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Obesity and Periodontal Diseases: The Emerging Role of Metabolic Health and Chronic Inflammation in Oral Disease

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Abstract

Introduction and Purpose: Obesity is a chronic metabolic disease characterized by excessive adipose tissue accumulation and persistent low-grade systemic inflammation. Periodontitis is one of the most common chronic inflammatory diseases worldwide and represents a major cause of tooth loss in adults. Increasing evidence suggests that obesity may contribute to the development and progression of periodontal diseases through shared inflammatory and metabolic pathways. The aim of this review was to summarize current evidence regarding the relationship between obesity and periodontal diseases, with particular emphasis on biological mechanisms, epidemiological findings, and clinical implications.

Material and Methods: A narrative review of the literature was conducted using PubMed/MEDLINE, Scopus, and Web of Science databases. Priority was given to systematic reviews, meta-analyses, cohort studies, and experimental investigations published between 2021 and 2026. Studies evaluating obesity-related inflammatory mechanisms, adipokines, microbiome alterations, and periodontal outcomes were included.

Results: Available evidence consistently demonstrates a positive association between obesity and periodontitis. Meta-analyses report increased odds of periodontal disease among individuals with obesity, with estimated odds ratios ranging from 1.31 to 2.46. Proposed mechanisms include chronic low-grade inflammation, dysregulated adipokine secretion, enhanced osteoclastogenesis, insulin resistance, oxidative stress, and obesity-associated alterations in the oral and gut microbiome. Evidence regarding the influence of obesity on periodontal treatment outcomes remains inconclusive.

Conclusions: Current evidence supports obesity as an important potential risk factor for periodontal diseases. The interaction between metabolic dysfunction and periodontal inflammation highlights the need for a multidisciplinary approach integrating oral health assessment with metabolic risk evaluation. Further prospective and interventional studies are required to clarify causality and determine whether weight reduction strategies can improve periodontal outcomes.

Keywords: obesity, periodontitis, metabolic health, chronic inflammation, oral health, risk factors

Introduction

Periodontal diseases rank among the most prevalent chronic inflammatory conditions worldwide, with severe periodontitis affecting an estimated 1.07 billion individuals globally.[1][2] These conditions are characterized by progressive destruction of tooth-supporting structures, potentially leading to tooth loss. Beyond oral consequences, periodontitis has been associated with cardiovascular disease, diabetes mellitus, and adverse pregnancy outcomes.[3][4]

Obesity represents one of the most pressing public health challenges of the 21st century, affecting over 600 million adults globally.[3][4] Contemporary understanding recognizes adipose tissue as a metabolically active endocrine organ capable of secreting numerous bioactive molecules. In obesity, adipose tissue undergoes pathological remodeling characterized by adipocyte hypertrophy, macrophage infiltration, and dysregulated secretion of inflammatory mediators, promoting chronic low-grade systemic inflammation.[5][3]

Over the past two decades, growing evidence has suggested a possible association between obesity and periodontal diseases.[1][2][3] Multiple epidemiological studies have reported higher prevalence and severity of periodontitis among individuals with elevated body mass index (BMI).[1][2][6] Proposed biological mechanisms include adiposity-associated hyperinflammation, altered immune responses, metabolic disturbances, and microbial dysbiosis.[3][5]

The aim of this review is to comprehensively summarize the current evidence on the relationship between obesity and periodontal diseases, with particular emphasis on biological mechanisms, epidemiological associations, modifying factors, effects on periodontal treatment outcomes, and the clinical relevance of this relationship in contemporary periodontal practice.

Materials and Methods

Review Design

This narrative review was conducted to summarize and critically evaluate current evidence on the relationship between obesity and periodontal diseases.

Literature Search Strategy

A comprehensive literature search was performed in March 2026 using PubMed/MEDLINE, Scopus, and Web of Science. Search terms included: "obesity," "overweight," "body mass index," "periodontitis," "periodontal disease," "adipokines," "inflammation," and "microbiome". These terms were combined using Boolean operators (AND, OR) in order to identify studies relevant to the subject of the review.

Inclusion and Exclusion Criteria

Inclusion criteria:

- Systematic reviews and meta-analyses investigating obesity-periodontitis associations

- Prospective cohort and cross-sectional studies

- Experimental studies on biological mechanisms

- Clinical trials evaluating treatment outcomes

- English language publications

Exclusion criteria:

- Case reports and expert opinion articles

- Studies lacking clear definitions of obesity or periodontal disease

- Publications with significant methodological limitations

Time Frame

Priority was given to publications from 2021-2026 in order to reflect the most recent evidence available on this topic. Earlier publications were also included when they provided important background information or foundational evidence relevant to the interpretation of current findings.

