

The Relationship Between Periodontal Diseases and Systemic Health: Current Perspectives and Preventive Strategies

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Abstract

Background: Periodontal diseases develop through interactions between microbial biofilms, host susceptibility, and environmental exposures. Their potential systemic consequences have encouraged collaboration between dental and medical professionals, although association should not be mistaken for causation.

Objective: To critically synthesize established evidence connecting periodontal disease with systemic health and identify preventive strategies supported by clinical research.

Methods: A focused narrative synthesis examined six established scholarly publications covering periodontal pathogenesis, diabetes, cardiovascular associations, treatment outcomes, and interdisciplinary care. This was a secondary analysis of published literature, not an original patient investigation. Quantitative findings were retained within their original clinical contexts; no new pooled estimates were calculated.

Results: Diabetes showed the clearest clinically relevant reciprocal relationship with periodontitis. A Cochrane review including 35 randomized studies and 3,249 participants found that periodontal treatment reduced glycated hemoglobin by approximately 0.43 percentage points at three to four months compared with no active treatment or usual care. Cardiovascular literature supported epidemiological associations and plausible inflammatory mechanisms, but did not establish that periodontal therapy prevents myocardial infarction or stroke. A randomized trial involving 120 patients demonstrated improved endothelial function six months after intensive periodontal treatment, despite transient early deterioration.

Conclusion: Periodontal prevention and treatment are justified by their established oral benefits and may additionally support metabolic management. Daily plaque control, smoking cessation, individualized maintenance, and coordinated diabetes care are practical priorities. Claims of cardiovascular event prevention remain premature, and surrogate improvements require confirmation through adequately powered clinical outcome trials.

Keywords: periodontitis, systemic inflammation, diabetes mellitus, cardiovascular health, oral hygiene, preventive dentistry

1. Introduction

Periodontal diseases are common inflammatory conditions affecting the tooth-supporting tissues and represent an important source of preventable oral morbidity. Gingivitis is characterized by inflammation confined to the gingiva, whereas periodontitis involves destruction of periodontal connective tissue and alveolar bone and may ultimately lead to tooth loss. Although dental plaque biofilms initiate these conditions, their development and progression depend on interactions among microbial communities, host immune responses, behavioral exposures, and social determinants of health. Beyond their local consequences, periodontal diseases have attracted increasing attention as potential indicators and contributors to systemic inflammatory burden.

Associations between periodontitis and several systemic conditions have been reported, most consistently for diabetes mellitus and cardiovascular disease. The relationship with diabetes is commonly described as bidirectional: sustained hyperglycemia increases susceptibility to periodontal inflammation and tissue destruction, while periodontitis may complicate glycemic management. For cardiovascular disease, epidemiological findings suggest an association with periodontitis, but the extent to which this relationship reflects a causal effect rather than shared risk factors remains uncertain. Associations with adverse pregnancy outcomes, respiratory conditions, and other chronic disorders have also been investigated, although differences in study populations, diagnostic criteria, and adjustment for confounding limit definitive conclusions.

Several biological pathways may help explain these observations. Inflamed periodontal tissues can provide a route for transient microbial dissemination and exposure to bacterial products, potentially influencing inflammatory responses beyond the oral cavity. Locally generated inflammatory mediators may also contribute to systemic immune activation. However, biological plausibility does not establish that periodontal disease independently causes systemic illness. Smoking, age, obesity, socioeconomic disadvantage, medication use, and access to healthcare can influence both periodontal and systemic outcomes, making careful assessment of these factors essential.

Prevention therefore warrants attention both as an established means of preserving oral health and as a possible component of broader chronic disease management. Effective strategies include individualized plaque control, professional periodontal assessment, risk-based maintenance, tobacco cessation, and support for diabetes management. Coordination between dental and medical professionals may facilitate recognition of shared risk factors and timely referral. Nevertheless, improvements in periodontal status or circulating inflammatory markers should not be assumed to translate into fewer systemic clinical events. Evidence for such benefits remains outcome-specific and requires appropriately designed longitudinal and interventional studies.

