Review Article

# Obesity and periodontal disease: A review

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## Abstract

Periodontal diseases usually refer to inflammatory disorders that are caused by pathogenic bacteria in the subgingival biofilm in association with impaired host immune response and connective tissue breakdown. The bacterial challenge exacerbates the cytokine production by the gingival epithelium, resulting in an uncontrolled inflammation that leads to tooth loss in adults from different populations. The prevalence of these diseases increases with aging, longer retention of teeth, and increased incidence of obesity and diabetes among the population. The prevalence demonstrates an increasing trend and a correlation with numerous comorbidities. Hence, as a family physician one should have the in-depth knowledge regarding the relationship between obesity and periodontitis to create awareness among people to provide primary care. Thus, it is relevant to develop new methods capable of detecting these diseases in the early stages and following up on their progression.

**Keywords:** Obesity, overweight, body mass index, periodontal disease, association, systemic conditions

## Introduction

Periodontal diseases are inflammatory diseases affecting the surrounding and supporting tissues of teeth, the periodontium. Gingivitis and destructive periodontal disease (periodontitis) are the two most common forms of periodontal diseases.[1] Gingivitis is an inflammatory reaction that often induces the pathogens residing in dental plaque (biofilm), which forms on the adjacent tooth surfaces. Destructive periodontal disease results in an apical loss of epithelial attachment along with the periodontal soft and hard tissues.[2] Unlike gingivitis, which is cured following the removal of local etiological factors, destructive periodontal disease is irreversible. Destructive periodontal disease is mediated by various intrinsic and acquired factors; two individuals with similar microbiological profile could show different susceptibility to periodontal diseases.[3] Several case-control and cohort studies have reported the contribution of systemic conditions and diseases in the onset and exacerbation of destruction periodontal disease. Preterm birth, cardiovascular diseases, and diabetes are examples of this conditions.[4-6]

In addition, a growing body of evidence during the last decade suggests obesity as a risk factor for destructive periodontal disease.[7,8] Although the majority of studies on destructive periodontal disease in individuals with obesity or metabolic syndrome concentrated on adults, some studies reported on evidence proposing that this potential link in children and adolescence exists.[9] In this paper, we review the evidence suggesting that destructive periodontal disease is linked to obesity and metabolic syndrome, as an example of inter-organ crosstalk under inflammatory conditions.

Obesity is a multifactorial condition with a wide range of etiological factors including genetic, biological, social, and behavioral factors, all of which likely interact to ultimately lead to a chronic imbalance between energy intake and energy expenditure. This imbalance could cause excessive fat accumulation and

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result in adverse health consequences. Obesity has reached epidemic proportions worldwide, largely because of increased consumption of a high caloric diet and a sedentary lifestyle. According to the World Health Organization, approximately 2.3 billion adults will be overweight and more than 700 million will be obese by 2015.[10] This phenomenon affects particularly in developed countries. Over the past two decades, the overall obesity rates have reached 24.1% and 34.4% in Canada and the United States, respectively.[11] Based on current Health Canada guidelines, a body mass index (BMI, kg/m$^2$) of 25–30 and over 30 are considered overweight and obese, respectively.[12] Obesity is categorized into three classes according to the increased health risks associated with increasing BMI levels: class I (BMI 30–34.9), class II (BMI 35–39.9), and class III (BMI ≥40).[13]

Pediatric obesity has also become a public health concern since it is more common for children to experience the negative health consequences of obesity, which used to be only seen in adulthood. In 2010, more than 40 million children under the age of 5 were estimated to be overweight worldwide.[14] In 2004, 26% of Canadian children and adolescents aged 2–17 years were overweight or obese.[15] Obesity has been associated with a wide spectrum of comorbidities, such as coronary artery disease, strokes, diabetes, arthritis, reproductive dysfunctions, and various cancers.[16]

## Role of Leptin, Adiponectin, Tumor Necrosis Factor-alpha

For many years, adipose tissue was considered as an inert organ that stored triglycerides. It is now clear that adipose tissue is a complex and metabolically active endocrine organ that secretes numerous immune-modulatory factors and plays a major role in regulating metabolic and vascular biology. Adipose cells, which include adipocytes, pre-adipocytes, and macrophages, secrete more than 50 bioactive molecules, known collectively as adipokines. Some of these adipokines act locally, whereas others are released into the systemic circulation where they act as signaling molecules to the liver, muscle, and endothelium.[17] Adipokines play a number of different roles, such as hormone-like proteins (e.g., leptin and adiponectin), classical cytokines (e.g., tumor necrosis factor-a, interleukin-6), proteins involved in vascular hemostasis (e.g., plasminogen activator inhibitor-1, tissue factor), regulators of blood pressure (angiotensinogen), promoters of angiogenesis (e.g., vascular endothelial growth factor), and acute-phase respondents (e.g., C-reactive peptide).

