

**The Relationship Between Periodontal Disease and Obesity:  
A 5 Year Review**

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Submitted to the Graduate Faculty of

University of Pittsburgh School of Dental Medicine in partial fulfillment

of the requirements for the degree of

Masters of Dental Science

University of Pittsburgh

2015

UNIVERSITY OF PITTSBURGH  
SCHOOL OF DENTAL MEDICINE

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## **The Relationship Between Periodontal Disease and Obesity: A 5 Year Review**

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**Purpose:** To expand upon the current evidence that there is an association between BMI and periodontal disease using a retrospective study design with a larger sample size, and thereby a greater statistical power than previously performed studies. We hypothesize that BMI is positively correlated with prevalence of periodontal disease.

**Materials and Methods:** Data from the electronic health records maintained by the University of Pittsburgh School of Dental Medicine from August 2008 to February 2014 was extracted for variables including age, gender, ethnicity, smoking history, diabetes, probing depths, height, and weight. Multivariate logistic regression was performed to determine the odds ratio for the association between periodontal disease status and all the other variables (BMI, age, ethnicity, gender, smoking status, and diabetes).

**Results:** A total of 27,052 subjects were included in the data set. Multivariate analysis of the data showed that the odds ratio of having periodontal disease with a BMI greater than or equal to 30 versus less than 30 was 1.22 when accounting for all the other confounding variables ( $p=0.013$ ). Furthermore, sex, age, ethnicity, and smoking were all associated with statistically significant odds ratio for development of periodontal disease when analyzed accounting for the other confounding variables ( $p<0.001$ ). Only diabetes did not show a statistically significant correlation with periodontal disease ( $p=0.394$ ).

**Conclusion:** Our results reaffirm that increased BMI is positively correlated with periodontal disease prevalence.

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## 1.0 INTRODUCTION

Periodontal disease is a chronic inflammatory condition that is widespread among the adult population. According to the Centers for Disease Control and Prevention, over 47% of the American population, or 64.7 million adults, have mild, moderate, or severe periodontal disease (Eke, 2012). Periodontal disease is a major cause of tooth loss, and treatment of the disease reduces tooth loss (Martin, 2011). A number of studies have shown that there are many different systemic risk factors for periodontal disease; these factors include tobacco use, obesity, diabetes, cardiovascular disease, osteoporosis, respiratory diseases, rheumatoid arthritis, certain cancers, erectile dysfunction, kidney disease and dementia (Otomo-Corgel, 2012, Bergström, 2006). Of these, obesity affects the largest number of adults in the United States (Centers for Disease Control and Prevention, 2004). By identifying patients at higher risk for periodontal disease, we can institute more effective screening and treatments. Additionally, if we correlate severity of periodontal disease with obesity, then those patients who are obese may be treated more intensively.

The World Health Organization (WHO) defines overweight and obese as body mass index (BMI) greater than or equal to 25 and 30, respectively (Obesity: Preventing and managing the global epidemic, 2000). More than one-third of the adult population 20 years and older are obese (Ogden, 2014). Increased BMI has been associated with inflammation in tissues throughout the body (Wellen, 2003). Specifically, it has been associated with periodontal

inflammation; there are proposed models that link specific cytokines and the inflammation of periodontitis (Genco 2005). Yet, the evidence for the association between increased BMI and periodontitis is still not as strong as for the other major periodontitis risk factors, namely tobacco use and diabetes. A systematic review conducted by Suvan, *et al.*, in 2011 detailed the current literature on the association between obesity and overweight and periodontitis (Suvan, 2011). They concluded that there is an association between increased BMI and periodontitis, although the magnitude is not clear. Included in this review was a prospective cohort study of 1504 patients specifically defining periodontal disease by attachment loss and showing the relationship between attachment loss and BMI (Kongstad, 2009).

We aim to expand upon the current evidence that there is an association between BMI and periodontal disease using a retrospective study design with a larger sample size, and thereby a greater statistical power. We hypothesize that BMI is positively correlated with prevalence of periodontal disease.

## **2.0 REVIEW OF THE LITERATURE**

### **2.1 PATHOLOGY AND DIAGNOSIS OF PERIODONTAL DISEASE**

Gingivitis is an inflammatory disease of the gingival tissues without clinical attachment loss (Treatment of plaque-induced gingivitis, chronic periodontitis, and other clinical conditions, 2001). It is primarily caused by bacterial plaque (The American Academy of Periodontology, 1999). This disease is only limited to the gingival tissues and does not cause destruction of connective tissue and bone. It is reversible destruction. After gingivitis has developed, two results are possible. If the gingivitis is treated, the oral tissues can heal. If it is not, continued inflammation can lead to periodontitis. The defining and distinct feature of periodontitis is clinical attachment loss, which is the destruction of the tooth and the supporting structures around the tooth: cementum, periodontal ligament, and alveolar bone (Parameter on chronic periodontitis with slight to moderate loss of periodontal support, 2000). Chronic periodontal disease is a chronic inflammatory condition caused by pathogenic bacteria and the host immune response.

