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Periodontitis and Insulin Resistance Implications for Glycemic Control and Integrated Diabetes Care

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Abstract

Periodontitis and Diabetes Mellitus are two globally prevalent chronic conditions that share a bidirectional relationship, with mounting evidence implicating insulin resistance as a central mechanistic link. Periodontal inflammation promotes systemic release of pro-inflammatory cytokines, oxidative stress, advanced glycation end products, and microbial translocation, all of which impair insulin signaling and exacerbate hyperglycemia. Clinical studies consistently demonstrate higher glycated hemoglobin (HbA1c) levels in individuals with periodontitis, while periodontal therapy yields modest but clinically meaningful improvements in glycemic control. These interactions highlight periodontitis as a modifiable risk factor in diabetes management. The implications are particularly significant for low and middle income countries, where high burdens of both conditions coincide with limited access to oral healthcare. This perspective underscores the importance of integrating periodontal care into diabetes prevention and management frameworks, advocating for multidisciplinary collaboration among endocrinologists, dentists, and public health practitioners. Addressing current gaps including interindividual variability, limited longitudinal evidence, and healthcare system barriers requires robust research and policy support. Ultimately, incorporating oral health into chronic disease care models represents not only a clinical necessity but also a public health imperative to improve quality of life and reduce complications for individuals living with diabetes.

Keywords: Periodontitis; Insulin Resistance; Glycemic Control; Diabetes Management; Public Health Integration

Introduction

Periodontitis, a chronic inflammatory disease of the supporting tissues of the teeth, is also highly prevalent worldwide, affecting nearly 50% of adults and representing one of the most prevalent oral health challenges globally [1]. Diabetes mellitus (DM) is one of the most pressing global health challenges, with more than 500 million people currently affected and projections estimating over 780 million cases by 2045 [2]. The coexistence of these two conditions has been consistently reported, and growing evidence indicates a bidirectional relationship between them [3]. Individuals with diabetes have an increased risk of periodontitis, while those with periodontitis are more likely to develop diabetes and experience poor glycemic outcomes [4].

The biological mechanisms linking these two conditions are increasingly being clarified. Periodontal inflammation induces the release of pro-inflammatory cytokines such as tumor necrosis factor-α, interleukin-6, and C-reactive protein into the systemic circulation. These mediators impair insulin receptor signaling, reduce glucose transporter activity, and promote hepatic glucose output, all of which contribute

to insulin resistance [5]. Additionally, periodontal pathogens such as Porphyromonas gingivalis may translocate systemically, increasing endotoxemia and metabolic dysregulation [6]. Oxidative stress and the accumulation of advanced glycation end products further amplify these disturbances, creating a vicious cycle of inflammation and impaired glucose metabolism [7].

Clinical evidence demonstrates that individuals with periodontitis frequently exhibit higher glycated hemoglobin (HbA1c) levels compared with periodontally healthy individuals. Periodontal therapy has been associated with modest improvements in glycemic control, typically reflected as reductions in HbA1c values of 0.3–0.4% [8]. Although these reductions may seem small, they are clinically significant, as they correspond to decreased risk of diabetic complication. Despite these findings, substantial heterogeneity remains, and causality has not been definitively established.

The public health relevance of this interaction is particularly pronounced in low- and middle-income countries, where both diabetes and periodontitis are highly prevalent and access to oral healthcare is limited. Integrating oral health strategies into diabetes management may provide cost-effective avenues for improving outcomes in these populations [9]. While the coexistence of diabetes and periodontitis is recognized, the role of insulin resistance as a central mechanistic bridge has not been adequately highlighted. This Perspective aims to highlight how periodontal inflammation contributes to insulin resistance and influences diabetes management.

Pathophysiological Mechanisms Linking Periodontitis and Poor Glycemic Control

The biological mechanisms that underlie the relationship between periodontitis and diabetes are increasingly understood, with insulin resistance emerging as a key mediator. Periodontitis is characterized by chronic low-grade inflammation that extends beyond the oral cavity, influencing systemic metabolic processes [10].

