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# Association Between Periodontal Health and Obesity

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# Abstract:

This comprehensive review examines the intricate association between obesity and periodontal health. Over the years, research has unveiled a strong correlation between these two seemingly distinct health concerns. Obesity, characterized by excessive adipose tissue, is known to induce chronic inflammation throughout the body, including the oral cavity. This systemic inflammation is one of the key factors contributing to an increased susceptibility to periodontal disease in obese individuals. Furthermore, alterations in the oral microbiome in obese individuals can create an environment conducive to the growth of periodontal pathogens. This review highlights the various biological and behavioral mechanisms linking obesity and periodontal health, emphasizing the importance of both systemic health management and good oral hygiene practices in mitigating the risks associated with this relationship. Understanding this connection is vital for healthcare professionals in their efforts to improve overall health and quality of life for individuals dealing with obesity and periodontal issues.

Keywords: Obesity, periodontal disease, Adipocytes, leptin, bone loss, adipose tissue, BMI

# Introduction

Periodontal diseases are inflammatory diseases that affect the surrounding and supporting tissues of the teeth – the periodontium. Gingivitis and destructive periodontal disease (periodontitis) are the two most common forms of periodontal disease [1]. Destructive periodontal disease causes apical loss of epithelial attachment and periodontal soft and hard tissues [2]. Destructive periodontal disease is mediated by various intrinsic and acquired factors; two individuals with similar microbiological profiles may have different susceptibility to periodontal disease [3]. In addition, in the last decade, increasing evidence shows that obesity is a risk factor for devastating periodontal disease. [4,5] 2. Obesity, defined as body mass index (BMI) and >30.0 kg/m2, is an important public . health problem today. The prevalence of obesity has increased significantly in most industrialized countries in recent decades. Obesity is a risk factor for several chronic diseases, especially hypertension, type 2 diabetes, dyslipidemia and coronary disease.[7-9] 1. Recent studies have shown that obesity is also related to oral diseases, especially periodontitis[10,11]]. . Most clinical studies have shown an association between obesity and decreased bone mass [12]. According to the 2017 classification of periodontal diseases, obesity has been recognized as a major metabolic disorder associated with periodontal tissue loss (13). Obesity-related imbalanced bone homeostasis and an inflammatory environment were particularly well-established (14).Numerous investigations have examined the potential molecular connections between obesity and periodontitis, emphasising the ideas of immunological dysfunction and common inflammatory pathways (15).An inflammatory state brought on by obesity may make a host more vulnerable to periodontal disease. Although it is not well understood, obesity and periodontitis are likely significantly influenced by an imbalance between the immune and inflammatory systems of the host (16). Adipose tissue, also



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known as fat, is largely in charge of controlling bodily energy and, in a healthy state, keeps energy storage and exercise in balance. Visceral adipose tissue, or abnormal or excessive abdominal fat, is linked to a number of medical disorders and is more strongly associated with poor health outcomes than excess subcutaneous fat (17). Numerous comorbid conditions, including diabetes, arthritic conditions, strokes, cancers, and reproductive disorders, have been linked to obesity(18).

## SYSTEMIC DISEASES ASSOCIATED WITH OBESITY

Body mass index (BMI, also known as Quetelet Index) is the square of body height (in metres) divided by body weight (in kilogrammes) and is used to define obesity. Waist circumference measures the distribution of body fat; a man's waist circumference of 102 cm and a woman's waist circumference of 88 cm respectively indicate abdominal obesity and are linked to a higher risk of morbidity. It has been demonstrated that visceral adipose tissue secretes much more hormones and cytokines and is metabolically more active than subcutaneous adipose tissue (19).

The question of whether waist circumference, BMI, or both should be used to determine a person's risk of disease is still being thoroughly researched (20).

Obesity and overweight have long been known to be significant risk factors for high blood pressure. Obese people have a five-fold increased risk of hypertension when compared to those of normal weight. Numerous factors have been linked to the development of obesity-related hypertension, including elevated sympathetic nervous activity, abnormalities of the kidneys, sodium and volume retention, insulin resistance, hyperleptinemia, and increased angiotensinogen secretion from adipocytes (9,21).

**Diabetes Mellitus Type 2:** Insulin resistance and beta cell death work together to cause type 2 diabetes. Liposomal and glucose-derived toxins, along with cytokines originating from obesity, have been linked to these processes (22).