Proper diagnosis of periodontal disease can only be established with a thorough medical and dental history paired with complete clinical and radiographic assessments. Clinical features can include redness, swelling, bleeding on probing, mobility, and/or suppuration.

Chronic periodontal disease is divided into two categories: localized and generalized. It is further divided in three categories depending on the severity of the disease: mild, moderate, and severe. In localized disease less than 30% of the teeth are affected, while in generalized disease 30% or more of the teeth are affected (Armitage, 1999). Clinical attachment loss distinguishes between mild, moderate, or severe periodontitis. Clinical attachment loss is the distance from the cemento-enamel junction to the base of the periodontal pocket (Armitage, 1995). Mild to moderate periodontitis is loss of less than one third of the attachment apparatus. Generally, mild periodontitis is clinical attachment loss of 1-2 mm and moderate periodontitis is clinical attachment loss of 3-4 mm. There may be tooth mobility and class I furcation defects in the molars. In severe periodontitis more than one third of the attachment apparatus is lost, usually when clinical attachment loss is equal to or greater than 5 mm. With severe periodontitis, there may be mobility and greater than class I furcation involvements. (Parameter on chronic periodontitis with advanced loss of periodontal support, 2000).

## **2.2 TREATMENT OF PERIODONTAL DISEASE**

Treatment of chronic periodontal disease begins with removing or reducing the etiologic factors to prevent the progression of destruction. This starts with reducing the bacterial load by teaching effective oral hygiene, removing the supra and subgingival calculus through scaling and root planing, extracting hopeless teeth, recontouring restorations, restoring overhanging restorations, and adjusting occlusion (Parameter on chronic periodontitis with advanced loss of periodontal support, 2000). All of these initial phase procedures decrease the pathogenic bacterial load, decrease destruction of alveolar bone, and reduce the inflammatory process. The second

component of periodontal therapy includes either resective or regenerative surgery. The goal of resective surgery is to reduce the periodontal pocket to allow access to clean the gingival tissue and teeth more effectively. The goal of regenerative surgery is to bring back the periodontal apparatus that has been lost due to the disease process. The final and most important part of chronic periodontal disease therapy is periodontal maintenance to make sure that the disease stays well controlled and plaque levels stay low (Ramfjord, 1993). Prevention is the best and most effective treatment of plaque induced periodontal disease.

### **2.3 RISK FACTORS FOR PERIODONTAL DISEASE**

Preventing periodontal disease is accomplished through assessment and modification of risk factors. A risk factor is a process that is associated with development of a disease. Importantly, association does not equate to causation, although causation is frequently implied. Risk factors are practically divided into those that are modifiable and those that are not. The nonmodifiable risk factors are important in identifying those individuals at higher risk for development of periodontal disease, but do not necessarily affect treatment; conversely, if present, changing modifiable risk factors is the cornerstone of any treatment plan. There are a few well established modifiable risk factors for periodontitis, and numerous others that are under active research.

The most important and best defined modifiable risk factor is tobacco use. Development of periodontal disease and its severity are positively correlated with increasing tobacco use (Grossi, 1995). Furthermore, tobacco use decreases the effectiveness of periodontal treatments (Bostrom, 1998). While studies show an association between the periodontal disease and tobacco use, use of the Hill criteria confirms causality and leads to the conclusion that tobacco

use causes periodontal disease (Hill, 1965). The other important modifiable risk factor for periodontitis is diabetes (Kinane, 1997). Current literature definitely shows an association between the two, but fulfillment of the Hill criteria is not entirely possible. Evidence for a bi-directional relationship suggests that diabetes can worsen periodontitis, and that periodontitis can worsen diabetes (Taylor, 2001). Regardless, modifying these risk factors through tobacco use cessation and diabetic control are crucial steps in the treatment of these patient populations.

#### **2.4 BODY MASS INDEX AS A PERIODONTAL DISEASE RISK FACTOR**

A modifiable risk factor which may have a greater impact on the oral health of a larger percentage of the global population than either tobacco use or diabetes is increased body mass. The CDC defines overweight and obesity as weights that greater than is healthy for certain heights (Defining Overweight and Obesity, 2015). In order to simplify application of the aforementioned definition, the use of a standardized index has been adopted. The relationship of weight and height is shown using the body mass index. The formula,  $\text{weight (kg)} / [\text{height (m)}]^2 = \text{BMI}$ , creates a number that is applicable across all medical fields (Garrow, 1985). It is simple, easily measurable, and relatively accurate in predicting body adiposity. A defense of the use of BMI in defining adiposity is beyond the scope of this paper, but its widespread acceptance and use in both medical practice and medical research affirms its position as the current global standard in this respect.

