From Gumline to Waistline: The Link between Periodontal Disease and Obesity

Course Author(s): Maria L. Geisinger, DDS, MS; Mary Elizabeth Bush, BS
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Introduction
This continuing education course will review the current scientific evidence about the association between obesity status and periodontal disease and to aid in the clinical decision making to care for patients with periodontal disease and obesity in a dental setting.

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Overview
Periodontal diseases have been associated with numerous systemic diseases and conditions, including diabetes mellitus, rheumatoid arthritis, cardiovascular diseases, and obesity. Patients who are overweight or obese have an increased rate of periodontitis by 27% and 81%, respectively.\(^1\) While the mechanisms of these interactions are not fully elucidated, the elevated proinflammatory cytokine levels seen in obesity may directly or indirectly affect periodontal tissues.\(^2,3\) Obesity may also affect the host immune response and increase the risk for periodontal pathogens.\(^4-7\) Furthermore, periodontal inflammation and microbiota may affect nutrient absorption and processing from the gut and within adipose tissues.\(^8\)

This course seeks to improve the dental care provider's understanding of the interaction between periodontal disease and obesity as well as aid in the clinical decision making to care for patients with obesity and periodontal disease.

Learning Objectives
Upon completion of this course, the dental professional should be able to:
- Understand the current scientific literature about the association between periodontal health and obesity and discuss the interactions between these two conditions with patients.
- Be an active participant in an interdisciplinary team of health care providers treating patients with obesity and periodontal disease.
- Evaluate patients' risk factors and treatment needs based upon individualized patient needs and obesity status.
- Discuss with patients the common risk factors associated with periodontitis and obesity and be familiar with strategies to treat those risk factors.
- Discuss with interdisciplinary colleagues the importance of and effective methods for treatment of periodontal disease in patients who are obese.

Introduction
Overweight and obesity are defined by abnormal or excessive fat accumulation that represents a threat to general health.\(^9\) The American Medical Association (AMA) voted on June 18, 2013 to classify obesity as a disease based upon its comorbidities with other diseases and the endemic proportions of obesity in the United States.\(^10\) Obesity is an excess of body fat in proportion to lean body mass, to the extent that health is impaired.\(^11\) Current scientific understanding demonstrates that fat cells (adipocytes) play an active role in the regulation and modulation of inflammation and immunity which has led to obesity being labeled a chronic disease.\(^12\) The direct effects of obesity on systemic health include altered blood pressure, insulin resistance, dyslipidemia, and a chronic pro-inflammatory state.\(^13,14\) Comorbidities and indirect effects currently being investigated include Diabetes mellitus, coronary artery disease, stroke, respiratory disease, cancers, osteoarthritis, liver and gall bladder diseases.\(^14\) The morbidity and mortality associated with increasing obesity rates is of utmost public health concern, and successful efforts to reduce obesity rates will likely have significant benefits to the population.

Periodontitis is initiated by infectious agents resulting in tissue destruction caused by host inflammation within the supporting structures of the teeth.\(^15,16\) It has been shown to have an association with numerous systemic conditions in a bidirectional manner.\(^17\) The systemic inflammatory burden associated with periodontal diseases and the presence of putative periodontal pathogens that may affect patients' systemic health can alter the treatment recommendations for patients...
who have both periodontitis and obesity. Due to the number of individuals affected by both periodontal disease and obesity, the understanding of the interaction between periodontal disease and obesity is of utmost importance to the dental practitioner.

**Epidemiology of Obesity**

Obesity, defined as a body mass index (BMI) of ≥30 kg/m², is a major public health concern. More than one-third (36.5%) of U.S. adults are obese. Obesity and obesity related conditions, including heart disease, stroke, type 2 diabetes, etc. are some of the leading causes of preventable death, and the estimated annual medical costs related to obesity and its sequelae were $147 billion in 2008. Obesity is higher in middle-aged Americans and in some racial and ethnic minorities. Non-hispanic blacks have the highest rates of age-adjusted obesity (47.8%), followed by Hispanics (42.5%), non-Hispanic whites (32.6%), and non-Hispanic Asians (10.8%). Obesity is highest among individuals 40-59 years old (39.5%) when compared to rates in younger or older adults. Obesity is also associated with lower socio-economic status with lower income and educational levels correlated to higher rates of obesity. Given the high prevalence of obesity and the overlap of risk indicators between obesity and periodontal disease, it is critical dental care providers are aware of the interactions and able to adequately counsel patients and customize treatment protocols.

**Epidemiology and Etiology of Periodontal Disease**

Periodontitis is a chronic disease of the hard and soft tissue supporting the teeth caused by bacterial plaque resulting in progressive destruction of the periodontal ligament and alveolar bone. The disease typically has a slow to moderate rate of disease progression, but periods of accelerated attachment loss may be associated with local and/or systemic factors. Disease severity is classified as mild (1-2 mm), moderate (3-4), or severe (≥5 mm) based on the amount of clinical attachment loss (CAL). The prevalence of periodontitis has been estimated to be over 47% of U.S. adults, or 64.7 million individuals. Prevalence of periodontitis varied.

