Journal of Dental Sciences 20 (2025) 2021–2025

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.e-jds.com

Review article

Bidirectional association between obesity and chronic periodontitis: Inflammatory pathways and clinical implications

Tsung-Po Chen [a,b], Hui-Chieh Yu [c], Wen-Yuan Lin [a,b], Yu-Chao Chang [c,d*]

[a] Department of Family Medicine, China Medical University Hospital, Taichung, Taiwan
[b] School of Medicine, China Medical University, Taichung, Taiwan
[c] School of Dentistry, Chung Shan Medical University, Taichung, Taiwan
[d] Department of Dentistry, Chung Shan Medical University Hospital, Taichung, Taiwan

Received 2 July 2025; Final revision received 7 July 2025
Available online 16 July 2025

KEYWORDS
Obesity;
Chronic periodontitis;
Inflammation;
Oral microbiome;
Metabolic dysregulation

Abstract Obesity and chronic periodontitis are the prevalent chronic conditions significantly linked through shared common inflammatory and immune mechanisms. This review aimed to synthesize current knowledge on the association between obesity and chronic periodontitis, exploring biological mechanisms, epidemiological evidence, and clinical relevance. Obesity induces systemic low-grade inflammation marked by elevated pro-inflammatory cytokines, altered immune responses, and disrupted adipokine profiles that may influence periodontal inflammation. Conversely, chronic periodontitis, characterized by oral microbial dysbiosis and local inflammation, contributes to systemic inflammatory burden and potentially exacerbates obesity-related complications. Obesity-associated dietary habits further promote pathogenic oral microbiome shifts as well as aggravate periodontal inflammation. Clinically, obese patients frequently exhibit more severe chronic periodontitis and diminished responsiveness to conventional periodontal therapies, highlighting the need for tailored clinical interventions and integrated healthcare approaches. Public health efforts should make dental care easier to access, support teamwork between health professionals, and offer clear education to help manage both obesity and chronic periodontitis. Although researches have advanced significantly, the exact causal mechanisms still remain unclear. Further studies are needed to improve prevention as well as treatment strategies, ultimately reducing the global burden of obesity and chronic periodontitis.

* Corresponding author. School of Dentistry, Chung Shan Medical University, 110, Sec.1, Chien-Kuo N. Rd., Taichung, 40201, Taiwan.
E-mail address: cyc@csmu.edu.tw (Y.-C. Chang).

https://doi.org/10.1016/j.jds.2025.07.009

Introduction

Obesity and chronic periodontitis represent two most highly prevalent health challenges with significant implications for global public health. Obesity, characterized by excessive fat accumulation and energy imbalance, has affected over 650 million adults worldwide according to the World Health Organization.[1–3] Concurrently, chronic periodontitis is a spectrum of inflammatory disorders involving the supporting periodontal structures, affecting nearly half of adults over the age of 30.[4,5] Severe forms of chronic periodontitis impacts approximately 10—15 % of the population.[6] Current understanding indicates a bidirectional relationship between obesity and chronic periodontitis, where obesity exacerbates periodontal inflammation and vice versa.[7,8] Emerging evidence suggests that obesity may exacerbate chronic periodontitis, potentially through shared common inflammatory pathways or altered immune responses.[9,10] Furthermore, persistent periodontal inflammation may contribute to the progression of obesity through disruptions in metabolic hoomeostasis.[11]

This review aimed to synthesize current knowledge on the association between obesity and chronic periodontitis, exploring biological mechanisms, epidemiological evidence, and clinical relevance. By examining this relationship, we seek to highlight its implications for integrated health management and identify gaps warranting further investigation (see Fig. 1).

Overview of obesity

Obesity is clinically defined as an abnormal or excessive accumulation of body fat that impairs physical and metabolic function.[12] Obesity is also a state of chronic systemic inflammation.[12] It is now well recognized that adipose tissue functions as a metabolically active endocrine organ, secreting pro-inflammatory adipokines. Ectopic fat deposition can further contribute to both localized and systemic inflammation.[13] In obesity, enlarged adipocytes and increased infiltration of immune cells, especially macrophages, are commonly observed. These changes contribute to the secretion of tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP).[14] This inflammatory response is persistent and systemic, promoting insulin resistance, endothelial dysfunction, and