Overweight is defined as BMI of greater than or equal to 25 and less than 30. Obese is defined as BMI greater than or equal to 30. Worldwide obesity has doubled since 1980. More than 1.9 billion adults are overweight and over 600 million of those are obese (Obesity and

overweight, 2015). Childhood obesity and overweight are also dramatically prevalent, especially within the United States where 17% of children are obese (Ogden, 2014). As these children age, they will contribute to an increase in the overall prevalence of adulthood obesity and overweight.

Increased BMI has been associated with numerous diseases and other health problems. Coronary heart disease, type 2 diabetes, cancer, hypertension, dyslipidemia, stroke, liver and gallbladder disease, sleep apnea and respiratory disease, osteoarthritis, and gynecological disease are just some of the major disease processes that are strongly associated with increased BMI (Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults--The Evidence Report, 1998). Increased BMI has been strongly suggested as a causative factor in many of these diseases.

## **2.5 PRIOR RESEARCH ASSOCIATING BODY MASS INDEX AND PERIODONTAL DISEASE**

Perlstein and Bissada were the first to publish a study suggesting that a relationship exists between obesity and periodontal disease. By causing chronic periodontitis in groups of rats who were either obese or normal and measuring alveolar bone resorption, they showed that obese rats had more bone resorption. Their landmark study germinated an idea that would be the subject of continued research for the coming decades (Perlstein, 1977).

The National Health and Nutrition Examination Survey (NHANES III) conducted from 1988 to 1994 provided a large database of patients from which numerous studies were published. The results of the survey have been used to determine risk factors and associations between diseases. Great epidemiologic value has been obtained from the survey and national standards

for vital measurements such as blood pressure and weight arose from the gathered data. The survey included a dental periodontal examination of all patients over the age of 13. Periodontal measurements of attachment loss and pocket depth were included. The statistical power of the survey was tremendous due to the large number of patients (19,810). Al-Zahrani *et al.* analyzed the data to determine if obesity and periodontal disease were associated. Interestingly, they found a significant association between the two diseases only among young adults aged 18 to 34. An adjusted odds ratio of 1.76 was found for prevalence of periodontal disease in obese patients when accounting for various confounding factors. They were unable to demonstrate an association in adults older than 34. Lack of a significant association in older adults was postulated to be multifactorial. Early onset obesity may be more harmful than obesity that appears in adulthood and this could have played a role in the observed results. The influence of obesity on periodontal disease may have been masked by a dilutional effect wherein other risk factors such as age had a stronger influence on the prevalence of periodontal disease in older patients. Finally, due to the cross sectional nature of the study, cohort effects of different lifestyles in different time periods may have variably influenced the participant health in inextricable ways. Regardless of the reason, the overall result of the work of Al-Zahrani *et al.* was to demonstrate a definite association between periodontal disease and obesity (Al-Zahrani, 2003). Numerous additional studies have since corroborated these findings and linked obesity and periodontal disease (Saito, 1998, Saito, 2001, Saito, 2005, Sheiham, 2002, Nishida, 2005, Wood, 2003, DallaVecchia, 2005, Morita, 2011).

## 2.6 PATHOGENESIS OF THE ASSOCIATION BETWEEN BODY MASS INDEX AND PERIODONTAL DISEASE

The pathogenesis of the association between body mass index and periodontal disease is an area of active research. Obesity and overweight have definite systemic effects as is evident by their association with the numerous aforementioned medical diseases. The excess body fat in these patients results in measurable changes in levels of circulating cytokines which have far reaching effects on distant tissues and organs in the body.

One of the most important and ubiquitous pro-inflammatory cytokines is TNF- $\alpha$ . The acute phase reaction is characterized by an increase in circulating TNF- $\alpha$ . Macrophages are the primary producers of the molecule, but many other cell types can also release it. The adipocytes of obese patients have been shown to have increased TNF- $\alpha$  production; similarly, weight loss in obese individuals has been correlated with decreased TNF- $\alpha$  in adipose tissue (Hotamisligil, 1995). Once produced, TNF- $\alpha$  reaches cell surface receptors and has a catabolic effect through a signaling cascade, which in ultimately results in gene regulation and the activation of collagenases and other enzymes that break down the extracellular matrix (Brenner, 1989). TNF- $\alpha$  also increases osteoclastogenesis and differentiation from precursor cells, a pivotal step in inflammatory osteolysis (Kobayashi, 2000). Both extracellular matrix and bony destruction are key components of periodontitis and it is clear that increased TNF- $\alpha$  can promote both processes. Given the complexities of molecular interaction in the human body, definite proof of the pathway from increased adiposity to periodontitis has not been procured, but the basis for such a hypothesis has been affirmed (Nishimura, 2003).