![Figure 1. Obesity in the United States in 2015](image-url)
two-fold between the lowest and the highest levels of socioeconomic status (Figure 2).\textsuperscript{30}

Disease progression of periodontitis has been categorized into subpopulations demonstrating rapid progression (10-15\% of disease cases), moderate progression (80\% of disease cases), and mild/no progression (5-10\% of disease cases).\textsuperscript{23,31,32} The prevalence distribution of periodontal disease severity and disease progression in treated and untreated populations\textsuperscript{33} suggests that host factors may play the larger role in disease progression after bacterial initiation.\textsuperscript{34-39}

**Proposed Mechanisms of Interaction between Periodontal Disease and Obesity**

Numerous recent studies have demonstrated associations between periodontal disease prevalence and/or progression and obesity status.\textsuperscript{40-44} A recent systematic review demonstrated odds ratio (OR) associations between periodontitis and body mass index (BMI) category obese of 1.81 (1.47, 2.30), between periodontitis and BMI category overweight of 1.23 (1.06, 1.51), and between periodontitis and obese and overweight 2.13 (1.40, 3.26).\textsuperscript{1} In younger age groups, but not older cohorts, overall and abdominal obesity demonstrated an association with prevalence of periodontal disease (when measured through attachment loss \(\geq 3\) mm, probing depth \(\geq 4\) mm, and alveolar bone loss).\textsuperscript{45,46} This may be related to increasing inflammatory burden from cumulative exposure to various diseases, conditions, and environmental exposures that occur with aging, the extraction of periodontally affected teeth in older adults, and/or the immunological effects of exposure to periodontal pathogens in a naive environment. Periodontal therapy in patients with metabolic syndrome—which is a clustering of risk factors including: atherogenic dyslipidemia, elevated blood pressure, obesity, elevated serum glucose levels, a prothrombotic state, and a proinflammatory state has been shown to reduce systemic markers of inflammation at 9 months post-treatment.\textsuperscript{47} The mechanism for an association between obesity and periodontitis has been suggested to involve the proinflammatory state that
exists in obese patients, which results in insulin resistance and oxidative stress; others have implicated additional factors which may govern this interaction (Figure 3).

**Common Inflammatory Burden**
Evaluation of longitudinal data in large-scale cross-sectional studies, including the National Health and Nutrition Examination Survey (NHANES), demonstrates obesity is correlated with deep periodontal pockets, independent of glucose tolerance status and BMI is positively correlated with severity of periodontal attachment loss in a relationship modulated by insulin resistance. Adipose tissue secretes a host of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). TNF-α and IL-6 are the main inducers of acute-phase hepatic protein production, including C-reactive protein (CRP). Additionally, both TNF-α and IL-6 have been shown to impair intracellular insulin signaling, which may lead to insulin resistance. Human plasma levels of TNF-α, IL-6, and CRP are associated with obesity and insulin resistance, as well as periodontal inflammation and destruction of periodontal tissues, and periodontal treatment has been demonstrated to reduced levels of circulating TNF-α. In persons with periodontal disease, bacterial pathogens, endotoxins, and inflammatory cytokines may systemically trigger an upregulated leukocytosis, synthesis of acute-phase proteins (e.g. CRP, Amyloid A), and enhanced lipid metabolism as well as increase serum cholesterol and triglyceride levels. This pathway may then lead to an increased risk of other systemic diseases. Additionally, the introduction of periodontal pathogens and their toxic by-products in a hyper-inflammatory environment, as is produced in obesity, may lead to a more robust immune-inflammatory response favoring progression of clinical disease and tissue destruction (Figure 4).

It is interesting to note previous investigations have suggested periodontal disease has a greater effect on overall systemic inflammation in patients with normal BMI and this influence decreases as BMI increases. This suggests that the inflammatory burden associated with periodontal disease may be overwhelmed by the larger influence of obesity-related inflammation in overweight and/or obese
patients. When C-reactive protein (CRP) was used as a measure of systemic inflammation, the association between elevated CRP levels associated with extensive periodontal disease was dependent upon the subject's BMI.\textsuperscript{73} Obesity has also been shown to negate the effects of nonsurgical periodontal treatment on systemic CRP levels in patients with at least mild chronic periodontitis.\textsuperscript{72} This may indicate the interaction between periodontal disease and obesity may have a BMI “ceiling” for the effect, and individual patient responses to therapy may differ based upon their obesity status.