**Leptin** is secreted almost exclusively by adipocytes. Leptin signals through the central nervous system and peripherals pathways to suppress appetite and increase energy expenditure. Leptin mimics some of the actions of insulin by increasing glucose uptake in muscle and adipose tissue and by lowering hepatic glucose production.[18] Most obese individuals have elevated leptin levels that do not suppress appetite. Many consider this leptin resistance to be one of the features contributing to obesity's pathology. In obese patients with leptin resistance, leptin may elevate blood pressure and contribute to atherosclerosis and cardiovascular diseases.[19,20]

**Adiponectin** is produced primarily by adipocytes but surprisingly decreased in obese patients, especially those with abdominal obesity. Clinical studies demonstrate inverse associations between adiponectin and serum markers of inflammation.[21]

### Tumor necrosis factor-alfa

Obesity-associated tumor necrosis alpha is primarily secreted from macrophages accumulated in abdominal adipose tissue.[22] Although studies have not shown completely consistent results, it is thought that increased circulating tumor necrosis factor-alpha from adipose tissue contributes to poor health outcomes by increasing insulin resistance and by inducing C reactive peptide production and general systemic inflammation.[23]

## Adipokines and Obesity

Obesity is associated with an increased risk for developing characteristic features of metabolic syndrome, including hypertension, type 2 diabetes, and dyslipidemia. Interestingly, chronic exposure to periodontal pathogens endotoxin and increased cytokine production have been proposed to enhance the risk of causing type 2 diabetes and cardiovascular complications. Obesity has also recently been reported to be associated with periodontitis. Obesity induces macrophage accumulation in adipose tissue, promotes chronic low-grade inflammation, and increases adipokines derived from adipocytes. In this review, we summarize recent advances in understanding the roles of adipokines in chronic inflammatory states such as periodontitis and focus primarily on adiponectin, leptin, and resistin. Understanding the role of adipokines may help elucidate relationships among periodontitis, obesity, type 2 diabetes and cardiovascular diseases.

## Relationship of Obesity and Telomeres

Obesity is a chronic disease characterized by a high inflammatory burden which has been associated with shorter telomeres. A case-control study with 793 French children and young people aged 2-17 years old demonstrated that obese individuals presented telomeres 24% shorter than non-obese controls.[24] A great concern about that result is that, interestingly, another study found that former obese individuals present shorter telomeres in adipose tissue cells when compared to control individuals, which means that alteration in telomere length may be an irreversible phenomenon.[25] However, the relationship between adipocytes and leukocyte telomere length still needs further investigations.

### Polyphenols and periodontal disease

Inflammatory stimulation by periodontal pathogens increases the production of crevicular fluid and induces the chemotaxis of polymorphonuclear leukocytes, which, in order to inactivate periodontal pathogens, release singlet oxygen and hypochlorous acid into the crevicular fluid. The consequent oxidative stress is

countered by the antioxidant activity of ascorbate, albumin and urate present in the crevicular fluid and derived from plasma. However, this local oxidative stress may be increased by external factors or systemic conditions, such as smoking, diabetes, obesity and metabolic syndrome. When there is a disequilibrium between oxidative stress and antioxidant activity, periodontal tissue destruction may appear. These observations suggest that antioxidant-rich diets might inhibit periodontal disease development and progression, particularly in subjects exposed to environmental and dietary sources of oxidative stress.[26-29] Several studies also report that decreased antioxidant activities of crevicular fluid and saliva are associated with the development of periodontitis.[30-32] Polyphenols may contribute to increasing the antioxidant activity of oral fluids. Indeed, delivery of tea Polyphenols by holding green or black tea in the mouth for 2–5 min increases the antioxidant capacity of saliva, and daily consumption of two fresh grapefruits for 2 weeks increases the phagocytic capacity of the polymorphonuclear leucocytes in the gingival crevicular fluid.[33] The finding of an association between obesity and periodontitis adds to the small number of relatively recent studies that found a similar relationship.[9,34-37] Several studies found that the positive association between body mass and periodontitis was most pronounced among women.[7,38,39]

Despite all the evidence about obesity and periodontitis, recommendations on treatment planning, primary health care professionals need to be aware of the complexities of obesity to educate their patients about the importance of maintaining healthy body weight and good oral hygiene.