The aforementioned actions of TNF- $\alpha$  occur directly at the target site, the periodontium. Other research has suggested that TNF- $\alpha$  may act as more of an intermediary step in the pathway

towards periodontal inflammation. Work by Genco *et al.* proposes that the inflammatory cytokines such as TNF- $\alpha$  produced in obese individuals increases insulin resistance and results in diabetes. Subsequently, diabetes and elevated blood sugar cause the production of advanced glycation end products; these products go on to increase leukocyte inflammatory cytokine release at the local level, thereby producing periodontitis (Genco, 2005).

### 3.0 METHODOLOGY

This study was a retrospective chart review taking place at the University of Pittsburgh School of Dental Medicine Periodontics Department. Initial exempt approval from its Institutional Review Board was given in December 2008, and then data analysis began in March 2014. The axiUm [dental management software, Exan Group, Canada] electronic health records maintained by the University of Pittsburgh School of Dental Medicine from August 2008 to February 2014 was used as our data set. Only patient records with a reported height, reported weight, and complete periodontal charting were extracted for the study. The variables of age, gender, ethnicity, smoking status, and diabetic status at the initial visit were also extracted for the patients who had complete charts to be included in the study. Patient charts were de-identified by assigning each chart a number.

For the periodontal assessments, the comprehensive periodontal exams were extracted from the charts. Periodontal condition was determined by the probing depths. All measurements in the charts were taken by dentals students in their 3<sup>rd</sup> or 4<sup>th</sup> year of dental school or periodontal graduate program residents. Dental student measurements were confirmed by periodontal faculty or residents. Overall probing depth averaging greater than or equal to 4mm was considered positive for periodontal disease and probing depth averaging less than 4mm was considered negative for periodontal disease.

Body mass index (BMI) is a ratio of body weight to body height and was calculated based on reported height and weight measurements extracted. The standard calculation using the non-metric conversion formula  $[(\text{weight (pounds)}/\text{height (inches)}^2) \times 703]$  was used to calculate the BMI. Our definition of obesity is based on BMI. Patients were then categorized into underweight, normal weight, overweight, or obese based on the World Health Organization criteria of BMI <18, <25, <30, and >30 respectively.

The variables age, gender, ethnicity, smoking status, and diabetic status were extracted and categorized for each patient entry. Age at the time of the initial exam was categorized into less than 18, 18 to 35, 35 to 50, 50 to 75, and greater than 75. Gender was categorized into male, female, transgender, and unknown. Ethnicity was categorized into African American, American Indian, Asian, Caucasian, Hispanic, or other. Smoking status was categorized into yes (currently smoking and former smoker) and no (never smoked). History of diabetes was recorded.

Data analysis was performed using a statistical software package, Stata (Statacorp, 4905 Lakeway Drive, College Station, Texas). Multivariate logistic regression was performed to determine the odds ratio for the association between periodontal disease status and all the other variables (BMI, age, ethnicity, gender, smoking status, and diabetes).

## 4.0 RESULTS

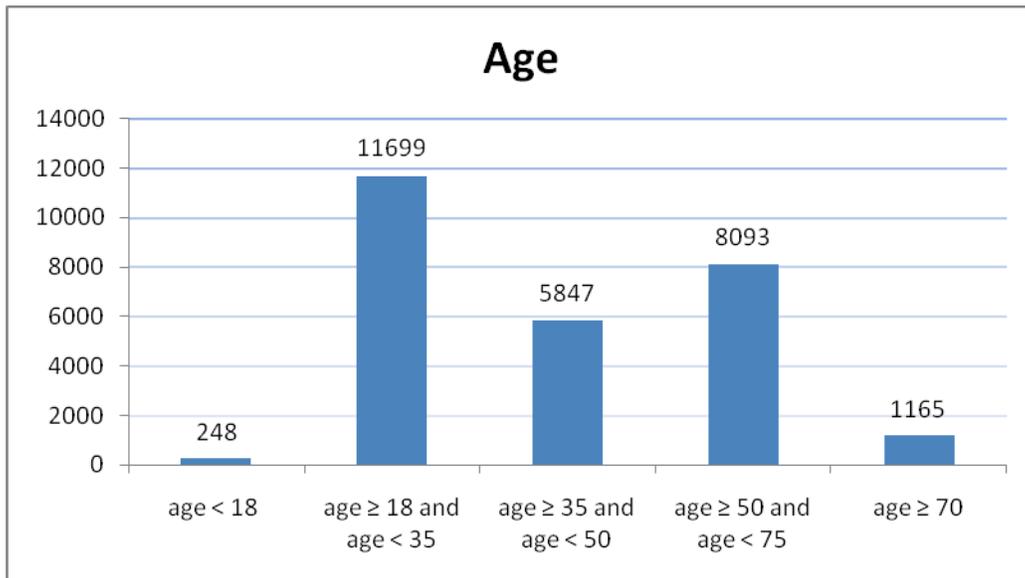
Data for 27,052 subjects were extracted from the electronic health records at the University of Pittsburgh School of Dental Medicine from August 2008 to February 2014. Demographics of the study population are shown in the subsequent Figures 1 through 7.

