

The Internet Journal of Allied Health Sciences and Practice

http://ijahsp.nova.edu

A Peer Reviewed Publication of the College of Allied Health & Nursing at Nova Southeastern University Dedicated to allied health professional practice and education <u>http://ijahsp.nova.edu</u> Vol.5 No. 1 ISSN 1540-580X

Obesity and Its Role in Oral Health

Ashley J. Karels, B.S.D.H.¹ Brigette R. Cooper, M.S.²

- 1. Minnesota State University, Mankato
- 2. Assistant Professor, Minnesota State University, Mankato

United States

Citation:

Karels, A., Cooper, B. Obesity and its role in oral health. *The Internet Journal of Allied Health Sciences and Practice*. Jan 2007, Volume 5 Number 1.

Abstract

Obesity is a serious public health concern that has reached epidemic proportions. This paper addresses the role obesity plays in several health conditions, in addition to how it negatively affects a person's oral health. Oral health care providers can have a positive impact on treatment outcomes by recognizing patients at risk for obesity and addressing these issues.

Overview of Obesity

Obesity is recognized as a growing health problem, currently representing the second leading cause of preventable death in the U.S.¹ It is the most common metabolic and nutritional disease in the U.S., with approximately fifty-nine million Americans classified as obese.² By definition, obesity is an unhealthy accumulation of body fat with an excessively high amount of adipose tissue in relation to lean body mass.² It is the end result of an imbalance between food eaten and energy expended.³ The Report of the Surgeon General stated the cause of obesity guite simply, saying obesity is a result from an energy imbalance by eating too many calories and getting too little exercise.⁴ Lifestyle accounts for approximately seventy percent of an individual's total risk for obesity and genetics account for the remaining thirty percent.⁵ It is more common in females, minorities, persons with low socio-economic status, and has increased dramatically among children and adults.6 The growing prevalence of obesity and increased body weight in the U.S. has created significant public health concerns. The impact of obesity on health status has the same outcome as twenty years of aging and has been indicated to exceed the impact of smoking or alcohol abuse.⁷ It is a risk factor for several chronic health conditions, as well as being associated with increased mortality.7 Hypertension, high cholesterol, type 2

diabetes, periodontal disease, heart disease, stroke, and certain cancers are a few negative health effects correlated to obesity.⁸ Early onset obesity, or obesity in younger adults, continues into adulthood and may be more harmful than obesity that appears in middle-age and older adults.⁷ Aging is correlated with an increase in body fat mass, so it is assumed most obesity in younger age groups begins earlier in life, while older obese persons may gain weight as part of the aging process.⁷

Role of Adipose Tissue

To fully understand obesity, the underlying factor of adipose tissue should be addressed. Adipose tissue is loose connective tissue composed of cells called adipocytes.³ Its major role is to store energy in the form of fat as it insulates and cushions the body, and it also functions as a reserve of nutrients.³ The liver plays an important role in fat and adipose tissue. The liver synthesizes lipoproteins to transport fat to other tissues where they are a source of energy.³ Lipoproteins are formed by triglycerides combined with proteins in the blood. Adipose tissue assists endocrine function by producing three hormones, resistin, leptin and tumor necrosis factor– α .⁸ Adipose tissue is primarily located beneath the skin, but is also found around internal organs.³ It accumulates in the deepest layer of skin, the subcutaneous layer, providing

insulation from heat and cold. This type of adipose tissue is also called subcutaneous fat. Fat around organs is termed visceral fat, where it provides protective padding.

Ways to Measure Obesity

The most common way to measure obesity is with the body mass index (BMI). The BMI represents weight levels associated with the lowest overall risk to health and is an indicator of overall adiposity.3 Body mass index is computed from weight in kilograms (kg) divided by square height in meters (m). There are four BMI categories: underweight (less than 18.5 kg/m²), normal (18.5 to 24.9 kg/m²), overweight (25.0 to 29.9 kg/m²), and obese (more than 30.0 kg/m²).² Persons twenty to forty percent above their ideal weight are labeled as mildly obese, with ninety percent of all obese people in this category.² Those persons forty-one to ninety-nine percent above their ideal weight are described as moderately obese, and about seven to eight percent of the obese fit into this category.² Persons one-hundred percent or more above their ideal weight are classified as severely, morbidly, or grossly overweight, which accounts for two to three percent of the obese population.² Men's bodies should contain between eleven and fifteen percent total body fat, and women should be within the range of eighteen and twenty-two percent body fat.² When men exceed twenty percent body fat and women exceed thirty percent body fat, they have reached obesity.²