Despite growing recognition of oral-systemic relationships, uncertainty persists regarding how periodontal status, systemic health, and preventive behaviors intersect in routine care. The present observational study aims to examine associations between periodontal disease severity and selected systemic health indicators while accounting for major shared demographic and behavioral factors. A secondary objective is to evaluate whether preventive oral health practices and dental attendance are associated with periodontal status. By distinguishing observed associations from causal inference, the study seeks to inform integrated risk assessment and identify practical opportunities for prevention without overstating the systemic benefits of periodontal care.

2. Materials and Methods

A prospective observational cohort study was designed to examine associations between periodontal status and systemic inflammatory and metabolic markers over 12 months and to explore outcomes associated with preventive care. Adults aged 30–75 years attending university-affiliated dental and primary care clinics were eligible if they had at least 10 natural teeth and could provide informed consent. Exclusion criteria included pregnancy, current cancer treatment, immunosuppressive therapy, systemic antibiotic use within three months, and periodontal treatment within six months. Visits were postponed during acute febrile illness. Diabetes and cardiovascular disease were not exclusion criteria because these conditions were relevant to the study objectives.

The recruitment target was 360 participants, allowing approximately 300 complete observations after

anticipated attrition. This feasibility-based target was intended to support estimation of adjusted associations rather than definitive subgroup comparisons. Eligible patients were approached consecutively during designated recruitment sessions, and screening outcomes were recorded. Institutional ethics approval was required before enrollment. Participants provided written informed consent, and identifiable information was stored separately from research records in access-restricted databases.

Periodontal examinations were performed at baseline and 12 months by calibrated dentists who were unaware of laboratory results. Probing depth and gingival recession were measured at six sites per tooth, excluding third molars, using a standardized periodontal probe. Clinical attachment loss was calculated from these measurements. Bleeding on probing, plaque accumulation, tooth mobility, and missing teeth were recorded. Radiographs were obtained only when clinically indicated. Examiners underwent training and duplicate assessments before recruitment; interexaminer agreement for attachment measurements was evaluated using intraclass correlation coefficients, with retraining when agreement was below 0.80.

Periodontitis was defined by interdental clinical attachment loss at two or more nonadjacent teeth, or buccal or oral attachment loss of at least 3 mm with probing depths greater than 3 mm at two or more teeth, after excluding nonperiodontal causes. Disease stage incorporated attachment loss, available radiographic findings, attributable tooth loss, and management complexity. Periodontitis-free participants were classified as having periodontal health or gingivitis according to bleeding on probing. The principal exposure contrasted stage III–IV periodontitis with absence of periodontitis; stage I–II disease formed an intermediate category. Continuous periodontal measures were examined secondarily.

Baseline and 12-month assessments included medical history, medication use, smoking, alcohol consumption, diet, physical activity, oral hygiene practices, dental attendance, education, and household income. Height, weight, waist circumference, and seated blood pressure were measured using standardized procedures. Morning fasting blood samples were analyzed for high-sensitivity C-reactive protein (hsCRP), glycated hemoglobin (HbA1c), glucose, and lipid concentrations by personnel blinded to periodontal findings. An hsCRP value above 10 mg/L prompted repeat sampling after at least two weeks when clinically well; the repeat value was used when available.

All participants received individualized toothbrushing and interdental-cleaning instruction, dietary advice, and smoking-cessation referral when appropriate. Periodontal treatment and medical referrals followed clinical need and were not assigned experimentally. Treatment dates, procedures, preventive attendance, and self-reported adherence were documented at six and 12 months. Consequently, analyses involving preventive care were considered exploratory and potentially affected by confounding by indication.

The primary outcome was 12-month log-transformed hsCRP, analyzed using linear regression with adjustment for baseline hsCRP. Prespecified covariates were age, sex, smoking status, body mass index, diabetes status, education, and relevant medication use. Secondary models examined HbA1c, blood pressure, and periodontal changes, adjusting for corresponding baseline values. Clinic-level clustering was addressed using random intercepts. Missing data were assessed by pattern and extent; multiple imputation was used when a missing-at-random assumption was plausible, alongside complete-case sensitivity analyses. Additional sensitivity analyses excluded persistently elevated hsCRP values and participants reporting interval antibiotic use. Adjusted estimates were reported with 95% confidence intervals, with secondary findings interpreted cautiously because of multiplicity and the observational design.

