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Beyond body weight: the systemic effects of obesity on periodontal health

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Abstract

Background: Obesity promotes "metaflammation" – a chronic, low-grade systemic inflammatory state closely tied to periodontitis. This pathological synergy accelerates the destruction of tooth-supporting tissues through shared immuno-metabolic pathways.

Aim: The aim of this study is to synthesize current scientific evidence regarding the systemic effects of obesity on periodontal health, focusing on the molecular, microbiological, and clinical mechanisms that link these two chronic conditions.

Materials and Methods: A comprehensive review of databases, including PubMed, Scopus, and Quality in Sport, was conducted to identify systematic reviews and clinical trials. The investigation concentrated on adipokine profiles, clinical parameters (CAL, PD, BOP), and the influence of metabolic interventions on periodontal outcomes.

Results:

Obesity accelerates alveolar bone loss by disrupting leptin/adiponectin and the RANKL/OPG axis. Systemic oxidative stress (elevated ROS, MDA) and endotoxemia (LPS/TLR-4) worsen local inflammation, which is sustained by HDAC1-mediated epigenetic memory. Clinically, obesity increases periodontitis risk by 31–35% (OR 1.31–1.35) and degrades pediatric oral health (DMFT, OHRQoL). Visceral adiposity impairs responses to non-surgical periodontal therapy (SRP), but weight loss restores healing and clinical attachment gain.

Conclusions:

- Adipose tissue dysfunction drives periodontal breakdown by reprogramming host immune and epigenetic landscapes.
- Mechanical debridement (SRP) often fails alone under unmanaged metabolic stress.
- Long-term stability requires interdisciplinary care combining oral therapy with metabolic stabilization and weight management.

Key words: Obesity, Periodontitis, Periodontal Health, Adipokines, Inflammation Mediators, Epigenomics

1. Introduction

Modern literature defines obesity as a chronic, relapsing metabolic disease characterized by the pathological or excessive accumulation of adipose tissue, which negatively impacts overall health (11, 18). In clinical practice and screening studies, the Body Mass Index ($\text{BMI} \geq 30 \text{ kg/m}^2$) remains the primary diagnostic tool, although increasing importance is being attributed to parameters assessing adipose tissue distribution, such as waist circumference (WC) and waist-to-hip ratio (WHR), which strictly correlate with visceral fat content (14, 18). A key aspect of the pathophysiology of obesity is the recognition of adipose tissue as an active endocrine organ whose dysfunction impairs the secretion of adipokines and pro-inflammatory mediators (3, 20). This process induces a low-grade, chronic, systemic inflammation, termed "metaflammation," which underlies numerous metabolic and cardiovascular complications (12, 13).

Periodontitis is characterized as a multifactorial, chronic immuno-inflammatory disease initiated by a dysbiotic bacterial biofilm accumulating adjacent to the teeth (19, 20). It leads to progressive, irreversible destruction of the tissues that support the tooth, including the periodontal ligament and alveolar bone, ultimately resulting in tooth loss (11, 20). It is emphasized that it is not the bacterial load itself, but rather an excessive and aberrant host immune response to the microbial challenge that determines the severity and rate of destructive progression (12, 13). Currently, periodontitis is recognized as a disease of significant systemic importance, exhibiting bidirectional links with diabetes, cardiovascular diseases, and metabolic syndrome (2, 4).

Epidemiological analyses indicate that both obesity and periodontitis are among the most prevalent chronic diseases worldwide (11, 12). Numerous cross-sectional and cohort studies present a strong and consistent correlation between increased BMI and a higher risk of development and greater severity of periodontal destruction (5, 15). It has been proved that individuals with obesity have a 31–35% higher probability of developing periodontitis (OR 1.31–1.35), a relationship that exhibits a dose-response pattern (11, 15). This association is

especially evident in young adults, women, and non-smokers (11, 15). The coexistence of both conditions manifests clinically through greater clinical attachment loss (CAL), deeper periodontal pockets (PD), and higher bleeding on probing (BOP) scores, ultimately translating into a higher risk of edentulism (11, 20).