Chronic inflammation plays a central role in linking periodontal disease with impaired glycemic control. Periodontal pockets harbor gram-negative anaerobic bacteria, including Porphyromonas gingivalis and Tannerella forsythia, which release lipopolysaccharides (LPS) into the circulation. LPS triggers Toll-like receptor signaling, promoting systemic release of tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP) [11]. Elevated TNF-α interferes with insulin receptor phosphorylation in adipocytes and muscle cells, reducing downstream insulin signaling. IL-6 further exacerbates hepatic glucose production, while CRP levels reflect the magnitude of systemic inflammation. Collectively, these cytokines induce insulin resistance and impair glucose uptake [12].

Insulin receptor signaling disruption is another critical pathway. Under normal physiology, insulin binding activates receptor tyrosine kinases, leading to phosphorylation of insulin receptor substrate (IRS) proteins and subsequent activation of phosphoinositide 3-kinase (PI3K)/Akt signaling. Periodontal inflammation alters this cascade: TNF-α induces serine phosphorylation of IRS-1, impairing signal transduction and preventing translocation of glucose transporter-4 (GLUT4) to the plasma membrane. Consequently, glucose uptake in skeletal muscle and adipose tissue is reduced, leading to hyperglycemia. In addition, inflammatory mediators promote hepatic gluconeogenesis, further worsening glycemic control [13].

Oxidative stress and advanced glycation end products (AGEs) provide another link between the two conditions. Hyperglycemia accelerates the formation of AGEs, which interact with the receptor for AGEs (RAGE) on periodontal and immune cells, amplifying local and systemic inflammation. Conversely, periodontal inflammation increases systemic oxidative stress, generating reactive oxygen species (ROS) that impair insulin signaling and damage pancreatic β-cells. This creates a vicious cycle: diabetes exacerbates periodontal inflammation through AGE-RAGE pathways, and periodontitis accelerates systemic oxidative stress, promoting further insulin resistance [14].

Microbiome dysbiosis also contributes to impaired glycemic control. Periodontitis is associated with an overgrowth of pathogenic bacteria, particularly Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans, and Treponema denticola. P. gingivalis expresses virulence factors such as gingipains, which disrupt host immune responses and facilitate systemic dissemination of bacteria and endotoxins. Animal studies demonstrate that chronic exposure to P. gingivalis LPS induces systemic endotoxemia, adipose tissue inflammation, and insulin resistance. Furthermore, periodontitis-induced dysbiosis may influence gut microbiota, aggravating metabolic endotoxemia and glucose intolerance [15,16].

Taken together, these mechanisms establish periodontitis not only as a local oral condition but as a systemic inflammatory disease with profound metabolic consequences. The convergence of cytokine release, oxidative stress, AGE-RAGE signaling, and microbial translocation provides multiple biologically plausible pathways through which periodontal inflammation exacerbates insulin resistance and worsens glycemic control.

Clinical and Epidemiological Evidence

Recent studies have further elucidated the bidirectional relationship between periodontitis and diabetes, highlighting the impact of periodontal inflammation on glycemic control. The studies demonstrated that severe periodontitis adversely affects diabetes by impairing insulin actions due to systemic microinflammation.

Clinical investigations have shown that insulin resistance, as reflected by the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), plays a significant mediating role in the relationship between obesity and periodontitis [17]. These findings underscore the importance of early metabolic regulation in mitigating obesity related periodontal risk and provide mechanistic insight into the interplay between systemic and oral health.

Furthermore, several studies found a statistically significant association between periodontitis and insulin resistance in adult patients with diabetes [8]. This association highlights the need for integrated care approaches that address both periodontal health and metabolic control in diabetic patients.

Epidemiological data indicate that the prevalence and severity of periodontitis are increased and advanced in individuals with diabetes. Severe periodontitis elicits adverse effects on diabetes by impairing insulin actions due to systemic microinflammation. These observations suggest that periodontal disease may contribute to the progression of diabetes and vice versa, emphasizing the necessity for comprehensive management strategies that encompass both conditions.