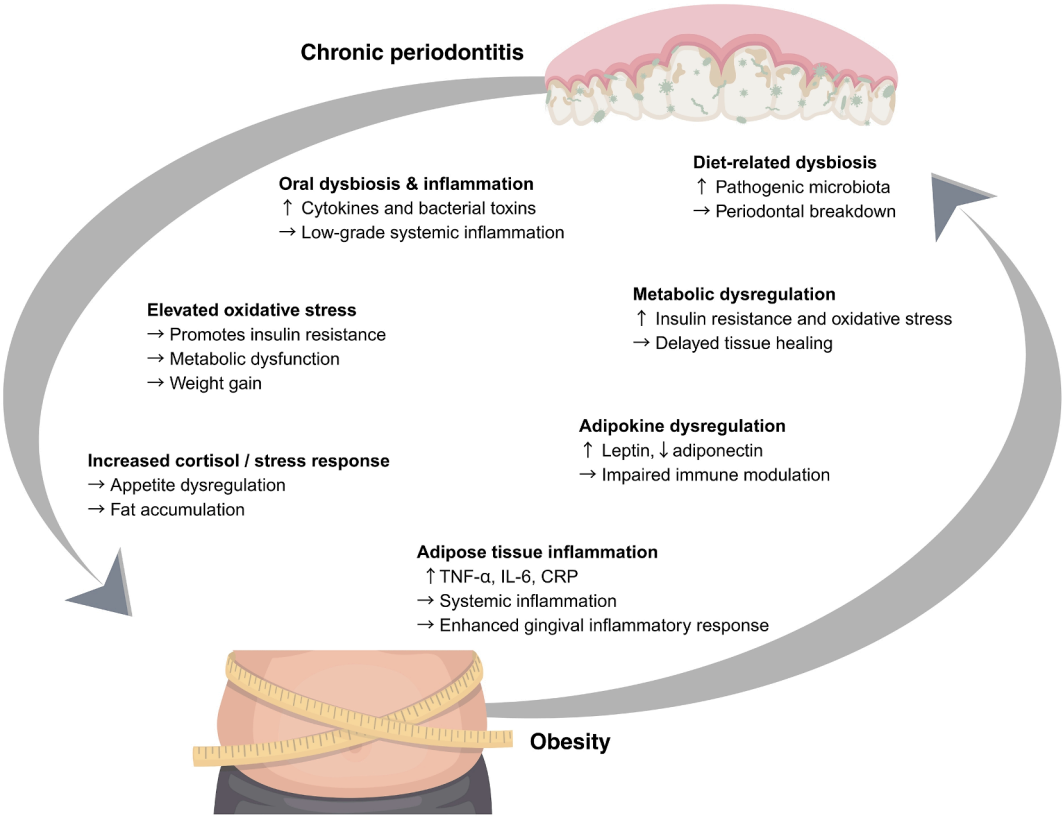

Figure 1 Proposed bidirectional mechanisms linking chronic periodontitis and obesity.

metabolic disturbances.[15] Central or abdominal obesity is especially associated with higher inflammatory activity compared to peripheral fat.[16] Chronic inflammation in obesity is believed to influence distant tissues, including the periodontium,[17] through circulating cytokines and immune dysregulation.[9,10] Recognizing obesity as an inflammatory condition helps explain its potential role in the pathogenesis of other chronic inflammatory diseases like chronic periodontitis.[9]

Overview of chronic periodontitis

Chronic periodontitis is a persistent inflammatory condition caused by an imbalance in the subgingival microbiota. This dysbiosis leads to the destruction of periodontal tissues, including gingiva, periodontal ligament, cementum, and alveolar bone.[18] The host's immune response plays a central role, with pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α contributing to periodontal tissue breakdown.[11,19] Though localized to the oral cavity, chronic periodontitis has many systemic implications. Bacterial components and inflammatory mediators can enter the bloodstream, promoting systemic inflammation. This systemic inflammation has been linked to various chronic diseases, including cardiovascular disease, diabetes, and obesity.[20–22] The recognition of chronic periodontitis as a contributor to systemic inflammation shifts its clinical relevance beyond oral health. Managing chronic periodontitis may reduce systemic inflammatory burden, particularly in individuals with comorbidities.[23]

Current understanding indicates a bidirectional relationship between obesity and chronic periodontitis, where obesity exacerbates periodontal inflammation and vice versa.[24] Shared common inflammatory mechanisms and altered oral microbiota may contribute to increase the prevalence and severity of chronic periodontitis in individuals with obesity.[8,25,26]