2. Research materials and methods

The research material for this study comprised high-quality peer-reviewed scientific papers indexed in major biomedical databases (PubMed, Scopus, Quality in Sport). The literature search was restricted to publications that evaluated the interrelationships among obesity, metabolic health, and periodontal status.

The inclusion criteria targeted:

- Systematic reviews, meta-analyses, and controlled clinical trials.
- Studies evaluating specific biochemical markers (adipokines, oxidative stress markers, collagen degradation products).
- Papers assessing clinical periodontal parameters (CAL, PD, BOP) in relation to anthropometric indices (BMI, WC).

Data extraction focused on synthesizing the molecular pathways (the RANKL/OPG axis, epigenetic regulation, and metabolic endotoxemia) and compiling clinical data regarding the efficacy of non-surgical periodontal therapy (SRP) combined with weight management interventions.

Articles include randomized controlled trials, meta-analyses, and systematic reviews from 2010 to the present year.

3. Research results

3.1 Pathophysiological Mechanisms Associating Obesity with Periodontal Destruction

The key link between obesity and periodontitis is the phenomenon of "metaflammation"—a low-grade, chronic inflammatory state generated by dysfunctional adipose tissue (12, 13). In the course of obesity, adipocyte hypertrophy and hyperplasia occur, initiating tissue hypoxia and the infiltration of immune cells, predominantly macrophages (12, 18). Altered adipose

tissue ceases to function merely as an energy reservoir, becoming an active endocrine organ that secretes a wide spectrum of pro-inflammatory mediators, including cytokines (TNF- α , IL-6) and specific proteins known as adipokines (3, 13).

The primary biochemical mechanism underlying periodontal tissue destruction is the dysregulation of adipokine secretion. Leptin, the concentration of which is pathologically elevated in obesity, exerts strong pro-inflammatory effects by activating the JAK-2/STAT3 signaling pathways (3). In periodontal cells, leptin stimulates the secretion of TNF- α and IL-6, directly aggravating the local inflammatory response (3, 20). Furthermore, high concentrations of leptin have been shown to impair the healing capacity of periodontal ligament (PDL) cells by reducing their proliferation and migration (3). Conversely, adiponectin, which exerts anti-inflammatory functions and sensitizes tissues to insulin, is paradoxically decreased in obesity (12, 13). Its deficiency in periodontal tissues results in the loss of inhibition of NF- κ B nuclear translocation, leading to the overproduction of matrix metalloproteinases (MMP-1, MMP-3) and pro-inflammatory cytokines (3).

Resistin and visfatin also have important functions in the pathogenesis of destructive changes. Resistin, primarily secreted by macrophages infiltrating adipose tissue, induces a disruption of the RANKL/OPG axis balance in periodontal cells, directly promoting osteoclastogenesis and alveolar bone resorption (3, 20). Visfatin, on the other hand, exacerbates local secretion of IL-1 β and TNF- α while downregulating the expression of growth factors important for tissue repair, such as VEGF and RUNX2 (3).

Another prominent mechanism entails amplifying oxidative stress. Excess adipose tissue induces an increased production of reactive oxygen species (ROS) accompanied by a reduction in the body's total antioxidant capacity (12, 20). Within the periodontium, ROS cause direct oxidative damage to fibroblasts, epithelial cells, and extracellular matrix components (12). The clinical manifestation of this process is reflected in elevated levels of malondialdehyde (MDA) and 8-hydroxydeoxyguanosine in the gingival crevicular fluid of obese patients, which are strongly correlated with the severity of clinical attachment loss (20).

These pathways are additionally compounded by insulin resistance, which frequently co-occurs with obesity. Chronic hyperinsulinemia impairs neutrophil functions, including their chemotaxis and phagocytic capacity, thereby weakening the periodontal defense barrier against bacterial biofilm (12, 20). Additionally, insulin resistance promotes the formation of advanced glycation end-products (AGEs). Through interaction with their specific receptors (RAGE),

AGEs upregulate the synthesis of pro-inflammatory mediators and disrupt collagen homeostasis, drastically compromising the integrity of the tooth's supporting apparatus (12, 15).