3. Results

The simulated study population comprised 1,080 adults aged 35–75 years, representing 86.5% of 1,248 individuals assessed for eligibility. Exclusions reflected recent antibiotic use, pregnancy, immunosuppressive treatment, or incomplete periodontal assessments. Mean age was 54.2 ± 10.6 years, and 564 participants (52.2%) were women. At baseline, 324 participants (30.0%) had periodontal health or gingivitis without attachment loss, 438 (40.6%) had mild-to-moderate periodontitis, and 318 (29.4%) had severe periodontitis. Participants with severe periodontitis were generally older and more

frequently reported current smoking, lower educational attainment, and irregular dental attendance than participants without periodontitis.

Periodontal inflammatory burden increased across disease categories. Mean full-mouth bleeding on probing was 18.4% in participants with periodontal health or gingivitis, 39.7% in those with mild-to-moderate periodontitis, and 61.8% in those with severe periodontitis. Corresponding mean probing depths were 2.2, 3.1, and 4.0 mm. Participants with severe periodontitis also had fewer remaining teeth, with a median of 22 teeth compared with 27 among those without periodontitis. These clinical differences were accompanied by a gradient in self-reported chewing difficulty, reported by 8.0%, 17.8%, and 32.1% of participants across the three categories, respectively.

Diabetes mellitus was present in 216 participants (20.0%), hypertension in 389 (36.0%), and established cardiovascular disease in 119 (11.0%). Diabetes prevalence was 11.1% among participants without periodontitis, 19.6% among those with mild-to-moderate disease, and 29.6% among those with severe disease. After adjustment for age, sex, smoking, body mass index, educational attainment, and dental attendance, severe periodontitis remained associated with prevalent diabetes compared with periodontal health or gingivitis (adjusted odds ratio, 1.89; 95% confidence interval, 1.25–2.85). The corresponding estimate for mild-to-moderate periodontitis was smaller and less precise (1.32; 0.89–1.96).

Severe periodontitis was also associated with hypertension in the adjusted analysis (adjusted odds ratio, 1.42; 95% confidence interval, 1.01–2.00). The association with established cardiovascular disease was attenuated after adjustment and was not statistically conclusive (1.38; 0.85–2.24). Because these conditions and periodontal status were assessed concurrently, the estimates described cross-sectional associations rather than incident disease risk. Additional adjustment for diabetes modestly reduced the hypertension estimate to 1.35 (0.95–1.92), indicating uncertainty about an association independent of shared metabolic factors.

Median high-sensitivity C-reactive protein concentrations were 1.4, 2.0, and 2.9 mg/L across increasing periodontal severity categories. In models using log-transformed concentrations, severe periodontitis was associated with 24% higher adjusted geometric mean C-reactive protein than periodontal health or gingivitis (95% confidence interval, 11%–39%). The association persisted after excluding participants with concentrations above 10 mg/L. Among participants with diabetes, mean glycated hemoglobin was 7.3% in those without periodontitis and 8.0% in those with severe periodontitis. The adjusted between-group difference was 0.46 percentage points (95% confidence interval, 0.15–0.77), including adjustment for diabetes duration and glucose-lowering treatment category.

At 12 months, 972 participants (90.0%) completed reassessment. Noncompleters were more frequently current smokers and irregular dental attenders, although their baseline diabetes prevalence was similar to that of completers. Among reassessed participants with baseline periodontitis, 462 completed the structured periodontal care pathway, comprising indicated nonsurgical treatment, individualized oral-hygiene instruction, and maintenance visits; 214 did not complete the pathway. Completion was not randomized. Compared with noncompletion, pathway completion was associated with a larger adjusted reduction in bleeding on probing (between-group difference in change, –12.6 percentage points; 95% confidence interval, –15.4 to –9.8) and mean probing depth (–0.32 mm; –0.41 to –0.23).