There was a skewed age distribution with more young patients in the population. The 18 to 35 age group included 43% of the population. Twenty-one percent were between 35 and 50, 30% were between 50 and 75, and 4% were over 70. Only 1% were less than 18. There was a mostly even distribution of gender with 48% male and 52% female. Most patients were either Caucasian (48%) or African American (24%). A large percentage of patients (26%) were labeled as “Other” due to patient preference not to respond to the question, lack of data, or mixed ethnicity. Eight percent of the study population was diabetic, which amounted to 2330 subjects. Approximately half of the patients smoked and half did not. Over one quarter of the patients (29%) were positive for periodontal disease.

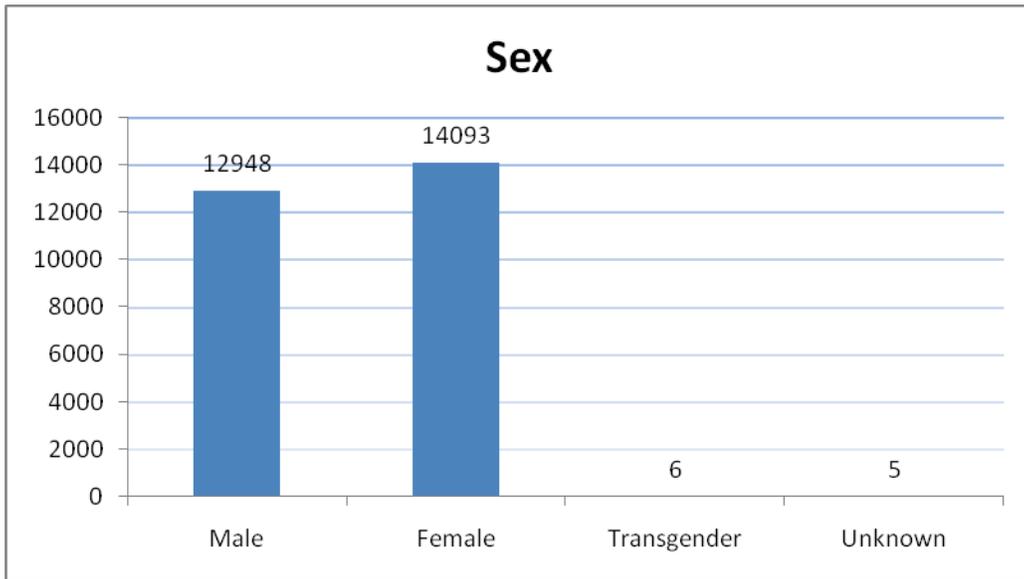
Most of the subjects fell between BMI of 19 and 40. Four percent had a BMI less than 19, 31% had a BMI between 19 and 25, 29% had a BMI between 25 and 30, 29% had a BMI between 30 and 40, and 8% had a BMI greater than 40.

Multivariate analysis of the data (Figure 8) showed that the odds ratio of having periodontal disease with a BMI greater than or equal to 30 versus less than 30 was 1.22 when accounting for all the other confounding variables ( $p=0.013$ ). Furthermore, sex, age, ethnicity,