3.2 The Impact of Obesity on Bone Metabolism and Collagen Homeostasis

The impact of obesity on the destruction of periodontal bony structures is driven by a disruption of the physiological equilibrium between bone formation and resorption processes (20). A key element of this mechanism is the disturbance of the RANKL/OPG axis. Under conditions of excess body mass, adipocytes and macrophages infiltrating adipose tissue secrete increased amounts of receptor activator of nuclear factor kappa-B ligand (RANKL). RANKL binds to the RANK receptor on the surface of osteoclast precursors, stimulating their differentiation and resorptive activity (20). Concurrently, a decrease in osteoprotegerin (OPG) levels is observed. Since OPG acts as a "decoy" receptor for RANKL, its downregulation abolishes the inhibition of osteoclastogenesis, leading to uncontrolled alveolar bone loss (3, 20). Saturated fatty acids, particularly palmitic acid, have been shown to independently aggravate this process by increasing the RANKL/OPG ratio in periodontal ligament (PDL) fibroblasts (20).

The destruction of the tooth's supporting apparatus in obesity is also associated with accelerated collagen breakdown, with hydroxyproline (Hyp) serving as its primary biochemical marker (6). Significantly elevated concentrations of Hyp have been observed in both saliva and plasma from obese patients, indicating accelerated metabolic turnover and extracellular matrix degradation (6). This process is catalyzed by matrix metalloproteinases, specifically MMP-8 (neutrophil collagenase), whose activity is synergistically stimulated by inflammatory mediators from adipose tissue and bacterial biofilms (6, 20). Additionally, obesity promotes the expansion of adipose tissue within the bone marrow, shifting the differentiation of mesenchymal stem cells (BMSCs) toward adipocytes at the expense of osteoblasts, thereby weakening the regenerative potential of bone (13, 20).

3.3 Microbiological and Epigenetic Aspects of the Obesity-Periodontium Relationship

The relationship between obesity and periodontitis is modulated by metabolic endotoxemia, which results from alterations in the composition of the intestinal and oral microbiomes (12). In obesity, increased intestinal barrier permeability enables the translocation of bacterial lipopolysaccharides (LPS) into the systemic circulation (12, 13). Circulating LPS binds to Toll-

like receptor 4 (TLR4) on periodontal immune cells, markedly exacerbating the local inflammatory response and promoting the proliferation of periodontopathogens (12). Microbiological analyses indicate that individuals with obesity show a higher abundance of specific bacterial species, such as *Tannerella forsythia* and *Porphyromonas gingivalis*, even within clinically healthy periodontal tissues (13, 20).

These mechanisms are further sustained by epigenetic modifications, which regulate gene expression without changing the underlying DNA sequence (9). Chronic inflammation induced by obesity has been observed to cause aberrant DNA methylation in the promoter regions of genes pivotal to the immune response, such as TNF- α , IL-6, and IFNG (9). These alterations directly correlate with the severity of tissue destruction and may lead to the development of an inflammatory memory ("innate immune memory") in periodontal cells (1, 9). Histone modifications, including dysregulated histone deacetylase (HDAC) activity, additionally play a key role (9). Elevated HDAC1 expression in periodontal tissues of patients with periodontitis has been shown to promote the production of pro-inflammatory mediators and suppress genes involved in bone formation, such as RUNX2. This creates a biochemical foundation for the synergistic action of obesity and bacterial infection (3, 9).

3.4 Clinical Implications and Effectiveness of Periodontal Therapy

Anthropometric indices, such as BMI and waist circumference (WC), show a strong positive correlation with the severity of clinical parameters used to describe periodontal status (15). Patients with obesity show significantly higher values of clinical attachment loss (CAL) and greater pocket depth (PD) compared to normal-weight individuals (11, 15). It has also been observed that parameters evaluating visceral obesity, particularly waist circumference, may serve as more precise risk indicators for bleeding on probing (BOP) and CAL than BMI alone (13, 20). In pediatric and adolescent populations, obesity is associated with a dramatic, nearly 14-fold increase in the risk of developing periodontal pockets exceeding 4 mm in depth (5, 20).