**Common Microbial Interactions**

Obesity status and change in body mass have been associated with viral infection and distinct gut microbiota signatures in obese and lean patients.\textsuperscript{74-77} It is currently unclear if the GI bacterial microflora shift precede obesity or are a result of an obesogenic diet. However, in human and animal models, interventions such as roux-en-Y gastric bypass surgery have demonstrated an ability to shift this gut microbiota from one associated with obese to one associated with lean control subjects.\textsuperscript{78,79} In those animals with newly acquired lean gut microflora, altered overall nutrient acquisition and energy regulation has been seen.\textsuperscript{79,81} Furthermore, the immediate drop in hyperglycemia in obese subjects who have undergone bariatric surgery, prior to substantial weight loss indicates the surgical procedure itself, and perhaps the alterations in the gut microflora associated with it, have a beneficial effect on comorbid inflammatory conditions separate from those associated with weight loss.\textsuperscript{79,80,82} Recent studies have also shown increased numbers and proportions of pathogenic periodontal bacteria in obese patients\textsuperscript{83} and obesity-induced insulin resistance in gingival tissues.\textsuperscript{84}

The primary etiology of periodontal disease is bacterial plaque, in particular virulent bacterial “complexes” and/or “consortia” that are associated with active periodontal disease sites and periodontal disease progression.\textsuperscript{85-87} In addition, nonsurgical periodontal therapy has been shown to alter the oral microflora significantly.\textsuperscript{88,89} Due to the direct access of bacteria and bacterial products to the systemic circulation through the ulcerated periodontal pocket epithelium\textsuperscript{90} as well as the potential mechanism of bacterial-induced signaling and bacterial epitope recognition by epithelial and mucosal immune cells within the alimentary canal,\textsuperscript{8,91,92} oral bacteria may influence systemic energy metabolism, immune system stimulation, and gut permeability (Figure 5).
The Effect of Periodontal Therapy on Obesity Status

Periodontal therapy both surgical and nonsurgical has been shown to decrease circulating inflammatory cytokines in patients with periodontitis.\textsuperscript{93-95} Two recent meta-analyses concluded there was no difference in clinical periodontal parameters after periodontal therapy between obese and normal weight patients, indicating nonsurgical periodontal therapy can be effective in reducing clinical signs of periodontal disease in obese patients.\textsuperscript{96,97} Furthermore, obese subjects did demonstrate an overall reduction in systemic inflammatory burden after periodontal therapy, although most studies demonstrated overall significantly higher levels of pro-inflammatory cytokines at baseline in obese patients when compared with their non-obese counterparts.\textsuperscript{97} Elevated adipokine levels seen in obesity have been indicated in altered or impaired periodontal healing in obese patients\textsuperscript{98} but these meta-analyses indicate obese patients do receive benefit from periodontal therapy and this benefit may have a systemic effect as well. Furthermore, even in instances where clinical outcomes of therapy are similar, obese patients have been shown to have a different immunologic and cytokine responses which may affect the longevity of maintenance of the results of therapy.\textsuperscript{99} Based upon these findings, adjunctive use of antibiotics, decreased maintenance intervals, and/or periodontal surgical therapy may be advantageous in obese patients whose increased inflammatory burden may make controlling periodontal disease progression more challenging.

Clinical Decision Making for Treatment of Patients with Periodontal Disease and Obesity in a Dental Setting

Caring for patients with both obesity and periodontitis calls for careful evaluation, quantification and ongoing monitoring of existing periodontal inflammation, and also consultation with physician, in regards to obesity management. Obese patients may also require additional evaluations for comorbid diseases with oral presentations, such as diabetes mellitus and cardiovascular disease. Further, adjunctive therapies should be considered in patients who may demonstrate dampened response to periodontal therapy due to the overall inflammatory burden associated with obesity (Figure 6).

Summary

Both periodontal disease and obesity are multifactorial diseases that have been shown to increase host inflammatory burden. Furthermore, enteric bacteria, including periodontal pathogens, may play a role in nutrient absorption and metabolism. These diseases share common risk factors,
inflammatory pathways, and are associated with similar comorbid medical conditions. Periodontal disease and the inflammatory burden it creates should be discussed with patients and the use of advanced therapies to control the increased inflammatory load in patients with both obesity and periodontitis should be considered.

Comanagement of patients with obesity and periodontal disease with their treating physician will allow for screening of patients for additional comorbidities and can be used to further characterize the patients’ overall inflammatory burden (e.g., through CRP level testing) which can then inform treatment choices for oral disease treatment including personalization of the treatment plan and maintenance protocols. Counseling patients about the effects that their obesity may have on their oral condition is also an important step in encouraging patients to decrease their weight and control their overall inflammatory levels.