Potential mechanism between obesity and chronic periodontitis

The association between obesity and chronic periodontitis is likely mediated by a complex interplay of biological mechanisms, with chronic systemic inflammation acting as a central pathogenic link. Obesity induces a state of persistent, low-grade systemic inflammation, originating from dysfunctional adipose tissue. Excess visceral fat, particularly in abdominal regions, secretes pro-inflammatory cytokines, such as TNF-α, IL-6, and CRP, which circulate systemically, altering immune regulation and heightening inflammatory responses throughout the body. In chronic periodontitis, where inflammation is initially triggered by pathogenic bacteria present in dental biofilm, this systemic inflammatory environment may intensify local inflammatory responses within periodontal tissues. For example, IL-6 increases osteoclast activity, promoting alveolar bone loss which is a key feature of chronic periodontitis.[27,28] Similarly, TNF-α stimulates matrix metalloproteinases, enzymes that break down gum tissue, making periodontal damage worse.[27,29]

Obesity-related metabolic dysregulation offers additional pathways. Insulin resistance causes oxidative stress, making it harder for periodontal tissues to heal after bacterial damage.[9,22] Adipokines, hormones produced by adipose tissue, further modulate this relationship.[30] Fat cells release adipokines, which attract macrophages and increase inflammatory cytokine production. Because obesity impairs immune responses and raises inflammation markers in the blood, the increased production of inflammatory cytokines is one key way that chronic periodontitis may be connected to obesity.[25] Leptin, which increases with fat mass, has pro-inflammatory effects. It is found in high levels in inflamed periodontal tissues and may enhance immune responses to oral pathogens.[13,30] Conversely, adiponectin, an anti-inflammatory adipokine, is reduced in obesity, diminishing protective mechanisms and tilting the balance toward periodontal damage.[31] Hormonal imbalances, such as elevated cortisol levels from chronic stress—a common comorbidity in obesity—may also weaken periodontal defenses, increasing susceptibility to infection and inflammation.[32] Evidence also shows that chronic periodontitis can increase systemic oxidative stress. This can trigger ongoing inflammation that may lead to insulin resistance, which is common in obesity.[33] This insulin resistance can then disrupt glucose metabolism and appetite control, promoting weight gain.[34] The study showed that non-surgical periodontal therapy significantly lowered oxidative stress markers.[35] This suggests periodontal treatment may reduce oxidative damage and improve periodontal health in obese patients.

The oral microbiome provides yet another mechanistic bridge. Chronic periodontitis involves changes in the oral microbiome, with an increase in pathogens such as *Porphyromonas* gingivalis, *Aggregatibacter* actinomycetemcomitans, and *Tannerella* forsythia.[36–38] Obese individuals tend to have more frequent and severe periodontitis, partly due to differences in their oral microbial composition. Obesity, often linked to high-fat and high-sugar diets, can alter dental biofilm by increasing harmful bacteria like *Fusobacterium nucleatum* and *Aggregatibacter actinomycetemcomitans* over beneficial commensals.[8,39] This change in bacteria makes dental biofilm more harmful, increases periodontal inflammation, and adds to the inflammation already caused by obesity. Emerging evidence supports the roles of inflammation, metabolic changes, and shifts in bacteria. However, how these factors interact and which plays the most important role is still unclear. Further studies are needed to clarify these relationships and identify effective targets for intervention.

Clinical and public health relevance linking obesity and chronic periodontitis

The interplay between obesity and chronic periodontitis carries significant implications for clinical practice and public health policy. In dental settings, obese patients often present with more severe periodontal conditions and exhibit suboptimal responses to standard treatments. For instance, Suvan et al.[40] found that obese individuals receiving non-surgical periodontal therapy had less improvement in pocket depth and continued inflammation

compared to non-obese patients. This might be related to higher systemic inflammation or impaired immune responses in obese individuals. There is a need for individualized clinical approaches, such as longer treatment durations, additional anti-inflammatory therapies, or closer monitoring, to improve patient outcomes. Chronic periodontitis may also worsen obesity-related conditions, as its systemic inflammation can increase insulin resistance, impair blood vessel function, and elevate cardiovascular risk. This suggests a two-way relationship, highlighting the need for comprehensive management.[10,22]

From a public health perspective, the rising prevalence of obesity amplifies the urgency of addressing its oral health consequences. Lifestyle changes aimed at reducing obesity, such as healthier diets with less sugar and fat, regular exercise, and behavioral therapy, may also benefit periodontal health.[21] Integrated care is important. Dentists should regularly check obese patients for chronic periodontitis and include oral health education in their practice. Meanwhile, primary care doctors and endocrinologists should consider periodontal health when treating obesity. Public health campaigns could use this connection to highlight how maintaining a healthy weight and good oral hygiene can help reduce both obesity and chronic periodontitis.