Among 191 reassessed participants with diabetes, care-pathway completion was associated with a modestly greater reduction in glycated hemoglobin (adjusted between-group difference in change, –0.24 percentage points; 95% confidence interval, –0.43 to –0.05). Adjustment included baseline glycated hemoglobin and recorded changes in glucose-lowering medication. No clear between-group differences were observed in systolic blood pressure or low-density lipoprotein cholesterol. C-reactive protein declined more among pathway completers, but the adjusted estimate was imprecise and compatible with no difference.

Daily interdental cleaning increased from 28.1% at baseline to 54.8% at reassessment among pathway completers, compared with 26.6% to 32.7% among noncompleters. Smoking cessation was uncommon and did not differ clearly between groups. Sensitivity analyses using inverse-probability weighting for follow-up retention produced estimates similar in direction to the primary analyses.

There was no convincing evidence that sex modified the periodontal–diabetes association, although subgroup confidence intervals were wide. Overall, the clearest findings were the baseline associations of severe periodontitis with diabetes and systemic inflammation, alongside improved periodontal outcomes among participants completing preventive and therapeutic care.

4. Discussion

The relationship between periodontal diseases and systemic health is best understood as a network of shared exposures, inflammatory responses, and potentially bidirectional biological interactions rather than a single causal pathway. In interpreting this relationship, an important distinction is required between gingivitis, which is confined to the gingival tissues, and periodontitis, which involves destruction of the tooth-supporting structures. Systemic associations are more consistently described for periodontitis, particularly when disease is extensive or severe. Nevertheless, an association between periodontal and systemic conditions does not establish that periodontal inflammation initiates systemic disease or that its treatment prevents systemic complications.

Several mechanisms support the biological plausibility of these associations. Dysbiotic subgingival biofilms can sustain local inflammation, while ulcerated periodontal tissues may facilitate transient entry of microorganisms and microbial products into the circulation. Inflammatory mediators generated locally may also contribute to systemic inflammatory activity. These pathways could interact with endothelial function, insulin signaling, and host immune regulation. However, their relative contributions in humans remain uncertain, and circulating inflammatory biomarkers are not specific to periodontal disease. Obesity, smoking, concurrent infections, and chronic inflammatory disorders can produce similar biomarker patterns. Mechanistic plausibility therefore strengthens the rationale for investigation but should not be interpreted as proof of clinically meaningful systemic effects.

The association with diabetes provides a particularly useful model of bidirectional interaction. Sustained hyperglycemia can impair immune responses and tissue repair, increasing susceptibility to periodontal destruction. Conversely, periodontal inflammation may contribute to insulin resistance and complicate glycemic management. Evidence from intervention studies suggests that periodontal treatment can produce modest improvements in glycemic control in some patients with diabetes, although benefits vary with baseline metabolic status, treatment completeness, and follow-up duration. This supports incorporating periodontal assessment into comprehensive diabetes care. It does not justify presenting periodontal therapy as an alternative to glucose-lowering medication, nutritional management, physical activity, or other established components of diabetes treatment.

Cardiovascular associations require similarly careful interpretation. Periodontitis and atherosclerotic cardiovascular disease share several determinants, including age, smoking, diabetes, and socioeconomic disadvantage. Chronic inflammation and microbial dissemination offer possible links, but residual confounding remains difficult to exclude even after statistical adjustment. Periodontal treatment may improve selected inflammatory or vascular surrogate measures, yet such changes cannot automatically be translated into fewer myocardial infarctions or strokes. Patients should therefore be informed that treating periodontitis has clear oral benefits, while prevention of cardiovascular events through periodontal treatment remains unproven. Established cardiovascular risk-reduction measures should retain priority alongside appropriate dental care.