Other ways to measure obesity include waist circumference (WC), waist-to-hip ratio, and total body fat. Waist circumference is measured to determine abdominal obesity.7 The cutoff point from normal to overweight patients for WC is 102 centimeters (cm) for men and 88 cm for women.7 A high WC is associated with an increased risk for type two diabetes, dyslipidemia, hypertension, and cardiovascular disease in persons with a BMI in a range between 25 and 34.9 kg/m.10 Waist-to-hip ratio is simply taking the waist measurement and comparing it to the hip measurement to determine fat distribution in the body. Total body fat is measured to compare amount of body fat in the body to amount of lean body mass, which determines how much healthy body tissue is present. Body mass index, waist circumference, waist-hip ratio, and total body fat are factors in the incidence of obesity in conjunction with periodontal disease.¹²

Obesity's Role in Periodontal Disease

Periodontitis is an inflammatory disease process that results in permanent destruction to tissues of the periodontium, including the loss of gingival connective tissue, destruction of the periodontal ligament, and resorption of alveolar bone.¹² Inflammation is the body's response to injury by disease-causing organisms, and inflammation in the mouth is the body's reaction to bacterial infection of the tissues that support the teeth.¹ Periodontal

disease is a common chronic disease divided into two categories, gingivitis and periodontitis. Gingivitis is an early, reversible stage of periodontal disease, and periodontitis is an irreversible, progressed stage of periodontal disease. It has been estimated at least thirtyfive percent of U.S. adults between ages thirty and ninety have periodontal disease.7 Several systemic factors are associated with increased risk of periodontal disease, including diabetes, smoking, osteoporosis, stress, and age.⁶ Periodontitis is more prevalent among smokers than non-smokers, older than younger, and males than female.13 Obesity is a risk factor for periodontitis, with a higher prevalence of periodontitis instituted among obese patients.¹⁴ A larger percentage of obese individuals at risk for periodontitis will develop this disease as a result of their obesity condition.13 Increased BMI is associated with gingival bleeding, a symptom of periodontal disease. Additionally, periodontal disease is associated with weight gain.¹⁵ Obesity, especially upper-body obesity, is extensively associated with deep probing depths, which is a sign of destruction associated with periodontal disease.¹⁶

Adipokines and Their Role in Obesity

Adipose tissue is an active endocrine organ that secretes numerous cytokines, or protein mediators, collectively known as adipokines.14 Tumor necrosis factor alpha (TNF- α), interleukin-6 (IL-6), interleukin-8 (IL-8), and plasminogen activator inhibitor-1 (PAI-1) are some of the adipokines that are most prevalent in the inflammatory response.14 Many of these cytokines are secreted in proportion to the amount of adipose tissue present.15 These adipokines affect the metabolism of the entire body and contribute to low-grade systemic and vascular inflammation due to accumulation of gram-negative bacteria and inflammatory mediators.¹⁵ Several of these adipokines in plasma are enhanced in obese patients with large amounts of adipose tissue.¹⁴ Increasing body fat may stimulate a hyperinflammatory response in periodontal disease.¹⁵ Obesity may have the potential for transforming the host's immunity and inflammatory system, causing the patient to be more at risk to the effects of microbial plaque.¹⁵

Among the many inflammatory and immune mediators that are established in gingival crevicular fluid (GCF), the cytokine TNF- α plays a significant role in the pathogenesis and development of periodontitis, and an increased level of TNF- α has been found in GCF in patients with periodontitis.¹⁴ Additionally, the cytokine IL-8 is also enhanced in GCF in subjects with periodontal disease.14 IL-8 is a powerful neutrophil chemoattractant that is produced in response to various inflammatory stimuli, including TNF- α .¹⁴ Obesity contributes to a proinflammatory environment by producing pro-inflammatory cytokines, and the levels of TNF- α and IL-8 in GCF are directly linked to the extent of obesity in relation to BMI.¹⁴ The cytokine TNF-a negatively affects the host immunity in periodontal tissue, causing obesity to function as a risk factor for periodontal disease.¹⁴

Periodontal blood vessels in obese persons show a thickening in their innermost membrane, which indicates diminished blood flow in the periodontium.¹⁶ Plasminogen activator inhibitor-1 (PAI-1) is another adipokine enhanced in visceral fat, which generates agglutination of blood and raises the risk of ischemic vascular disease.¹⁶ The plasminogen-activating system plays an important role in gingival inflammation, and PAI-1 may decrease blood flow in the periodontium of obese patients and promote development of periodontitis.¹⁷