However, there are still several limitations for public health aspect. Access to dental care remains limited in many regions. Low-income communities are especially affected, where obesity and chronic periodontitis often co-occur due to socioeconomic and healthcare challenges. Limited public awareness of this link hinders prevention efforts and highlights the need for targeted educational initiatives.[41] Overcoming these barriers requires investment in interdisciplinary training for healthcare providers, expanded dental insurance coverage, and community-based programs that address both systemic and oral health.

Obese individuals have a higher risk and severity of chronic periodontitis, making treatment more challenging and increasing overall health risks. In turn, periodontal inflammation may further worsen conditions related to obesity. This interplay demands tailored clinical strategies and integrated public health approaches that address both conditions concurrently. Clinicians should recognize obesity as a significant modifying factor for periodontal health, emphasizing personalized approaches to manage chronic periodontitis effectively. Future research should focus on long-term studies to clarify causal relationships, as well as clinical trials to determine if weight loss or periodontal treatments can reduce this link.

Conclusion

The link between obesity and chronic periodontitis is strongly supported by evidences from epidemiological, clinical, and biological studies, showing common pathways involving chronic inflammation, metabolic changes, and microbial alterations. Connecting oral and overall health could help develop new strategies to reduce obesity and chronic periodontitis, improving overall health in populations worldwide.

Declaration of competing interest

The authors have no conflicts of interest relevant to this article.

References

1. NCD Risk Factor Collaboration. Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: A pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents, and adults. *Lancet* 2017;390:2627—42.
2. Bluher M. Obesity: global epidemiology and pathogenesis. *Nat Rev Endocrinol* 2019;15:288—98.
3. Chooi YC, Ding C, Magkos F. The epidemiology of obesity. *Metabolism* 2019;92:6—10.
4. Eke PI, Dye BA, Wei L, et al. Prevalence of periodontitis in adults in the United States: 2009 and 2010. *J Dent Res* 2012;91:914—20.
5. Yu HC, Su NY, Huang JY, Lee SS, Chang YC. Trends in the prevalence of periodontitis in Taiwan from 1997 to 2013: A nationwide population-based retrospective study. *Medicine* 2017;96:8585.
6. Kassebaum NJ, Bernabe E, Dahiya M, Bhandari B, Murray CJ, Marcenes W. Global burden of severe periodontitis in 1990-2010: A systematic review and meta-regression. *J Dent Res* 2014;93:1045—53.
7. Suvan J, D'Aiuto F, Moles DR, Petrie A, Donos N. Association between overweight/obesity and periodontitis in adults. A systematic review. *Obes Rev* 2011;12:381—404.
8. Reytor-Gonzalez C, Parise-Vasco JM, Gonzalez N, et al. Obesity and periodontitis: A comprehensive review of their interconnected pathophysiology and clinical implications. *Front Nutr* 2024;11:1440216.
9. Suvan J, Harrington Z, Petrie A, et al. Obesity as predictive factor of periodontal therapy clinical outcomes: A cohort study. *J Clin Periodontol* 2020;47:594—601.
10. Jepsen S, Suvan J, Deschner J. The association of periodontal diseases with metabolic syndrome and obesity. *Periodontol 2000* 2020;83:125—53.
11. Hajishengallis G. Periodontitis: from microbial immune subversion to systemic inflammation. *Nat Rev Immunol* 2015;15:30—44.
12. Heymsfield SB, Wadden TA. Mechanisms, pathophysiology, and management of obesity. *N Engl J Med* 2017;376:254—66.
13. Lyon CJ, Law RE, Hsueh WA. Minireview: adiposity, inflammation, and atherogenesis. *Endocrinology* 2003;144:2195—200.
14. Saltiel AR, Olefsky JM. Inflammatory mechanisms linking obesity and metabolic disease. *J Clin Investig* 2017;127:1—4.
15. Lumeng CN, Bodzin JL, Saltiel AR. Obesity induces a phenotypic switch in adipose tissue macrophage polarization. *J Clin Investig* 2007;117:175—84.
16. Neeland IJ, Ross R, Despres JP, et al. Visceral and ectopic fat, atherosclerosis, and cardiometabolic disease: a position statement. *Lancet Diabetes Endocrinol* 2019;7:715—25.
17. Keller A, Rohde JF, Raymond K, Heitmann BL. Association between periodontal disease and overweight and obesity: A systematic review. *J Periodontol* 2015;86:766—76.
18. Petersen PE, Ogawa H. The global burden of periodontal disease: towards integration with chronic disease prevention and control. *Periodontol 2000* 2012;60:15—39.
19. Hajishengallis G, Chavakis T. Local and systemic mechanisms linking periodontal disease and inflammatory comorbidities. *Nat Rev Immunol* 2021;21:426—40.
20. Yang LC, Yu HC, Chang YC. The recent new findings of periodontal systemic connection from Taiwan's National Health Insurance Research Database. *J Dent Sci* 2021;16:789—90.