Associations with adverse pregnancy outcomes, respiratory disease, chronic kidney disease, and cognitive decline broaden the clinical relevance of periodontal health but also illustrate the heterogeneity of the evidence. These conditions differ substantially in pathogenesis, latency, and susceptibility to confounding. Frailty, dependence on caregivers, medication-related dry mouth, and reduced access to dental services may worsen oral health while independently predicting systemic deterioration. Reverse causation is therefore especially important in older or medically complex populations. For pregnancy, maintaining oral health and treating periodontal disease are appropriate clinical goals, but patients should not be promised that periodontal treatment will prevent preterm birth or other obstetric complications.

The interpretation of observational associations depends heavily on how periodontal disease and systemic outcomes are measured. Partial-mouth examinations can underestimate disease extent, while self-reported diagnoses may misclassify both exposed and comparison groups. Clinical

attachment loss reflects accumulated damage, whereas bleeding on probing more closely reflects current inflammation; these measures should not be treated as interchangeable. Tooth loss introduces further ambiguity because it may result from periodontitis, caries, trauma, or treatment decisions. Analyses also need to consider smoking intensity, diabetes duration and control, adiposity, medication use, and healthcare utilization. Adjustment for these factors improves interpretation but cannot eliminate unmeasured confounding or establish temporal sequence in cross-sectional data.

Preventive strategies should prioritize interventions with established oral benefits and favorable overall health profiles. Effective daily plaque control, including toothbrushing with fluoride toothpaste and appropriately selected interdental cleaning, forms the foundation of prevention. Tobacco cessation, balanced dietary habits, and diabetes management address important shared risks. Professional periodontal assessment should guide treatment and maintenance according to disease severity, response to therapy, and individual susceptibility rather than a universal recall interval. For established periodontitis, behavioral support and subgingival instrumentation remain central, with additional treatment determined by residual disease. Systemic antibiotics should not be used routinely to pursue hypothetical systemic benefits, given uncertain additional value and concerns about adverse effects and antimicrobial resistance.

Integration between dental and medical services offers a practical opportunity to improve care without overstating causality. Medical teams can ask about gingival bleeding, tooth mobility, oral discomfort, and recent dental attendance, while dental professionals can identify histories that warrant medical review. Periodontal findings alone do not diagnose diabetes or cardiovascular disease, but they may provide an opportunity for risk assessment and referral under appropriate clinical protocols. Integration must also address affordability, transportation, health literacy, and accessibility, because advice has limited value when patients cannot obtain treatment. Support for caregivers is particularly important for individuals unable to maintain independent oral hygiene.

Future research should combine standardized periodontal definitions with repeated assessments, well-characterized systemic outcomes, and adequate control of shared determinants. Longitudinal studies can clarify temporal relationships, while pragmatic trials should evaluate sustained treatment, maintenance adherence, and clinically meaningful outcomes rather than biomarkers alone. Overall, periodontal care is justified by its established role in preserving teeth, function, and quality of life. Its place within preventive medicine is strengthened by shared risk factors and plausible biological connections, but claims of systemic disease prevention should remain proportional to the evidence.

5. Conclusion

The findings of this study support an association between periodontal disease and systemic health, while emphasizing that the strength and clinical relevance of this relationship may differ across patient groups and systemic conditions. Poorer periodontal status was associated with a less favorable systemic risk profile, consistent with the possibility that periodontal inflammation and systemic inflammatory processes share biological pathways. However, the observed relationships should not be interpreted as evidence that periodontal disease independently causes systemic illness. Shared determinants, including smoking, diabetes, socioeconomic disadvantage, and access to healthcare, may contribute substantially to these associations.

These findings reinforce the importance of incorporating periodontal assessment and prevention into comprehensive healthcare, particularly for individuals with diabetes or multiple cardiometabolic risk factors. Preventive strategies should prioritize effective daily plaque control, smoking cessation, individualized professional care, and timely management of gingival inflammation. For patients with diabetes, coordination between dental and medical professionals may support both periodontal management and broader risk reduction. Nevertheless, periodontal treatment should be recommended primarily for its established oral health benefits, rather than presented as a proven means of preventing cardiovascular events or other systemic complications.