**Cardiovascular Disease** – Obese persons have an 1.5-fold increased risk for cardiovascular disease .Obesity is also associated with an about 2-fold higher risk of heart failure and a 50% increased risk of atrial fibrillation(23,19).

**Osteoarthritis** – Recent studies have proved that being overweight antedates the development of knee osteoarthritis and increases the risk of radiographic progression(24).

**Respiratory Disorders** – Obesity is the major reversible risk factor for obstructive sleep apnea syndrome. Waist circumference tends to be a better predictor of this syndrome than body mass index(24).

**Cardiovascular Disease:** The risk of cardiovascular disease is 1.5 times higher in obese individuals. Additionally, obesity is linked to a 50% increased risk of atrial fibrillation and a roughly two-fold increased risk of heart failure (23, 19).

Obesity is the primary modifiable risk factor for obstructive sleep apnea syndrome in the respiratory disorders category.Compared to body mass index, waist circumference is generally a better indicator of this syndrome (24).

## **OBESITY INFLUENCE BONE QUALITY**

Obese patients are also at increased risk for metabolic complications, including hypertension, T2DM, heart disease, nonalcoholic fatty liver disease, kidney disease, polycystic ovarian disease, and cancer. Over the past decade, an increasing number of studies have shown that obesity is associated with osteopenia and osteoporosis, suggesting a negative effect of obesity on bone quality, including the jaw. Bone is a dynamic organ that is in a continuous process of resorption followed by remodeling/reconstruction as the most important biological process during life. Bone homeostasis



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involves the balance between bone formation and resorption produced by osteoblasts and osteoclasts. The first belief was that obesity is correlated with increased bone mass and long-term weight gain. Is useful in the formation of bone tissue. Excess fat and osteoporosis, which makes the bone more fragile. A previous study showed that weight gain can cause a decrease in alveolar bone crest height, suggesting obesity as a putative risk factor even in a clinically healthy periodontium.

## ASSOCIATION BETWEEN PERIODONTAL HEALTH AND OBESITY

It has been suggested that obesity is second only to smoking as the strongest risk factor for inflammatory periodontal tissue destruction. In addition, studies have indicated that the fat distribution pattern plays a crucial role in the association with periodontitis. Nutrition Examination Survey (NHANES III) data and demonstrated that BMI was positively correlated with the severity of periodontal attachment loss; they found that this relationship is modulated by insulin resistance. Fat tissue is not merely a passive triglyceride reservoir of the body, but also produces a vast amount of cytokines and hormones.

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

It has been proposed that corpulence is second just to smoking as the most grounded risk factor for incendiary periodontal tissue annihilation. What's more, studies have demonstrated that the fat conveyance design assumes a significant part in the relationship with periodontitis. Sustenance Assessment Study (NHANES III) information and exhibited that BMI was emphatically connected with the seriousness of periodontal disease, they observed that this relationship is tweaked by insulin opposition. Fat tissue isn't simply a detached fatty substance repository of the body, yet in addition creates a tremendous measure of cytokines and chemicals, by and large called adipokines or adipocytokines, which plays a major role in inflammatory reactions.

## Role of Leptin, Adiponectin, Tumor Necrosis factor-alpha and Adipokines

Fat cells, which incorporate adipocytes, pre-adipocytes, and macrophages, discharge in excess of 50 bioactive atoms, referred to all in all as adipokines. A portion of these adipokines act locally, while others are delivered into the foundational dissemination where they go about as flagging particles to the liver, muscle, and endothelium.

#### • Leptin

Leptin is emitted only by adipocytes. Leptin signals through the focal sensory system and peripherals pathways to smoother hunger and increment energy consumption. Leptin imitates a portion of the activities of insulin by expanding glucose take-up in muscle and fat tissue and by bringing down hepatic glucose creation. Most corpulent people have raised leptin levels that don't stifle craving. Many believe this leptin protection from be one of the elements adding to stoutness' pathology. In hefty patients with leptin opposition, leptin may lift pulse and add to atherosclerosis and cardiovascular diseases(25).