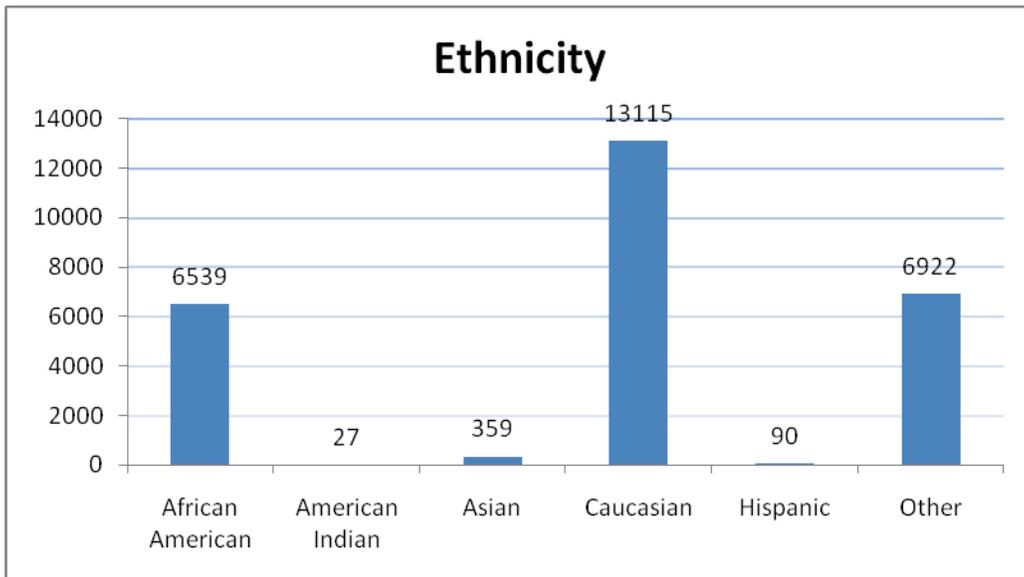
and smoking were all associated with statistically significant odds ratio for development of periodontal disease when analyzed accounting for the other confounding variables ( $p < 0.001$ ). Only diabetes did not show a statistically significant correlation with periodontal disease ( $p = 0.394$ ).