## Conclusion

Periodontal diseases, chronic inflammation by its nature, have been linked to many systemic conditions. Despite all these pieces of evidence indicating the higher risk of destructive periodontal disease in obesity and metabolic syndrome, the underlying biological mechanism(s) is yet to be fully understood. Studying the common etiological factors in obesity, metabolic syndrome, and destructive periodontal disease would be a potential approach to delineate biological mechanisms explaining the higher risk of destructive periodontal disease under these conditions. Inflammation is indeed one of the common factors in the pathogenesis of the destructive periodontal disease, obesity, and metabolic syndrome. Knowing this fact helps family physicians to provide primary care for the people.

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## Conflicts of interest

There are no conflicts of interest.

## References

1. Pihlstrom BL, Michalowicz BS, Johnson NW. Periodontal diseases. Lancet 2005;366:1809-20.
2. Larsen T, Fiehn NE. Dental biofilm infections-An update. Apmis 2017;125:376-84.
3. Van Dyke TE, Sheilesh D. Risk factors for periodontitis. J Int Acad Periodontol 2005;7:3-7.
4. Offenbacher S, Lieff S, Boggess KA, Murtha AP, Madianos PN, Champagne CM, *et al.* Maternal periodontitis and prematurity. Part I: Obstetric outcome of prematurity and growth restriction. Ann Periodontol 2001;6:164-74.
5. Scannapieco FA, Bush RB, Paju S. Associations between periodontal disease and risk for atherosclerosis, cardiovascular disease, and stroke. A systematic review. Ann Periodontol 2003;8:38-53.
6. Taylor GW. Bidirectional interrelationships between diabetes and periodontal diseases: An epidemiologic perspective. Ann Periodontol 2001;6:99-112.
7. Alabdulkarim M, Bissada N, Al-Zahrani M, Ficara A, Siegel B. Alveolar bone loss in obese subjects. J Int Acad Periodontol 2005;7:34-8.
8. Nishimura F, Iwamoto Y, Mineshiba J, Shimizu A, Soga Y, Murayama Y. Periodontal disease and diabetes mellitus: The role of tumor necrosis factor-alpha in a 2-way relationship. J Periodontol 2003;74:97-102.
9. Al-Zahrani MS, Bissada NF, Borawskit EA. Obesity and periodontal disease in young, middle-aged, and older adults. J Periodontol 2003;74:610-5.
10. Katz AR. Noncommunicable diseases: Global health priority or market opportunity? An illustration of the World Health Organization at its worst and at its best. Int J Health Serv 2013;43:437-58.
11. McGuire S. Shields M., Carroll M.D., Ogden C.L. Adult obesity prevalence in Canada and the United States. NCHS data brief no. 56, Hyattsville, MD: National Center for Health Statistics, 2011. AdvNutr. 2011;2:368–369.
12. Lau DC, Douketis JD, Morrison KM, Hramiak IM, Sharma AM, Ur E. 2006 Canadian clinical practice guidelines on the management and prevention of obesity in adults and children [summary]. CMAJ 2007;176:S1-13.
13. Tjepkema M. Adult obesity. Health Rep 2006;17:9-25.
14. Dhurandhar NV, Bailey D, Thomas D. Interaction of obesity and infections. Obes Rev 2015;16:1017-29.
15. Taljaard M, Tuna M, Bennett C, Perez R, Rosella L, Tu JV, *et al.* Cardiovascular disease population risk tool (CVDPoRT): Predictive algorithm for assessing CVD risk in the community setting. A study protocol. BMJ Open 2014;4:e006701.
16. Guh DP, Zhang W, Bansback N, Amarsi Z, Birmingham CL, Anis AH. The incidence of co-morbidities related to obesity and overweight: A systematic review and meta-analysis. BMC Public Health 2009;9:88.
17. Trayhurn P, Wood IS. Adipokines: Inflammation and the pleiotropic role of white adipose tissue. Br J Nutr 2004;92:347-55.
18. Matsuzawa Y. White adipose tissue and cardiovascular disease. Best Pract Res Clin Endocrinol Metab 2005;19:637-47.
19. Correia ML, Haynes WG. Obesity-related hypertension: Is there a role for selective leptin resistance? Curr Hypertens Rep 2004;6:230-5.
20. Reilly MP, Iqbal N, Schutta M, Wolfe ML, Scally M, Localio AR, *et al.* Plasma leptin levels are associated with coronary atherosclerosis in type 2 diabetes. J Clin Endocrinol Metab 2004;89:3872-8.