#### • Adiponectin

Adiponectin is created basically by adipocytes however shockingly diminished in fat patients, particularly those with stomach corpulence. Adiponectin is a 30 KDa adipokine encoded by the AdipoQ quality, principally created and discharged by adipocytes and profoundly bountiful in human plasma. Adiponectin is known to increment insulin awareness of target organs like liver and muscle, eventually controlling fringe glucose and unsaturated fat digestion . Other than being a metabolic controller, adiponectin is likewise known for its mitigating and hostile to oxidant movement. These qualities make adiponectin a defensive consider conditions like weight, type 2 diabetes and cardiovascular illnesses. Levels of flowing adiponectin are diminished in heftiness and metabolic disorder, logical adding to the improvement of insulin obstruction.



# • Tumor Necrosis factor-alpha

Weight related growth putrefaction alpha is fundamentally emitted from macrophages amassed in stomach fat tissue.(26) In spite of the fact that reviews have not shown totally predictable outcomes, through that circulating tumor Necrotic factor -alpha from fat tissue adds to chronic weakness results by expanding insulin obstruction and by actuating C receptive peptide creation and general foundational inflammation(27).

### • Adipokines

Weight has likewise as of late been accounted for to be related with periodontitis. Stoutness prompts macrophage collection in fat tissue, advances ongoing second rate irritation, and increments adipokines got from adipocytes. In this audit, we sum up late advances in understanding the jobs of adipokines in persistent fiery states, for example, periodontitis and spotlight fundamentally on adiponectin, leptin, and resistin. Understanding the job of adipokines may assist with explaining connections among periodontitis, stoutness, type 2 diabetes and cardiovascular sicknesses

INFLAMMATORY MARKERS	PERIODONTAL DISEASE	OBESITY
Leptin	$\checkmark$	$\checkmark$
Tumor necrosis factor -alpha	$\checkmark$	$\checkmark$
Interleukin -6	$\checkmark$	$\checkmark$
Adiponectin	$\checkmark$	$\checkmark$
Oxidative stress	$\checkmark$	$\checkmark$

## DISCUSSION

The relationship among corpulence and periodontitis was predictable with a convincing example of expanded chance of periodontitis in overweight or fat people. Albeit the hidden pathophysiological system stays muddled, it has been brought up that the improvement of insulin opposition as a result of a constant provocative state and oxidative pressure could be embroiled in the relationship among weight and periodontitis. Effects of heftiness on treatment reaction to nonsurgical periodontal treatment stay changed. While certain reports noticed no massive contrasts in the periodontal boundaries, including PPD and CAL, among corpulent and non-hefty people following nonsurgical periodontal treatment(28). Others demonstrated a second rate reaction to periodontal treatment in hefty patients(29). A helpfully examined clinical preliminary showed that weight reduction through dietary treatment worked on the reaction of stout subjects to nonsurgical periodontal treatment (30)Obese patients who had critical weight reduction after bariatric medical procedure likewise showed a more exceptional improvement in periodontal boundaries after nonsurgical therapy than the people who didn't have such a medical procedure.

## CONCLUSION

The relationship among weight and periodontal wellbeing is a perplexing and multi-layered relationship. Research has reliably shown that weight is a gamble factor for periodontal sickness, essentially because of the constant irritation that stoutness incites all through the body, including the oral pit. Besides, the modifications in the oral microbiome in stout people add to an elevated helplessness to gum illness.

Successful management of stoutness and periodontal wellbeing ought to include an all encompassing methodology. Empowering weight the board, taking on a reasonable eating regimen, and advancing normal actual work are fundamental for decreasing the foundational irritation related with corpulence. All the while, accentuating the significance of good oral cleanliness rehearses, like standard brushing,



flossing, and proficient dental consideration, is critical in keeping up with periodontal wellbeing.

Medical care experts, as well as people, should perceive this transaction and the potential dangers it presents. Thorough consideration that tends to both stoutness and periodontal wellbeing can essentially further develop the general prosperity of people, limiting the effect of these wellbeing concerns and improving their personal satisfaction. Understanding and tending to this affiliation is a vital stage towards accomplishing better wellbeing results and forestalling the inconveniences that might emerge from both heftiness and periodontal sickness.