Implications for Insulin Resistance and Diabetes Management

Periodontitis represents a modifiable risk factor in diabetes care. Chronic periodontal inflammation contributes to systemic insulin resistance, impairing glycemic control and increasing the likelihood of diabetes-related complications [18]. Effective management of periodontitis, therefore, has the potential to improve metabolic outcomes and reduce overall disease burden. Non-surgical periodontal therapy, including scaling and root planing, adjunctive antimicrobial therapy, and oral hygiene optimization, has been associated with reductions in glycated hemoglobin (HbA1c) by approximately 0.3–0.5% [19,20]. These improvements, although modest, are clinically meaningful, as even small decreases in HbA1c correspond to reduced risk of microvascular and macrovascular complications.

Integrating dental care into routine diabetes management offers a multidisciplinary approach to optimize patient outcomes. Collaboration between endocrinologists, dentists, and public health practitioners ensures early identification of periodontal inflammation and timely intervention, mitigating the systemic impact of oral disease on insulin signaling. Screening for periodontitis during diabetes consultations enables risk stratification and targeted preventive strategies, particularly in populations with limited access to oral healthcare [21].

Inflammation-driven insulin resistance underlines the importance of holistic management strategies. Interventions aimed at reducing systemic inflammatory burden, such as optimization of glycemic control, weight management, and lifestyle modifications, complement periodontal therapy in improving insulin sensitivity. Nutritional counseling, physical activity, and smoking cessation reinforce both metabolic and oral health outcomes [22].

From a pharmacological perspective, the use of anti-inflammatory agents, such as sub-antimicrobial dose doxycycline, has demonstrated potential in reducing both periodontal inflammation and systemic markers of insulin resistance. These adjunctive therapies can enhance the metabolic benefits of standard periodontal interventions [23].

Public health implications are equally significant. In populations with high prevalence of diabetes and periodontitis, routine periodontal evaluation and treatment can be cost-effective, reducing healthcare expenditures associated with diabetes complications. Training healthcare providers to recognize the interrelationship between oral health and metabolic control supports implementation of integrated care models, particularly in low- and middle-income countries where oral healthcare access is limited [22].

Recognizing periodontitis as a modifiable risk factor reinforces the need for interdisciplinary approaches to diabetes management. Coordinated strategies that combine medical, dental, and behavioral interventions provide opportunities to improve insulin sensitivity, optimize glycemic control, and reduce long-term complications. Implementation of such models necessitates policy support, education, and allocation of resources to bridge gaps in oral health access and metabolic care.

Public Health and Policy Considerations

Diabetes and periodontitis are both highly prevalent conditions in low- and middle-income countries (LMICs), where access to oral healthcare is often limited [24]. The intersection of these diseases amplifies the public health burden, as untreated periodontitis can exacerbate insulin resistance, leading to poor glycemic control and increased risk of diabetes-related complications. Integrating oral health into existing diabetes care programs has the potential to reduce morbidity and healthcare costs while improving overall patient outcomes [25].

Inequities in oral healthcare access contribute to disparities in both periodontal and metabolic health. Populations in rural areas and socioeconomically disadvantaged groups are disproportionately affected, with higher prevalence of untreated periodontitis and poorer glycemic control. Health policies that prioritize routine periodontal screening, preventive care, and timely treatment within primary healthcare frameworks are essential for addressing these disparities [26].

Cost-effectiveness analyses indicate that incorporating periodontal care into diabetes management can reduce the long-term economic burden associated with diabetes complications. Preventive periodontal interventions decrease the need for complex medical treatments by mitigating systemic inflammation and improving metabolic control [27,28]. Training healthcare providers, including physicians, nurses, and community health workers, to recognize oral-systemic interactions supports early identification and intervention.

Policy-level integration requires development of guidelines that encourage multidisciplinary collaboration between medical and dental professionals. Implementation strategies may include reimbursement models that cover periodontal assessment and therapy for patients with diabetes, inclusion of oral health indicators in national diabetes registries, and public health campaigns to raise awareness of the oral-systemic connection [29,30].