Results of the Literature Review

Obesity as a Systemic Disease

Definition and Classification

Obesity is defined as abnormal or excessive accumulation of adipose tissue that adversely affects health.[3][4] Body mass index (BMI), calculated as weight (kg) divided by height squared (m^2), serves as the primary screening tool. According to World Health Organization criteria, normal body weight is defined as a BMI of 18.5–24.9 kg/m^2, overweight as 25.0–29.9 kg/m^2, and obesity as ≥30 kg/m^2.[3][4]

BMI limitations include inability to distinguish fat from lean mass and variable correlation with cardiometabolic risk across populations.[6] Complementary measures include waist circumference, which better reflects visceral adiposity and metabolic risk.[6]

Adipose Tissue as an Endocrine Organ

Adipose tissue is recognized as an active endocrine organ secreting adipokines including leptin, adiponectin, and resistin, as well as pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6).[5][4] In obesity, pathological adipose tissue remodeling leads to dysregulated adipokine secretion and chronic low-grade systemic inflammation.[5][3] This inflammatory state contributes to insulin resistance, endothelial dysfunction, and metabolic disturbances underlying obesity-related comorbidities.[5][4]

Periodontal Diseases: Pathogenesis and Risk Factors

Definition and Pathogenesis

Periodontal diseases encompass inflammatory conditions affecting tooth-supporting tissues.[3][4] Gingivitis is characterized by reversible inflammation confined to the gingival tissues, while periodontitis involves irreversible destruction of periodontal attachment, including connective tissue loss and alveolar bone resorption.[3][4]

Pathogenesis involves complex interactions between microbial biofilm and host immune-inflammatory responses.[3] Key periodontal pathogens implicated in disease progression include Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola.[3] However, disease expression depends not only on microbial challenge but also on host susceptibility, with dysregulated inflammatory responses playing a central role in periodontal tissue destruction.[3]

Risk Factors

Established risk factors include tobacco smoking, diabetes mellitus, advanced age, genetic predisposition and socioeconomic status.[3][4] In recent years, obesity has been proposed as a potential modifiable risk factor, with growing evidence suggesting metabolic and inflammatory mechanisms may link these conditions.[1][2][3]

Biological Mechanisms Linking Obesity and Periodontitis

Multiple interconnected biological mechanisms have been proposed to explain associations between obesity and periodontal diseases.[3][5]

Chronic Low-Grade Systemic Inflammation

The most extensively investigated mechanism involves chronic low-grade inflammation characteristic of obesity.[5][3][4] Dysfunctional adipose tissue secretes elevated levels of pro-inflammatory cytokines including TNF-α, IL-6, and C-reactive protein.[5][4] These mediators may amplify inflammatory responses in periodontal tissues.[5][3]

TNF-α and IL-6 have been specifically implicated as interorgan inflammatory mediators linking obesity and periodontitis.[4] Both cytokines promote periodontal tissue destruction through enhanced osteoclast differentiation, increased matrix metalloproteinase production, and modulated immune cell function.[5][4]

Dysregulated Adipokine Secretion

Adipokines are key regulators of metabolic homeostasis and immune function.[5][4] Leptin, elevated in obesity, possesses pro-inflammatory properties and may promote inflammatory responses and bone resorption in periodontal tissues.[5] Adiponectin, which exhibits anti-inflammatory properties, is paradoxically decreased in obesity, potentially increasing periodontal disease susceptibility.[5][4] Resistin, elevated in obesity, promotes insulin resistance and inflammatory responses.[5]

Enhanced Osteoclast Activity

Recent research has identified novel preosteoclast populations expanded in obesity that contribute to enhanced bone destruction.[7] Monocytic myeloid-derived suppressor cells (M-MDSCs) are expanded in obesity and demonstrate enhanced osteoclastogenic potential.[7] In experimental periodontitis, obese mice showed greater M-MDSC expansion and increased osteoclast formation, associated with more severe alveolar bone loss.[7]

Microbiome Alterations

Emerging evidence suggests that obesity may influence both gut and oral microbiome composition.[8] Research using fecal microbiota transplantation demonstrated that gut dysbiosis from obese mice worsened alveolar bone destruction through elevated uric acid production.[8] Obesity-associated gut microbiota showed increased purine degradation pathway activity, leading to elevated serum uric acid that promoted periodontal bone destruction.[8]

Studies have also reported altered oral microbiome composition in obese individuals, with increased abundance of periodontopathogenic species even in periodontally healthy individuals.[3]

Metabolic Disturbances

Obesity-related metabolic disturbances including insulin resistance, oxidative stress, and dyslipidemia may affect periodontal health.[5][9] Insulin resistance promotes inflammatory responses and impairs wound healing, while oxidative stress contributes to tissue damage.[5][9]