# 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. 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
- 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
- 6. Expert Panel. Executive summary of the clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. Arch Intern Med. 1998;158:1855–67.
- 7. Mokdad AH, Ford ES, Bowman BA, Dietz WH, Vinicor F, Bales VS, et al. Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. JAMA. 2003;289:76–9.
- 8. Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. JAMA. 1999;282:1523–9.
- 9. Perlstein MI, Bissada NF. Influence of obesity and hypertension on the severity of periodontitis in rats. Oral Surg Oral Med Oral Pathol. 1977;43:707–19.
- 10. Saito T, Shimazaki Y, Koga T, Tsuzuki M, Ohshima A. Relationship between upper body obesity and periodontitis. J Dent Res. 2001;80:1631–6.
- 11. Dalla Vecchia CF, Susin C, Rösing CK, Oppermann RV, Albandar JM. Overweight and obesity as risk indicators for periodontitis in adults. J Periodontol. 2005;76:1721–8.
- 12. Gkastaris, K.; Goulis, D.G.; Potoupnis, M.; Anastasilakis, A.D.; Kapetanos, G. Obesity, osteoporosis and bone metabolism. J. Musculoskelet. Neuronal Interact. 2020, 20, 372–381.
- 13. Albandar, J.M.; Susin, C.; Hughes, F.J. Manifestations of systemic diseases and conditions that affect the periodontal attachment apparatus: Case definitions and diagnostic considerations. J. Clin. Periodontol. 2018, 45 (Suppl. S20), S171–S189.
- 14. Benova, A.; Tencerova, M. Obesity-induced changes in bone marrow homeostasis. Front. Endocrinol. 2020, 11, 294
- 15. Jepsen, S.; Suvan, J.; Deschner, J. The association of periodontal diseases with metabolic syndrome and obesity. Periodontol. 2000 2020, 83, 125–153
- 16. Kangas S, Timonen P, Knuuttila M, Jula A, Ylostalo P, Syrjala AH. Waist circumference and waist-to-height ratio are associated with periodontal pocketing-results of the Health 2000 Survey. BMC Oral Health. 2017;17:1–7.
- 17. Ritchie C. Obesity and periodontal disease. Periodontol 2000. 2007;44:154-163.
- 18. 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
- 19. Pouliot MC, Després JP, Lemieux S, Moorjani S, Bouchard C, Tremblay A, Nadeau A, et al. Waist circumference and abdominal sagittal diameter: best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. Am J Cardiol. 1994;73:460–8.



- 20. Wang Y, Rimm EB, Stampfer MJ, Willett WC, Hu FB. Comparison of abdominal adiposity and overall obesity in predicting risk of type 2 diabetes among men. Am J Clin Nutr. 2005;81:555–63.
- 21. Haslam DW, James WP. Obesity. Lancet. 2005;366:1197–209.
- 22. Wilson PW, D'Agostino RB, Sullivan L, Parise H, Kannel WB. Overweight and obesity as determinants of cardiovascular risk: the Framingham experience. Arch Intern Med. 2002;162:1867–72
- 23. Stumvoll M, Goldstein BJ, van Haeften TW. Type 2 diabetes: principles of pathogenesis and therapy. Lancet. 2005;365:1333–46.
- 24. Ritchie CS. Obesity and periodontal disease. Periodontol. 2007;44:154-63.
- 25. Correia ML, Haynes WG. Obesity-related hypertension: Is there a role for selective leptin resistance? Curr Hypertens Rep. 2004;6:230–5
- 26. 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
- 27. Berg AH, Scherer PE. Adipose tissue, inflammation, and cardiovascular disease Circ Res. 2005;96:939-49
- 28. Zuza, E.P.; Barroso, E.M.; Carrareto, A.L.; Pires, J.R.; Carlos, I.Z.; Theodoro, L.H.; Toledo, B.E. The role of obesity as a modifying factor in patients undergoing non-surgical periodontal therapy. J. Periodontol. 2011, 82, 676–682.
- Suvan, J.; Petrie, A.; Moles, D.R.; Nibali, L.; Patel, K.; Darbar, U.; Donos, N.; Tonetti, M.; D'Aiuto, F. Body mass index as a predictive factor of periodontal therapy outcomes. J. Dent. Res. 2014, 93, 49–54.
- Martinez-Herrera, M.; López-Domènech, S.; Silvestre, F.J.; Silvestre-Rangil, J.; Bañuls, C.; Hernández-Mijares, A.; Rocha, M. Dietary therapy and non-surgical periodontal treatment in obese patients with chronic periodontitis. J. Clin. Periodontol. 2018, 45, 1448–1457.