The increased serum TNF-a concentration in obese patients may initiate or intensify pre-existing periodontal disease in several ways.¹³ TNF-a may stimulate fibroblasts (connective tissue cells) to synthesize matrix-degrading enzymes.13 It may also stimulate osteoclasts (bone destroying cells) to activate bone resorption. Adipokine IL-6 has been shown to be expressed in human adipose tissue and released into the circulation where it is involved in destruction of both periodontal alveolar bone and connective tissue.¹³ Successful periodontal treatment, including removal of microbial biofilm, may reduce the levels of circulating TNF- α in persons with periodontal disease and improve metabolic control in diabetes through reduction of insulin resistance.8 Moreover, periodontal treatment is known to decrease the serum level of glycosylated hemoglobin, high sugar-containing red blood cell components, and have a positive effect on diabetic control.¹⁶

TNF-a mediates injury in different organs and periodontal tissues in the body.¹⁶ The liver is involved in lipid metabolism, and adipose tissue is shown to secrete TNF-a, causing liver injury.16 In addition, TNF-a from adipose tissue is directly associated with insulin resistance.16 An increase in trialvcerides in the liver is contingent on increased flow of free fatty acids, primarily obtained from visceral adipose tissue, and is associated with insulin resistance.¹⁶ An increase of lipids in the blood, known as hyperlipidemia, is frequently associated with infectious diseases, and a correlation between periodontitis and hyperlipidemia has been reported.¹⁶ Hepatic dyslipidemia, an abnormal amount of fat in the liver that causes pain, may be associated with insulin resistance if produced by periodontal pathogens.¹⁶ Small amounts of bacterial endotoxins or lipopolysaccharides (LPS) produced by periodontal pathogens may include changes in lipid metabolism in adipose tissue and liver.¹⁶ If LPS mediates the release of TNF-α from adipose tissue, developed from gram-negative bacteria in periodontal pockets, it may be affiliated with hepatic dyslipidemia.¹⁶

Obesity and Diabetes

The significant increase in adult onset type two diabetes in the U.S. is primarily due to the increase in obesity by reducing glucose tolerance status in these patients.9 Currently, approximately thirteen million people in the U.S. are diabetic, and the incidence of type two diabetes and obesity is increasing among adolescents.¹⁸ Obese patients require more insulin to maintain homeostasis in their blood glucose, a state known as hyperinsulinemia.9 Diabetes mellitus is a chronic metabolic disorder resulting either from the pancreas failing to produce insulin (type one diabetes mellitus) or with inadequate insulin secretion to sustain normal metabolism from insulin resistance (type two diabetes mellitus).³ Type two diabetes mellitus primarily affects obese middle-aged individuals with sedentary lifestyles.³ The cytokine TNF-a secreted by adipose tissue is assumed to be critical in the pathogenesis of non-insulin dependent diabetes mellitus and in insulin resistance.14 TNF-a from adipose tissue is directly associated with insulin resistance, where it has been suggested to explain the association between obesity and periodontitis.13 Patients with type two diabetes are twice as prone to periodontal disease as healthy patients, all other factors being equal.¹³ Obesity is a risk factor for both type two diabetes and periodontal infection, and diabetes also heightens risk for gum disease.14 Inflammation links all three, triggered by the proinflammatory cytokines manufactured by fat tissue and produced locally by gum infection.19

Obesity has been known to lower insulin sensitivity, which leads to insulin resistance.⁹ Giant fat cells, or adipocytes, are often apparent in fat tissues of obese patients and have multifunctional properties.⁹ Fat cells are known to have an important role in regulating body energy, secreting many biologically active molecules and hormones.⁹ Leptin, a hormone produced by adipose tissue, plays an essential role in regulating body weight by increasing storage of body fat, suppressing appetite, and contributing to the secretion of insulin by the pancreas.⁹ The concentration of serum leptin is elevated in obese people due to increased levels of adipose tissue and negatively regulates their appetite.⁹ This condition is also known as leptin resistance. Obese persons may be characterized by two distinct states of resistance, leptin resistance and insulin resistance.⁹

Obesity Prevention

The entire body ceases to function properly with excess fat and begins to go through changes with a negative result. Prevention is the key aspect to a healthy body before obesity may lead to disease and damage to the body, including oral health. To reverse the trend of obesity in the U.S., many Americans should increase their activity and make more sensible food choices.¹⁵ Fruits, vegetables, whole grains, and low-fat milk products are important to a healthy diet and are excellent sources of essential nutrients.¹⁵ Diets high in dietary fiber have several beneficial effects, including decreased heart disease and type two diabetes.¹⁵ Regular physical activity is an important factor in accomplishing and maintaining a healthy body weight for adults and children.¹⁵ Nutrition is an essential component of oral health, which is an important part of systemic health. Nutrient deficiencies, especially in vitamin C and calcium, may compromise the systemic reaction to inflammation and infection and alter nutrient need. Low dietary intake of calcium and vitamins C are associated with periodontal disease, and deficiency in nutrients may compromise wound healing and inflammatory response.¹⁴ Interventions aimed at preventing and reducing obesity through the promotion of healthful eating and physical activity may be effective in improving periodontal health. Nutritional factors, such as nutrient intake and adiposity, may play an essential role in periodontal disease development and pathogenesis, and may affect the two main subsets of periodontal disease, gingivitis and periodontitis. Nutrients act as antioxidants that may moderate gingival inflammation. Promotion of

healthy nutrition and adequate physical activity are beneficial in preventing the progression of periodontal disease.