The effectiveness of conventional non-surgical periodontal therapy (SRP—scaling and root planing) in obese patients remains an issue in ongoing debate, although evidence leaning toward an impaired therapeutic response prevails (12, 20). Clinical analyses indicate that patients with excess body mass achieve statistically lower pocket-depth reduction and less clinical attachment gain following curettage procedures than normal-weight controls (12, 13). This phenomenon is ascribed to systemic immune dysregulation, impaired wound healing, and

reduced microvascular perfusion within periodontal tissues (12, 20). Nevertheless, some studies suggest that with exact plaque control, these therapeutic discrepancies may be minimized or eliminated (14, 20).

Weight reduction is a vital factor that modulates periodontal treatment outcomes. Intentional weight loss achieved through dietary modification or bariatric intervention has been found to significantly improve the clinical response to SRP therapy (12, 20). Patients undergoing bariatric surgery exhibit a more profound improvement in periodontal parameters compared to obese individuals receiving periodontal treatment alone (20). This mechanism relies on the reduction of circulating pro-inflammatory cytokines (TNF- α , IL-6), thereby dampening systemic inflammation while promoting oral tissue regeneration (3, 12).

3.5 Special Populations and Dietary Components

Investigations targeting children and adolescents show a significant, though complex, coexistence of obesity and periodontal conditions in younger populations (7). It has been observed that adolescents with an increased body mass index (BMI) are more likely to be diagnosed with plaque-induced gingivitis and may develop more severe periodontal destruction at an earlier age compared to their normal-weight peers (7, 16). Specifically, children in higher BMI categories have been shown to exhibit worse oral health status in permanent teeth, as measured by the Decayed, Missing, and Filled Teeth (DMFT) index (7). Furthermore, the presence of both obesity and oral disease appears to impact Oral Health-Related Quality of Life (OHRQoL) (16). Although some findings are statistically non-significant, a higher prevalence of OHRQoL impact has been reported in overweight and obese adolescents, manifesting through functional limitations and psychological discomfort (16). These associations are frequently worsened by low socioeconomic status, which restricts access to healthy lifestyle choices and promotes the consumption of high-calorie foods (7, 16).

Dietary habits operate as critical modifiers of periodontal disease severity by modulating systemic and local inflammatory cascades (10). Frequent intake of refined carbohydrates and high-glycemic-load foods has been shown to increase gingival inflammation and bleeding, even in the absence of increased total energy intake (10). Conversely, a balanced intake of unprocessed complex carbohydrates, vegetable proteins, and omega-3 fatty acids exerts anti-inflammatory and protective effects on periodontal tissues (10). It has been established that the Western diet, characterized by a high omega-6-to-omega-3 fatty acid ratio (up to 15:1),

markedly increases pro-inflammatory cytokine levels, whereas consumption of fruits and vegetables rich in antioxidants and micronutrients supports periodontal healing and mitigates oxidative stress (10).

3.6 Salivary Biomarkers and Public Health Directives

Salivary cytokine profiles have been evaluated as potential non-invasive biomarkers to reflect the combined effects of obesity and periodontitis (17). In clinical settings, individuals categorized as having a medium or high risk of periodontitis—based on a diagnostic cumulative risk score (CRS)—show higher salivary concentrations of interleukin-1 receptor antagonist (IL-1Ra) and IL-8, alongside lower concentrations of IL-10 and tumor necrosis factor-alpha (TNF- α) (17). While obesity is linked to systemic low-grade inflammation, evidence suggests that salivary cytokine concentrations are more directly dependent on periodontal status than on BMI alone (17). Specifically, in the obese population and among smokers, elevated IL-8 and reduced IL-10 concentrations are detected in saliva, suggesting that local periodontal infection may override the systemic effects of obesity on the oral biochemical environment (17).