Figure 6. Risk Factors Associated with Obesity and Periodontitis to Consider during Periodontal Therapy.
Course Test Preview
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1. Obesity has been labeled a chronic disease by the American Medical Association (AMA) because fat cells (adipocytes) play an active role in the regulation and modulation of inflammation and immunity.
   a. True
   b. False

2. Periodontitis has both infectious and inflammatory components that ultimately result in clinical disease progression and tissue destruction. It has been shown to have an association with numerous systemic conditions in a bidirectional manner.
   a. Both statements are true.
   b. The first statement is true, the second statement is false.
   c. The first statement is false, the second statement is true.
   d. Both statements are false.

3. Obesity is defined by the American Medical Association (AMA) as a body mass index (BMI) of __________?
   a. ≥20 kg/m²
   b. ≥25 kg/m²
   c. ≥30 kg/m²
   d. ≥35 kg/m²

4. In the United States, ______ income and educational levels are correlated to higher rates of obesity.
   a. higher
   b. lower

5. A recent study indicates that over ____% of U.S. adults have periodontitis.
   a. 33
   b. 42
   c. 47
   d. 65

6. A recent systematic review demonstrated odds ratio (OR) associations between periodontitis and body mass index (BMI) category obese of _____.
   a. 0.78
   b. 1.15
   c. 1.5
   d. 1.81

7. In patients with both periodontal disease and obesity, the correlation between BMI and severity of periodontal attachment loss is __________.
   a. positive
   b. negative
   c. neutral
8. ___________ patients are more likely to demonstrate an association between overall and abdominal obesity and periodontal disease (when measured through attachment loss ≥3 mm, probing depth ≥4 mm, and alveolar bone loss).
   a. Younger
   b. Older
   c. Male
d. Female

9. Both clinical signs of periodontal inflammation and obesity levels are associated with increased plasma levels of which pro-inflammatory mediators?
   a. TNF-α
   b. IL-6
   c. C-reactive protein (CRP)
d. All of the above.

10. Patients who are obese demonstrate a ___________ effect of periodontal therapy on systemic levels of CRP than lean patients.
    a. increased
    b. decreased
    c. similar

11. Obese patients have shown ___________ numbers and proportions of pathogenic periodontal bacteria compared to lean patients.
    a. increased
    b. decreased
c. similar

12. Oral microbiota may affect obesity status by ___________.
    a. oral bacteria increase metabolic efficiency
    b. oral bacteria increase appetite
    c. oral bacteria redirect energy metabolism
d. All of the above.

13. While nonsurgical periodontal therapy has been shown to be effective in reducing clinical signs of periodontal disease in obese patients, most studies demonstrated overall ___________ levels of pro-inflammatory cytokines at baseline in obese patients when compared with their non-obese counterparts.
    a. higher
    b. lower
c. similar

14. Which of the following systemic conditions has been associated with obesity and periodontal disease?
    a. Diabetes Mellitus
    b. Cardiovascular Disease
c. Dislipidemia
d. All of the above.
15. Co-management of patients with obesity and periodontal disease with their treating physician should be a standard practice to allow for ___________.
   a. screening of patients for additional comorbidities
   b. characterization of the patient's overall inflammatory burden
   c. personalization of periodontal therapy and maintenance
   d. All of the above.
References


About the Authors

Maria L. Geisinger, DDS, MS

Dr. Geisinger is an Associate Professor at the University of Alabama at Birmingham (UAB) in the Department of Periodontology where she teaches a broad range of classes and serves as the Director of the Advanced Education in Periodontology Program. She received her Bachelor's of Science in Biology from Duke University graduating cum laude and completed her dental training at Columbia University College of Dental Medicine. She completed her Certificate in Periodontology and Master's of Clinical Science at the University of Texas Health Science Center in San Antonio. Dr. Geisinger is a Diplomate in the American Board of Periodontology. In her role at UAB, she is involved in clinical and translational research examining the interactions between periodontal diseases and systemic health. Her research focuses on periodontal-systemic interactions, periodontal regenerative therapies, implant dentistry, and educational technology. She is a member of the ADA, AAP, SAP, ADEA, AADR/IADR, AAWD, AAUW, and the President-elect of the AAPF.

Email: miagdds@uab.edu

Mary Elizabeth Bush, BS

Mary Elizabeth Bush is a senior dental student at the University of Alabama at Birmingham (UAB) School of Dentistry. Mary Elizabeth graduated magna cum laude from Washington and Lee University with a Bachelor's of Science degree in Biology. During her time in dental school, she has been an active member of many student organizations including SPEA, AAWD, and ASDA. Mary Elizabeth currently serves as the national Vice Chair of Organizational Affairs for the Student Professionalism and Ethics Association in Dentistry (SPEA). Her research in dental school has focused on the relationship between periodontal disease and obesity. After graduating dental school in June 2017, Mary Elizabeth will attend the Louisiana State University School of Dentistry for a residency in Periodontology.

Email: mebush17@uab.edu