Community-based programs play a critical role in bridging gaps in access. Mobile dental clinics, school-based oral health programs, and public-private partnerships can provide preventive and therapeutic services to underserved populations, enhancing both oral and metabolic health outcomes. Additionally, culturally tailored education and behavioral interventions support adherence to oral hygiene practices and diabetes self-management [22].

Addressing the combined burden of diabetes and periodontitis requires a systems-level approach that aligns healthcare delivery, policy, and education. Recognition of periodontitis as a modifiable risk factor for poor glycemic control emphasizes the importance of integrated public health strategies. Strengthening health systems to provide comprehensive care can reduce disease progression, improve quality of life, and alleviate long-term economic strain on both patients and healthcare infrastructures.

Challenges and Unanswered Questions

Despite increasing recognition of the interplay between periodontitis and diabetes, several challenges limit full clinical and public health integration. One critical issue is heterogeneity in patient response, some individuals with severe periodontitis maintain adequate glycemic control, while others experience significant metabolic deterioration. The factors underlying this variability, including genetic predisposition, immune response differences, and microbial composition, remain incompletely understood [31].

Causality between periodontitis and diabetes is difficult to establish. Observational evidence demonstrates association, but longitudinal studies are limited, and confounding factors such as diet, physical activity, and socioeconomic status complicate interpretation. There is also

insufficient mechanistic data clarifying how periodontal inflammation quantitatively influences insulin resistance pathways at the cellular and systemic levels.

Socioeconomic and cultural factors present additional barriers. In LMICs, limited access to dental care, lack of awareness regarding oral-systemic health connections, and financial constraints impede preventive and therapeutic interventions. Cultural beliefs and practices related to oral hygiene may also influence disease prevalence and response to treatment, necessitating tailored education and intervention strategies.

Implementation challenges in healthcare systems further constrain progress. Integrating dental services into primary diabetes care requires coordination among multiple healthcare professionals, policy support, and allocation of resources. Reimbursement structures often do not cover periodontal treatment for patients with diabetes, limiting adoption of comprehensive care models.

There is also a gap in long-term evidence regarding the durability of glycemic improvement following periodontal therapy. While reductions in HbA1c are documented in short-term studies, sustainability over years and the effect on the incidence of diabetic complications remain unclear. Evaluating outcomes across diverse populations and healthcare settings is essential to determine the generalizability of current findings [32,33]

Finally, research gaps exist regarding the microbiome and systemic immune modulation. Understanding how specific periodontal pathogens, microbial dysbiosis, and host inflammatory responses interact to influence insulin sensitivity could inform precision-targeted therapies. Further investigation into these mechanisms will advance the development of integrated interventions that simultaneously address oral and metabolic health.

Addressing these challenges requires multidisciplinary collaboration, longitudinal and mechanistic research, and policies that support equitable access to preventive and therapeutic oral healthcare. A comprehensive approach that accounts for biological, behavioral, and systemic factors is necessary to optimize diabetes management and reduce the global burden of both diseases.

Conclusion

Periodontitis is not merely a localized oral condition but a systemic inflammatory disease that significantly contributes to insulin resistance and poor glycemic control in individuals with diabetes. By impairing insulin signaling through chronic inflammation, oxidative stress, and microbial dysbiosis, periodontitis exacerbates metabolic dysfunction and increases the risk of diabetes-related complications. Recognizing periodontitis as a modifiable risk factor underscores the need for integrated diabetes care that brings together medical, dental, and public health disciplines. Incorporating routine periodontal assessment and treatment into diabetes management, particularly in resource-limited settings, offers a cost-effective strategy to improve metabolic outcomes, reduce complications, and advance global public health goals.

Transparency Statement

The lead author Baraka affirms that manuscripts is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted

Data Availability Statement

The authors confirm that data supporting the findings of this study are available within the article.

Authors Contributions

Baraka Kalinga conceived the study design, drafted the manuscripts, carried out the analysis and authored the original draft, Theresia S. Kundi carried out the analysis, authored the original draft and revised the manuscript. All authors read and approved the final manuscript.

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