From a public health perspective, periodontitis is recognized as a major "social disease" (Volkskrankheit) due to its high prevalence—affecting 10–15% of the global population in its severe form—and its substantial socioeconomic burden (19). As a primary cause of tooth loss in adults, its mass management needs a strategic, systemic approach centered on the regular removal of dysbiotic biofilms and long-term supportive periodontal therapy (19). The combination of staging and grading systems into clinical practice enables more customized and effective therapy (19). Given that periodontal disease is now acknowledged as an immunoinflammatory condition with a mutual relationship with systemic health, mass prevention strategies must address common risk factors shared with other noncommunicable diseases, such as unhealthy diets and metabolic imbalance (10, 19).

3.7 Synthesis of Molecular and Clinical Patterns in Obesity-Related Periodontitis

The synthesized data disclosed distinct molecular and clinical patterns linking obesity to periodontal breakdown.

At the molecular level, obesity induces adipocyte hypertrophy, tissue hypoxia, and macrophage infiltration, accelerating the secretion of pro-inflammatory cytokines (TNF- α , IL-6) and

specific adipokines (3, 13). Leptin, which is pathologically elevated, activates the JAK-2/STAT3 pathway, stimulating further cytokine release and impairing the healing ability of periodontal ligament (PDL) cells (3, 20). Conversely, anti-inflammatory adiponectin is paradoxically decreased, leading to uninhibited NF- κ B nuclear translocation and upregulation of matrix metalloproteinases (MMP-1, MMP-3) (3, 12). Resistin and visfatin independently disrupt the RANKL/OPG axis, shifting mesenchymal stem cell differentiation toward adipocytes, boosting osteoclastogenesis, and decreasing bone-forming markers like RUNX2 (3, 20). Furthermore, elevated production of reactive oxygen species (ROS) causes direct damage to fibroblasts, as evidenced by increased levels of malondialdehyde (MDA) and 8-hydroxydeoxyguanosine in the gingival crevicular fluid (12, 20).

At the structural and microbiological levels, accelerated collagen breakdown was evidenced by significantly elevated salivary and plasma hydroxyproline (Hyp) levels, driven by MMP-8 activity (6). Microbiological shifts include increased intestinal and oral mucosal permeability, leading to metabolic endotoxemia via bacterial lipopolysaccharide (LPS) translocation and TLR-4 receptor activation (12, 13). This dysbiosis promotes an increased abundance of periopathogens such as *Tannerella forsythia* and *Porphyromonas gingivalis* (13, 20). Epigenetic alterations were verified through aberrant DNA methylation of immune-response genes (TNF- α , IL-6, IFNG) and upregulation of histone deacetylase 1 (HDAC1), creating a persistent "inflammatory memory" in periodontal cells (1, 9).

Clinically, visceral adiposity parameters (WC) showed a stronger correlation with elevated BOP and CAL scores than BMI alone (13, 20). Among pediatric groups, obesity correlated with a nearly 14-fold increase in the risk of presenting pocket depths greater than 4 mm (5, 20). Therapeutic data indicated an impaired response to non-surgical periodontal treatment (SRP) in obese individuals, showing lower PD reduction and less CAL gain compared to normal-weight controls (12, 13). However, intentional weight loss through dietary restriction or bariatric surgery resulted in a significant reduction of circulating cytokines (TNF- α , IL-6), fundamentally improving clinical periodontal healing outcomes (3, 12, 20).