Epidemiological Evidence

Cross-Sectional Studies

Numerous cross-sectional studies have examined obesity-periodontitis associations across diverse populations, consistently reporting higher prevalence and severity of periodontitis among individuals with elevated BMI.[1][2][6][4]

A cross-sectional analysis of 11,256 NHANES participants (2009-2014) found significant positive association between weight-adjusted-waist index and moderate/severe periodontitis (OR = 1.08, 95% CI: 1.01-1.17).[6]

Systematic Reviews and Meta-Analyses

Multiple systematic reviews and meta-analyses have synthesized evidence on obesity-periodontitis associations.[1][2][10]

A 2025 systematic review including 19 studies with 41,107 patients found obesity significantly associated with increased periodontitis risk (OR = 1.31, 95% CI: 1.22-1.41).[1]

Similarly, an updated 2022 meta-analysis including 37 studies reported an overall odds ratio of 1.35 (95% CI: 1.05-1.75).[2] Subgroup analyses revealed the association was strongest in younger adults aged 18-34 years (OR = 2.21, 95% CI: 1.26-3.89) and in European populations (OR = 2.46, 95% CI: 1.11-5.46).[2]

A comprehensive 2018 meta-review examining 14 systematic reviews concluded that obese individuals are more likely to have periodontal diseases with greater severity than non-obese individuals, with cross-sectional evidence congruent with longitudinal studies.[10]

Longitudinal Studies

Longitudinal studies have provided further support for a temporal relationship between obesity and periodontal disease.[11][10] Several cohort studies have shown that baseline obesity or weight gain over time is associated with a higher incidence and greater progression of periodontitis, suggesting that obesity may function as a true risk factor rather than merely a correlated condition.[11][10]

Factors Modifying the Obesity-Periodontitis Relationship

Several factors may modify the association between obesity and periodontal diseases.[2][6][3][10]

Diabetes mellitus frequently coexists with obesity and represents an independent risk factor for periodontitis.[3][10] Some studies have found obesity-periodontitis associations attenuated in diabetic populations.[6]

Tobacco smoking remains the most significant modifiable behavioral risk factor for periodontitis and may confound obesity-periodontitis associations.[3][10]

Age influences both obesity prevalence and periodontitis risk. Obesity-periodontitis associations appear stronger in younger adults than older populations.[2]

Socioeconomic status is associated with higher obesity prevalence, reduced dental care access, and increased periodontitis risk, potentially confounding observed associations.[3][10]

Impact of Obesity on Periodontal Treatment Outcomes

The effect of obesity on the outcomes of periodontal therapy remains uncertain, as available studies have produced inconsistent results.[10][12][13][14]

Studies Suggesting Impaired Responses

Several investigations have suggested that obesity may adversely affect the response to non-surgical periodontal treatment.[13][14] A 2014 study involving 260 adults reported that higher BMI and obesity were associated with worse mean probing pocket depth and percentage of sites >4mm at 2 months, independent of age, smoking, or plaque levels.[13]

A 2020 cohort study comparing 57 obese and 58 normal-weight non-smokers with periodontitis found that obesity was associated with worse clinical measures at 2 and 6 months after therapy, independent of confounders.[14]

Interpretation

Available evidence suggests that obesity may adversely influence the response to non-surgical periodontal therapy, although the magnitude and consistency of this effect remain uncertain.[10][12][13][14] Reported discrepancies across studies may be related to methodological heterogeneity, including differences in sample size, definitions of obesity, periodontal treatment protocols, follow-up duration, and adjustment for confounding factors.[10][12] Additional high-quality prospective and interventional research is needed to better define the impact of obesity on periodontal therapy outcomes.

3.7. Clinical Significance and Implications

The accumulating evidence linking obesity and periodontitis has important implications for clinical practice.[3][4][10]

Risk Assessment

Obesity should be considered as a potential modifiable risk factor during periodontal evaluation.[3][10] Routine assessment of BMI and waist circumference can be incorporated into dental practice.[2][3][15] Individuals with obesity may benefit from intensified preventive and supportive periodontal care.[3][10]

Patient Education

Dental professionals should educate patients about potential relationships between obesity and periodontal health.[3][4][10] Discussions should emphasize that maintaining healthy body weight may benefit oral health, delivered sensitively to avoid stigmatization.[3]

Interdisciplinary Collaboration

The relationship between obesity and periodontitis highlights the importance of collaboration between dental professionals and physicians, endocrinologists, and dietitians.[3][10] Oral healthcare professionals need to be aware of specific management considerations for obese patients, including modified practice facilities and tailored supportive therapy.[3][16]

Limitations of Current Evidence

Despite the growing number of studies on this topic, several important limitations remain.[1][2][3][10]

Study design limitations: Most studies are cross-sectional, precluding determination of causality.[1][2][10][17] Due to lack of longitudinal interventional studies and randomized clinical trials, there is insufficient evidence to determine cause-effect relationships.[3]