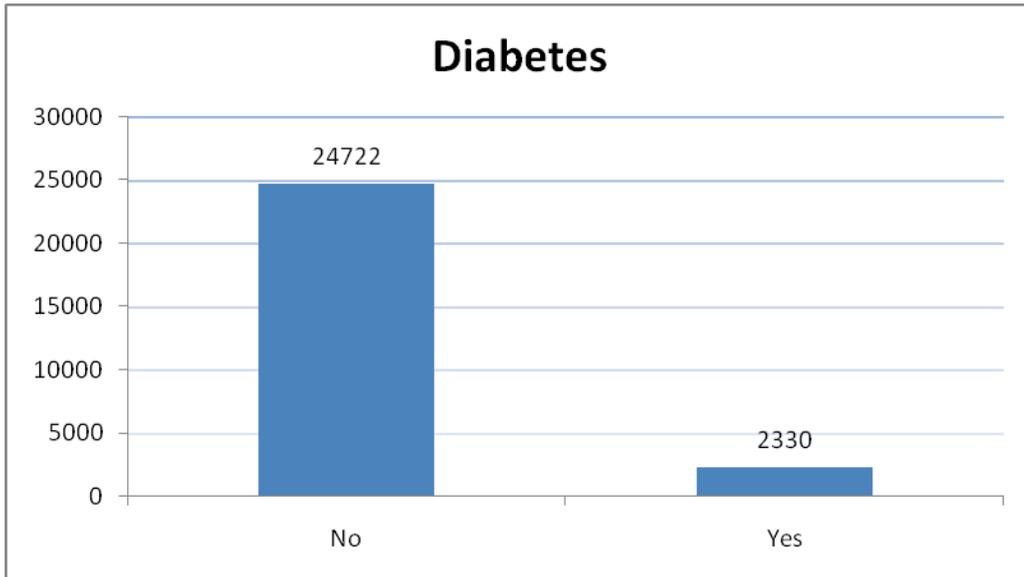
**Figure 1. Age Distribution**



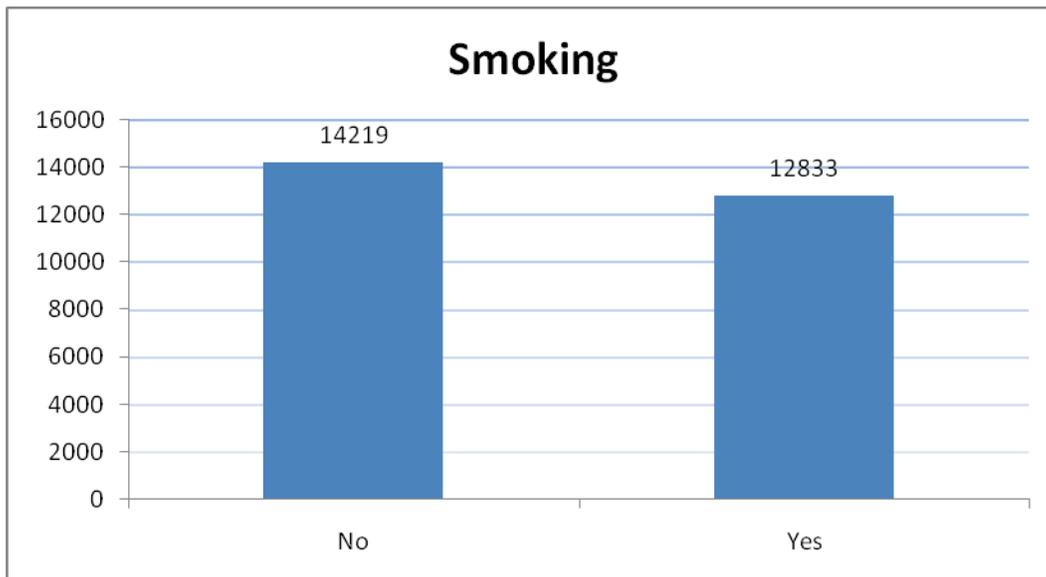
**Figure 2. Sex Distribution**



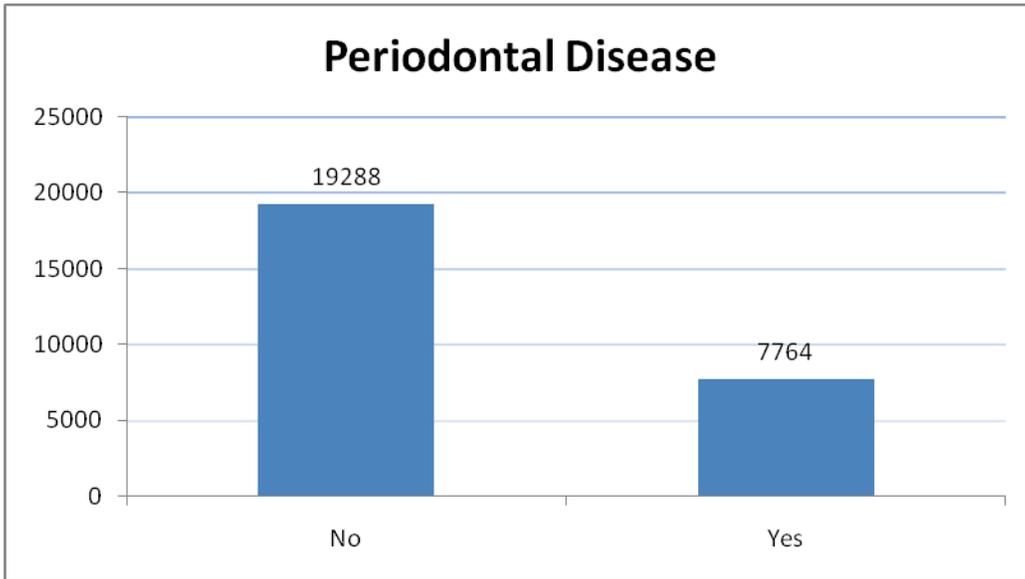
**Figure 3. Ethnicity Distribution**



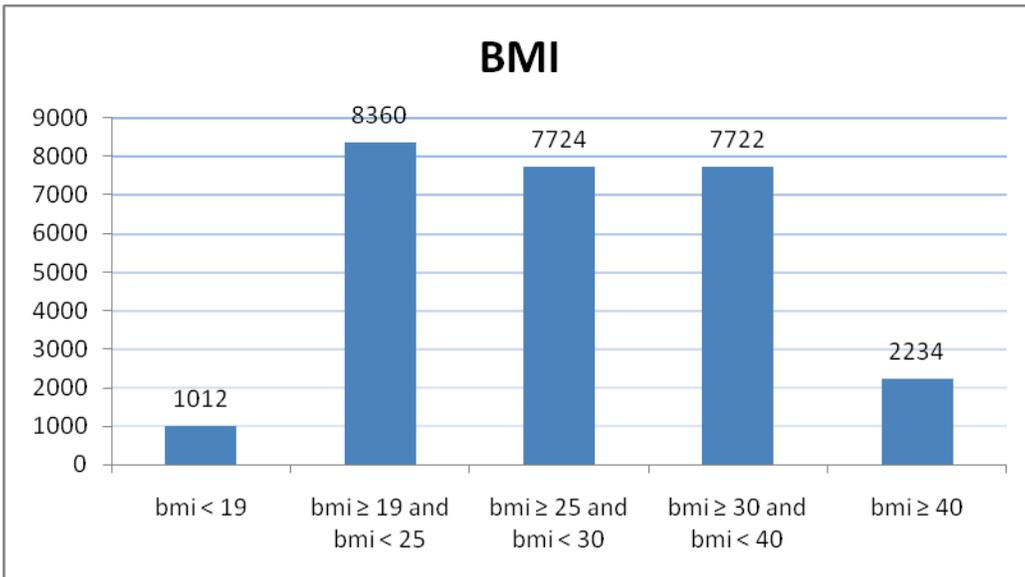
**Figure 4. Diabetes Distribution**



**Figure 5. Smoking Distribution**



**Figure 6. Periodontal Disease Distribution**



**Figure 7. Body Mass Index Distribution**

<b>Independent variables in the model</b>	<b>P-value of individual Z test</b>	<b>Odds Ratio</b>	<b>Overall P-value of Chi-square test</b>
Sex	<0.01	0.37	<0.01
Age	<0.01	2.38	<0.01
Ethnicity	<0.01	1.41	<0.01
Diabetes	<0.01	2.74	<0.01
Smoking	<0.01	2.48	<0.01
BMI	<0.01	1.61	<0.01
for Sex when the other variables are all adjusted	<0.001	0.41	<0.01
for Age when the other variables are all adjusted	<0.001	2.06	
for Ethnicity when the other variables are all adjusted	<0.001	1.43	
for Diabetes when the other variables are all adjusted	0.394	1.31	
for Smoking when the other variables are all adjusted	<0.001	1.93	
for BMI when the other variables are all adjusted	0.013	1.22	

**Figure 8. Univariate and Multivariate Analysis Results**

## 5.0 DISCUSSION

Results of our study confirm that there is an association between the prevalence of increased BMI and the prevalence of periodontal disease with an odds ratio of 1.22 for BMI greater than or equal to 30 versus BMI less than 30. All the other variables, except diabetes, were also significantly correlated with periodontal disease prevalence. Surprisingly, diabetes was not significantly associated with periodontal disease prevalence.

While we have shown the association between BMI and periodontal disease, the next important step is to explore causation, which may be best done using Hill's criteria. First, the strength and consistency of the association has been established in our study and in multiple prior studies. Second, we have considered many alternate explanations of the association by accounting for confounding variables in our study (age, sex, ethnicity, tobacco use, and diabetes). Third, it is fully plausible that obesity is not just associated with periodontitis, but that it actually predisposes to its development. Fourth, establishing temporality would be possible if a longitudinal study were to show that obese people have a higher incidence, not just prevalence, of periodontitis. Clinical experience suggests that that would be the case and future research could easily explore this idea. Overall, the idea that obesity is at least a contributing or predisposing factor to the development of periodontitis is coherent.

By building upon the existing foundation of research on this topic, we have added to the list of diseases with which obesity is associated. The global healthcare community now has