Conclusion

In conclusion, obesity has a negative effect on a person's overall health, which includes periodontal health. Promotion of healthy nutrition and adequate physical activity may help prevent or slow the progression of periodontal disease. Oral health care providers can have an impact on treatment outcomes by recognizing patients at risk and addressing these concerns. The devastating health consequences of obesity suggest further research is necessary regarding the relationship among obesity, periodontal disease, and other systemic and chronic disease. Studies might address the role oral health care practitioners' play in emphasizing prevention and lifestyle changes, in addition to the multiple medical problems and challenges that may influence dental treatment when treating patients suffering from obesity.

References

- Flegal KM, Carroll MD, Orden CL, Johnson CL. Prevalence and trends in obesity among US adults. JAMA. 2000; 288:1723-7.
- 2. Donatelle R. Health: the basics. 6th ed. Los Angeles, CA: Pearson Education; 2002. p. 268-289.
- Venes D. Taber's cyclopedic medical dictionary. 20th ed. Philadelphia: F.A. Davis; 2005. p. 46, 65, 182, 485, 579-583, 1254, 1504-1505, 2096, 2327.
- A Report of the Surgeion General [homepage on the Internet]. Physical activity and health [cited 2006 November 10]. Available from: <u>www.cdc.gov/nccdphp/sgr.htm</u>.
- 5. Meinz D. Nutrition, diet, and dentistry today. Gen Dent. 2004; 52(10): 387-9.
- Chapper A, Munch A, Schermann C, Piacentini, CC, Fasolo MTM. Obesity and periodontal disease in diabetic pregnant women. Braz Oral Res. 2005; 19(2): 83-7.
- Al-Zahrani MS, Bissada NF, Borawski EA. Obesity and periodontal disease in young, middle-aged, and older adults. J Perio. 2003; 74(5): 610-5.
- Goldie MP, Risbeck CA. The fattening of America and its effect on oral health. Dimensions of Dental Hygiene. 2006; 4(1): 16-7.
- 9. Nishimura F, Murayama Y. Periodontal inflammation and insulin resistance: lessons from obesity. J Dent Res. 2004; 80(8): 1690-4.
- National Heart, Lung, and Blood Institute [homepage on the Internet]. Guidelines on overweight and obesity [cited 2006 March 26]. Available from: <u>www.nhlbi.nih.gov/guidelines/obesity/e_txtbk/txgd/4142.htm</u>.
- Wood N, Johnson RB, Streckfus CF. Comparison of body composition and periodontal disease using nutritional assessment techniques. J Clin Perio. 2003; 30(4): 321-7.
- Nield-Gehrig JS, Willman DE. Foundations of periodontics for the dental hygienist. 1st ed. Baltimore (MA): Lippincott Williams & Wilkins; 2004. p. 36, 87-91, 103-106.
- 13. 13. Alabdulkarim M, Bissada N, Al-Zahrani M, Ficara A, Siegel B. (2005). Alveolar bone loss in obese subjects. J Int Acad Perio. 2005; 7(2): 34-8.
- Lundin M, Yucel-Lindberg T, Dahllof G, Marcus C, Modeer T. (2004). Correlation between TNF-α in gingival crevicular fluid and body mass index in obese subjects. <u>Acta Odontologica Scandinavica</u>. 2004; 62(10): 273-7.
- 15. Ritchie CS, Kinane DF. Nutrition, inflammation, and periodontal disease. Nutrition. 2003; 19(5): 475-6.
- Saito T, Shimazaki Y, Koga T, Tsuzuki M, Ohshima A. Relationship between upper body obesity and periodontitis. J Dent Res. 2001; 80(7): 1631-6.
- Saito T, Shimazaki Y, Kiyohara Y, Kato I, Kubo M, Iida M., Yamashita Y. Relationship between obesity, glucose tolerance, and periodontal disease in Japanese women: the hisayama study. J Perio Res. 2005; 40(8): 346-353.

- Cappelli DP. Diabesity and oral health implications. Comp Cont Educ Dent. 2003; 1(3): 164-5.
 Denco RJ. The three-way street. Scientific American Oral and Whole Body Health. 2006; 18-22