Heterogeneity in assessment: Studies have employed various obesity indicators and periodontitis definitions, complicating comparisons.[1][2][10]

Confounding factors: Obesity frequently coexists with diabetes, smoking, poor diet, and lower socioeconomic status, making it challenging to isolate obesity's independent effects.[3][10]

Limited mechanistic studies: While multiple mechanisms have been proposed, much evidence derives from animal models, with limited human studies directly demonstrating causation.[3][8][7]

Future Research Directions

Addressing knowledge gaps requires well-designed studies focusing on several areas.[1][3][10]

Prospective cohort studies with standardized definitions and measurements are needed to establish temporal relationships.[1][3][10]

Interventional studies examining effects of weight loss on periodontal outcomes would provide critical evidence regarding causality.[3][10]

Mechanistic studies should elucidate biological pathways in humans, including the roles of adipokines, inflammation, and microbiome alterations.[3][5][8]

Standardization of methods would improve comparability across studies.[1][2][10]

Discussion

This review summarizes current evidence on the obesity-periodontitis relationship, revealing consistent epidemiological associations supported by plausible biological mechanisms, while highlighting important limitations.

Strength of Epidemiological Evidence

Epidemiological evidence demonstrates a positive association between obesity and periodontitis across diverse populations.[1][2][4][10][18] Meta-analyses consistently report elevated odds ratios ranging from 1.31 to 2.46.[1][2] The consistency across study designs strengthens confidence in a genuine association.

However, the magnitude is modest compared to established risk factors such as smoking and diabetes.[3][10] Substantial heterogeneity exists across studies, and the predominance of cross-sectional designs limits causal inference.[1][2][10]

Biological Plausibility

The association between obesity and periodontitis is supported by several biologically plausible mechanisms.[5][3][8][4][7][19] The chronic low-grade systemic inflammation characteristic of obesity provides a compelling framework for understanding how obesity might amplify periodontal destruction.[5][3][4]

Recent advances include the discovery that obesity-associated gut microbiome dysbiosis worsens periodontal bone destruction through elevated uric acid and identification of expanded preosteoclast populations in obesity.[8][7] These findings advance mechanistic understanding but require further validation in humans.

Clinical Relevance

The clinical significance of the obesity-periodontitis relationship depends largely on whether obesity influences periodontal treatment outcomes and whether weight management may improve periodontal health.[10][12][13][14] Evidence regarding treatment responses remains inconsistent, with some studies suggesting attenuated responses while others report comparable improvements.[13][14][12]

The critical question of whether weight loss interventions improve periodontal outcomes remains unanswered.[3][10] Well-designed interventional studies are needed to establish whether weight management should be recommended as part of comprehensive periodontal care.

Integration into Clinical Practice

Contemporary periodontology emphasizes comprehensive risk assessment incorporating systemic factors.[3][10][20] Evidence supports considering obesity as a potential modifiable risk factor, though obesity should be viewed as one component of overall metabolic status rather than in isolation.[3][10]

Challenges include the complexity of obesity management, need for interdisciplinary collaboration, and incomplete evidence base for specific clinical recommendations.[3][10]

Conclusions

This review yields several key conclusions:

1. Consistent epidemiological associations exist between obesity and periodontitis, with meta-analyses demonstrating odds ratios of 1.31-2.46. The association is stronger in younger adults and varies by geographic region.

2. Multiple plausible biological mechanisms link these conditions, including chronic inflammation, dysregulated adipokine secretion, enhanced osteoclast activity, and microbiome alterations.

3. Evidence regarding obesity's impact on treatment outcomes remains inconsistent, with some studies suggesting attenuated responses while others report comparable improvements.

4. Important methodological limitations characterize current evidence, including predominance of cross-sectional studies, heterogeneous assessment methods, and potential confounding.

5. Clinical implications support integrating metabolic risk assessment into periodontal evaluation. Obesity should be considered as a potential modifiable risk factor, with emphasis on interdisciplinary collaboration.

6. Future research priorities include prospective cohort studies, randomized controlled trials of weight loss interventions, and mechanistic studies in humans.

In summary, obesity appears to be associated with an increased risk of periodontitis through complex inflammatory and metabolic pathways. Although a causal relationship has not yet been definitively established, the current evidence supports a more holistic approach to periodontal care that takes the patient’s metabolic health into account. Further high-quality research is needed to support the development of evidence-based clinical recommendations.

Disclosure

AI Transparency Statement

Artificial intelligence tools were used as assistive resources during manuscript preparation for literature organization and language refinement. All scientific interpretation, critical analysis, and conclusions remain entirely the responsibility of the human authors.

Supplementary Materials

No supplementary materials are associated with this manuscript.

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