therapy rather than optional additions.

Information exchanged between professionals should be concise and clinically actionable. A dental referral should describe periodontal diagnosis, severity, bleeding, relevant symptoms, treatment provided, and the specific reason for medical assessment. A medical response should communicate diagnoses, current medications, glycaemic or cardiovascular control, and precautions relevant to dental care. This prevents vague statements such as “infection may affect the heart” from creating fear without changing management.

DISCUSSION

The relationship between periodontitis and systemic disease broadens the role of oral healthcare but also requires disciplined interpretation. The most defensible conclusion is that periodontitis contributes to inflammatory burden and shares determinants with major chronic diseases. The evidence is strongest for the bidirectional diabetes relationship. For cardiovascular, respiratory, pregnancy-related, rheumatological, renal, and neurological outcomes, associations and plausible mechanisms exist, but intervention evidence is weaker or inconsistent. A clinically useful review must preserve these distinctions.

Systemic outcomes are influenced by multiple factors. Scaling and root planing reduce subgingival biofilm and periodontal inflammation, but blood pressure, HbA1c, vascular events, and pregnancy outcomes are also shaped by medication, diet, physical activity, smoking, genetics, social context, and adherence. Small improvements in biomarkers should not be converted into claims of prevention of major events. Periodontal treatment should be offered because it preserves teeth and improves oral health, with potential systemic benefits presented as additional and probabilistic.

Umbrella reviews confirm that oral health is linked with several non-communicable diseases, yet they also identify heterogeneity, residual confounding, reverse causation, and publication bias [3]. Future research should use standardised periodontal definitions, adequately measured confounders, long-term follow-up, and clinically meaningful endpoints. Integrated care should be



evaluated not only by laboratory markers but by referral completion, smoking cessation, diabetes detection, periodontal stability, quality of life, and cost-effectiveness.

CONCLUSION

Periodontitis is a locally destructive disease that may also add to systemic inflammatory burden. The most reliable bidirectional relationship is with diabetes; periodontal treatment can modestly improve short-term glycaemic control but does not replace medical management. Cardiovascular, respiratory, pregnancy-related, and immune-inflammatory associations are clinically important yet should not be overstated as proven causation. The safest and most effective approach is integrated care: identify shared risk factors, provide evidence-based periodontal treatment and maintenance, screen for warning signs, communicate clearly across professions, and refer patients when findings fall outside the dental scope of practice.

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