Table 1. *Comprehensive synthesis of the molecular, microbiological, and clinical patterns bridging obesity and periodontal destruction.*

Pathway / Category	Key Parameters, Mechanisms, and Findings
Molecular (Metaflammation)	Adipocyte hypertrophy and hypoxia (12, 18) activate HIF-1 α (20) and M1 macrophage migration (12). Leptin ($\uparrow$) inhibits periodontal ligament (PDL) regeneration via the JAK-2/STAT3 pathway (3, 20), while decreased adiponectin (13) triggers the NF- κ B pathway (3) and tissue-destructive matrix metalloproteinases: MMP-1, MMP-3 (3).
Structural & Bone	Accelerated collagen breakdown (6) is confirmed by elevated salivary hydroxyproline (Hyp) levels (6). Obesity disrupts the RANKL/OPG axis (20), which combined with oxidative stress—marked by increased MDA and 8-OHdG (12, 20)—leads to irreversible alveolar bone resorption (20).
Microbiological & Epigenetic	Metabolic endotoxemia: bacterial LPS (<i>P. gingivalis</i>) translocates into systemic circulation (12, 13), activating TLR-4 receptors (12, 20). Epigenetic changes, including HDAC1 overexpression (9) and aberrant immune gene methylation (1, 9), create an "innate immune memory" that impairs healing (9).
Clinical Epidemiology (Adults)	Obesity increases periodontitis risk by 31–35% (OR 1.31–1.35) (11, 15). Visceral obesity parameters (Waist Circumference - WC, WHR) (14, 18) correlate more strongly with attachment loss (CAL) and bleeding (BOP) than BMI alone (14, 20). Obesity attenuates the efficacy of SRP (12).
Special Populations (Children)	Obese children exhibit an almost 14-fold higher risk (OR 14.15) of developing periodontal pockets >4 mm (5, 20). The coexistence of obesity and dental caries (DMFT index) (7, 16) drastically impairs Oral Health-Related Quality of Life (OHRQoL) (16).
Modifiers & Therapy	A Western diet with an Omega-6:3 ratio of 15:1 (10, 11) fuels systemic inflammation. Weight reduction (diet, bariatric surgery) (8, 12) is essential to downregulate systemic cytokines (3) and significantly improve clinical periodontal therapy outcomes (12, 20).

4. Discussion

The findings in this study confirm that the relationship between obesity and periodontitis is not merely coincidental but is driven by shared, complex metabolic-inflammatory pathways. The transition of adipose tissue from an energy reservoir to an aggressive endocrine entity establishes "metaflammation" as a systemic driver of local tissue destruction.

A critical point of discussion is the impaired therapeutic outcome of SRP in obese individuals. The chronic hyperinsulinemia and insulin resistance associated with excess body mass impair essential neutrophil functions, such as chemotaxis and phagocytosis, thereby weakening the primary defense line against the oral biofilm (12, 20). This is exacerbated by advanced glycation end-products (AGEs) interacting with RAGE receptors, which permanently compromises the integrity of the tooth's supporting apparatus (12, 15).

The discovery of epigenetic modifications, such as hypermethylation and HDAC1 upregulation, constitutes a paradigm shift. It demonstrates that metabolic stress can hardwire a prolonged hyper-inflammatory state into periodontal tissues, explaining why standard local treatments might fail unless the underlying systemic metabolic disorder is managed (3, 9). The clinical superiority of monitoring waist circumference (WC) over BMI, as highlighted in the results, underscores that visceral fat, rather than total body mass, is the true driver of periodontal breakdown (13, 20). Encouragingly, data on bariatric interventions and dietary weight loss show that downregulating systemic inflammation directly enhances periodontal tissue regeneration, supporting the necessity of metabolic stabilization (3, 12).

5. Conclusions

1. Obesity acts as an independent, biologically active risk factor for periodontitis, utilizing shared pathways of metaflammation, oxidative stress, and epigenetic remodeling to accelerate alveolar bone and collagen destruction.
2. The presence of visceral obesity and insulin resistance significantly impairs the clinical efficacy of standard non-surgical periodontal therapy (SRP).
3. Successful, long-term management of periodontitis in patients with obesity cannot be achieved through local dental interventions alone; it requires systemic metabolic stabilization and intentional weight reduction.

4. Periodontal care must transition toward an interdisciplinary model involving close cooperation between dentists, clinical dietitians, and endocrinologists or diabetologists to co-manage the patient's metabolic and oral health.

Disclosure

Supplementary Materials

Not applicable.

Author Contributions

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

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The authors deny any conflict of interest.

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