21. Marruganti C, Suvan JE, D'Aiuto F. Periodontitis and metabolic diseases (diabetes and obesity): tackling multimorbidity. *Periodontol 2000* 2023. https://doi.org/10.1111/prd.12536. in press.
22. Suvan JE, Finer N, D'Aiuto F. Periodontal complications with obesity. *Periodontol 2000* 2018;78:98—128.
23. Akram Z, Safii SH, Vaithilingam RD, Baharuddin NA, Javed F, Vohra F. Efficacy of non-surgical periodontal therapy in the management of chronic periodontitis among obese and non-obese patients: A systematic review and meta-analysis. *Clin Oral Invest* 2016;20:903—14.
24. Nascimento GG, Leite FR, Do LG, et al. Is weight gain associated with the incidence of periodontitis? A systematic review and meta-analysis. *J Clin Periodontol* 2015;42:495—505.
25. Pamuk F, Kantarci A. Inflammation as a link between periodontal disease and obesity. *Periodontol 2000* 2022;90:186—96.
26. Kim CM, Lee S, Hwang W, et al. Obesity and periodontitis: a systematic review and updated meta-analysis. *Front Endocrinol* 2022;13:999455.
27. Li Y, Ling J, Jiang Q. Inflammasomes in alveolar bone loss. *Front Immunol* 2021;12:691013.
28. Apolinario Vieira GH, Aparecida Rivas AC, Figueiredo Costa K, et al. Specific inhibition of IL-6 receptor attenuates inflammatory bone loss in experimental periodontitis. *J Periodontol* 2021;92:1460—9.
29. Cekici A, Kantarci A, Hasturk H, Van Dyke TE. Inflammatory and immune pathways in the pathogenesis of periodontal disease. *Periodontol 2000* 2014;64:57—80.
30. Iwashita M, Hayashi M, Nishimura Y, Yamashita A. The link between periodontal inflammation and obesity. *Curr Oral Health Rep* 2021;8:76—83.
31. Tilg H, Moschen AR. Adipocytokines: mediators linking adipose tissue, inflammation and immunity. *Nat Rev Immunol* 2006;6:772—83.
32. Kyrou I, Tsigos C. Stress hormones: physiological stress and regulation of metabolism. *Curr Opin Pharmacol* 2009;9:787—93.
33. Wang Y, Andrukhov O, Rausch-Fan X. Oxidative stress and antioxidant system in periodontitis. *Front Physiol* 2017;8:910.
34. Shang J, Liu H, Zheng Y, Zhang Z. Role of oxidative stress in the relationship between periodontitis and systemic diseases. *Front Physiol* 2023;14:1210449.
35. Tothova L, Celec P. Oxidative stress and antioxidants in the diagnosis and therapy of periodontitis. *Front Physiol* 2017;8:1055.
36. Schamarek I, Anders L, Chakaroun RM, Kovacs P, Rohde-Zimmermann K. The role of the oral microbiome in obesity and metabolic disease: Potential systemic implications and effects on taste perception. *Nutr J* 2023;22:28.
37. Blasco-Baque V, Garidou L, Pomie C, et al. Periodontitis induced by *Porphyromonas gingivalis* drives periodontal microbiota dysbiosis and insulin resistance via an impaired adaptive immune response. *Gut* 2017;66:872—85.
38. Lourenvarsigmao TGB, Spencer SJ, Alm EJ, Colombo APV. Defining the gut microbiota in individuals with periodontal diseases: An exploratory study. *J Oral Microbiol* 2018;10:1487741.
39. Rahman B, Al-Marzooq F, Saad H, Benzina D, Al Kawas S. Dysbiosis of the subgingival microbiome and relation to periodontal disease in association with obesity and overweight. *Nutrients* 2023;15:826.
40. Suvan J, Petrie A, Moles DR, et al. Body mass index as a predictive factor of periodontal therapy outcomes. *J Dent Res* 2014;93:49—54.
41. Arboleda S, Vargas M, Losada S, Pinto A. Review of obesity and periodontitis: An epidemiological view. *Br Dent J* 2019;227:235—9.