21. Ouchi N, Kihara S, Funahashi T, Nakamura T, Nishida M, Kumada M, *et al*. Reciprocal association of C-reactive protein with adiponectin in blood stream and adipose tissue. Circulation 2003;107:671-4.
22. Tsigos C, Kyrou I, Chala E, Tsapogas P, Stavridis JC, Raptis SA, *et al*. Circulating tumor necrosis factor alpha concentrations are higher in abdominal versus peripheral obesity. Metabolism 1999;48:1332-5.
23. Berg AH, Scherer PE. Adipose tissue, inflammation, and cardiovascular disease. Circ Res 2005;96:939-49.
24. Buxton JL, Walters RG, Visvikis-Siest S, Meyre D, Froguel P, Blakemore AI. Childhood obesity is associated with shorter leukocyte telomere length. J Clin Endocrinol Metab 2011;96:1500-5.
25. Moreno-Navarrete JM, Ortega F, Sabater M, Ricart W, Fernandez-Real JM. Telomere length of subcutaneous adipose tissue cells is shorter in obese and formerly obese subjects. Int J Obes (Lond) 2010;34:1345-8.
26. Battino M, Bullon P, Wilson M, Newman H. Oxidative injury and inflammatory periodontal diseases: The challenge of anti-oxidants to free radicals and reactive oxygen species. Crit Rev Oral Biol Med 1999;10:458-76.
27. Sculley DV, Langley-Evans SC. Salivary antioxidants and periodontal disease status. Proc Nutr Soc 2002;61:137-43.
28. Ritchie CS, Kinane DF. Nutrition, inflammation, and periodontal disease. Nutrition 2003;19:475-6.
29. Figuero E, Soory M, Cerero R, Bascones A. Oxidant/antioxidant interactions of nicotine, Coenzyme Q10, Pycnogenol and phytoestrogens in oral periosteal fibroblasts and MG63 osteoblasts. Steroids 2006;71:1062-72.
30. Diab-Ladki R, Pellat B, Chahine R. Decrease in the total antioxidant activity of saliva in patients with periodontal diseases. Clin Oral Investig 2003;7:103-7.
31. Sculley DV, Langley-Evans SC. Periodontal disease is associated with lower antioxidant capacity in whole saliva and evidence of increased protein oxidation. Clin Sci (Lond) 2003;105:167-72.
32. Pavlica Z, Petelin M, Nemec A, Erzen D, Skaleric U. Measurement of total antioxidant capacity in gingival crevicular fluid and serum in dogs with periodontal disease. Am J Vet Res 2004;65:1584-8.
33. Lee MJ, Lambert JD, Prabhu S, Meng X, Lu H, Maliakal P, *et al*. Delivery of tea polyphenols to the oral cavity by green tea leaves and black tea extract. Cancer Epidemiol Biomarkers Prev 2004;13:132-7.
34. Saito T, Shimazaki Y, Koga T, Tsuzuki M, Ohshima A. Relationship between upper body obesity and periodontitis. J Dent Res 2001;80:1631-6.
35. Wood N, Johnson RB, Streckfus CF. Comparison of body composition and periodontal disease using nutritional assessment techniques: Third national health and nutrition examination survey (NHANES III). J Clin Periodontol 2003;30:321-7.
36. Federica DS, Ludovico S, Vincenzo P, Francesco DA. Obesity and periodontal disease: A narrative review on current evidence and putative molecular links. Open Dent J 2019:13;526-36.
37. Prabhati G, Romesh S. Relationship between obesity and periodontitis: A cross sectional study. Int J Appl Dent Sci 2019;5:215-8.
38. Dalla Vecchia CF, Susin C, Rosing CK, Oppermann RV, Albandar JM. Overweight and obesity as risk indicators for periodontitis in adults. J Periodontol 2005;76:1721-8.
39. Silie A, Miguel V, Sergio L, Andres P. Review of obesity & periodontitis: An epidemiological view. Br J Dent 2019;227:235-9.