more incentive to combat the obesity epidemic because obesity is now associated with one more disease in one more organ system. The changes taken to reduce the prevalence of obesity should occur at both a policy and a patient level. Policy level changes would be such things as inclusion of risk of periodontal disease in obesity education materials. Patient level changes occur at every visit to a dental health professional. Dentists and dental hygienists are well suited to effect change through patient education. Weight loss education and reinforcement through motivational interviewing techniques have been shown to be effective in reducing patient weight (Armstrong, 2001). At each patient visit simple questions about patient weight loss goals and reminders that obesity is associated with periodontal disease should become a part of treatment planning.

Furthermore, obese patients may need more intense periodontal treatment and screening. By being aware of the increased prevalence of periodontitis in obese patients, dentists can be more sensitive in looking for early signs of clinical attachment loss. More frequent formal hygiene appointments and patient hygiene education could be instituted for obese patients with early signs. Just as smoking cessation has been shown to result in more effective periodontal treatment, so too could it be postulated that weight loss would have the same effect and obese patients could be educated to lose weight to improve the effectiveness of their dental treatments (Heasman, 2006).

The main limitation of the study is that height and weight were self reported by patients. These were not routinely measured at patient visits, but rather reported by the patients. However, past epidemiologic work has shown that self reported height and weight can be accurate in assessing the prevalence of obesity and overweight in a study population (Bowring, 2012). Other limitations to this study include the inability to account for the possible

confounding variables of socioeconomic status and diabetic control (by measuring HbA1C levels) because these were not gathered in the patient charts. Also, the retrospective study design meant that we were dependent on periodontal measurements taken by a diverse group of dental students. Patients who presented to the school already edentulous were excluded from the study because we did not have baseline data and clinical attachment loss levels. This is a limitation in the study because those patients could have lost all of their teeth due to severe periodontitis, which if it were the case would exclude a large number of the most severely diseased population.

## **6.0 CONCLUSION**

Our results reaffirm that increased BMI is positively correlated with periodontal disease prevalence. We hope this study will increase awareness among medical professionals so that there is increased patient education about the oral health risks of overweight and obesity, and that there is earlier recognition and treatment of periodontal disease in patients who are overweight and obese.

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