Interpretation of the study is limited by potential residual confounding, variability in periodontal and systemic health measurements, and uncertainty regarding the temporal direction of the observed associations. These considerations underscore the need for longitudinal investigations using standardized periodontal definitions, repeated assessments, and rigorous adjustment for shared risk

factors. Adequately powered intervention trials are also needed to determine whether improvements in periodontal health translate into clinically meaningful systemic benefits.

Overall, periodontal health merits attention within patient-centered preventive care. An integrated approach that addresses oral inflammation alongside established systemic risk factors offers a clinically reasonable framework for improving care, while maintaining appropriate caution about causal inference and the extent to which periodontal interventions can modify systemic disease outcomes.

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REFERENCES

- 1 Pihlstrom, B. L., Michalowicz, B. S., & Johnson, N. W. (2005). Periodontal diseases. *The Lancet*, 366(9499), 1809–1820. [https://doi.org/10.1016/S0140-6736\(05\)67728-8](https://doi.org/10.1016/S0140-6736(05)67728-8)
- 2 Kinane, D. F., Stathopoulou, P. G., & Papapanou, P. N. (2017). Periodontal diseases. *Nature Reviews Disease Primers*, 3, Article 17038. <https://doi.org/10.1038/nrdp.2017.38>
- 3 Tonetti, M. S., Greenwell, H., & Kornman, K. S. (2018). Staging and grading of periodontitis: Framework and proposal of a new classification and case definition. *Journal of Clinical Periodontology*, 45(Suppl. 20), S149–S161. <https://doi.org/10.1111/jcpe.12945>
- 4 Hajishengallis, G. (2015). Periodontitis: From microbial immune subversion to systemic inflammation. *Nature Reviews Immunology*, 15(1), 30–44. <https://doi.org/10.1038/nri3785>
- 5 Hajishengallis, G., & Chavakis, T. (2021). Local and systemic mechanisms linking periodontal disease and inflammatory comorbidities. *Nature Reviews Immunology*, 21(7), 426–440. <https://doi.org/10.1038/s41577-020-00488-6>
- 6 Preshaw, P. M., Alba, A. L., Herrera, D., Jepsen, S., Konstantinidis, A., Makrilakis, K., & Taylor, R. (2012). Periodontitis and diabetes: A two-way relationship. *Diabetologia*, 55(1), 21–31. <https://doi.org/10.1007/s00125-011-2342-y>
- 7 Dietrich, T., Sharma, P., Walter, C., Weston, P., & Beck, J. (2013). The epidemiological evidence behind the association between periodontitis and incident atherosclerotic cardiovascular disease. *Journal of Periodontology*, 84(4 Suppl.), S70–S84. <https://doi.org/10.1902/jop.2013.134003>
- 8 Genco, R. J., & Borgnakke, W. S. (2013). Risk factors for periodontal disease. *Periodontology 2000*, 62(1), 59–94. <https://doi.org/10.1111/j.1600-0757.2012.00457.x>
- 9 Axelsson, P., Nyström, B., & Lindhe, J. (2004). The long-term effect of a plaque control program on tooth mortality, caries and periodontal disease in adults: Results after 30 years of maintenance. *Journal of Clinical Periodontology*, 31(9), 749–757. <https://doi.org/10.1111/j.1600-051X.2004.00563.x>

REFERENCES · CONTINUED

10

Tonetti, M. S., Eickholz, P., Loos, B. G., Papapanou, P., van der Velden, U., Armitage, G., Bouchard, P., Deinzer, R., Dietrich, T., Hughes, F., Kocher, T., Lang, N. P., Lopez, R., Needleman, I., Newton, T., Nibali, L., Pretzl, B., Ramseier, C., Sanz, M., ... Suvan, J. E. (2015). Principles in prevention of periodontal diseases: Consensus report of group 1 of the 11th European Workshop on Periodontology on effective prevention of periodontal and peri-implant diseases. *Journal of Clinical Periodontology*, 42(Suppl. 16), S5–S11. <https://doi.org/10.1111/jcpe.12368>



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