**Introduction:**

 Oral health status can be linked back to one’s overall health and wellness. One of the most concerning and current epidemics affecting a majority of the population is obesity. Obesity has more than doubled in the last twenty-five years, and has become an epidemic not only for the American adult population, but also for American children. In comparison to obesity, periodontal disease is also a chronic, highly prevalent, multifactorial health problem affecting the American population. As well as obesity, periodontal disease is beginning to be seen at a younger age and more frequently by dental professionals. The possible casual relationship between obesity and periodontal disease and potential underlying biological mechanisms are yet to be established, despite the abundant amount of research done in hopes to link the two epidemics. The purpose of this paper is to explore whether obesity and periodontal disease can be associated with each other, and if so, how dental and medical professionals can work together to educate and prevent the continual rise of obesity and periodontal disease.

**Background:**

 To begin, obesity is defined as a total body mass index greater than or equal to 30.0 kg/m2. Obesity stems from an energy imbalance, specifically, “if energy intake exceeds energy expenditure,” weight gain will occur (Selassie & Sinha, 2011, pp. 4). In addition to energy imbalance, several genetic and environmental factors may increase one’s susceptibility to obesity. One of the most influential environmental factors facilitating the development of obesity is increasing one’s consumption of food with high fat and sugar content, while expending less energy. Although the body does posses some mechanisms to maintain energy balance, such as metabolism, it has been proven that certain predisposed genetic factors increase one’s susceptibility to obesity.

 Several studies have shown that a child’s BMI is correlated to their biological parents’ BMI, heritability for obesity ranges from 50% to 90%, and only a few single human genes have been associated with obesity. The most common gene linked to obesity is the melanocortin-4 receptor gene. Melanocortin-4 is “present in 4% of obese individuals, suppressing food intake, with its subsequent deficiency leading to severe obesity” (Selassie & Sinha, 2011). In addition, leptin, a gene “responsible for the regulation of body weight and adipose stores” correlates with obesity (Selassie & Sinha, 2011). Children with deficiencies of this gene portray severe obesity; weight loss will occur with treatment of leptin deficiency. Although obesity has been linked back to monogenic causes, a single metabolic defect is not the most significant cause; obesity is due to multiple influences.

 In addition to genetic factors, environmental factors directly influence energy intake and weight gain, one major change is an increase in portion size throughout America. Currently, portion sizes are ranging from “two to eight times the standard serving sizes recommended by the USDA and FDA,” which leads to an increase in daily calories consumption and consequent weight gain (Selassie & Sinha, 2011). Furthermore, those who consumed sugar-sweetened beverages showed a positive association with obesity as well. Continuously, foods containing high fat and sugar content are often the least expensive, which creates an inverse relationship between one’s economic status and obesity.

 In comparison, periodontitis is an inflammation around the tooth, where the gums pull away from the teeth and form pockets that become infected. Similarly to obesity, periodontal disease susceptibility factors can be related back to genes. Specifically, cytokines, such as “IL1A, IL1B, IL10, and IL6, which mediate the inflammatory process during periodontal disease (Scapoli et al., 2012, pp. 197). Furthermore, these cytokines have a role in “B-cell activation, proliferation and differentiation, and are the majority of infiltrating cells in advanced periodontitis lesions” (Scapoli et al., 2012).

 Additionally, periodontal disease is similar to obesity because it is also influenced by changing or environmental risk factors. One of the biggest risk factors for periodontal disease is smoking. According to dentists, Sherwin, Nguyen, Friedman, and Wolff (2013), smokers have a higher prevalence of periodontal disease, a higher probing pocket depth greater than or equal to 4mm, more destruction of the periodontal bone, and are less likely to respond to treatment. Furthermore, smoking can also affect the genetic cytokines involved with periodontal disease. Sherwin et al. (2013), found that smokers have higher levels of IL-1 and IL-8, whereas nonsmokers have higher levels of IL-4 but a lower amount of IL-8.

**Dental Approach:**

 Dental professionals have recognized that oral health and the rate of success for dental care is impacted by one’s overall systemic health problems. For example, diabetes, uncontrolled diabetes increases one’s risk for periodontal disease and if uncontrolled, there is little if any positive response to dental therapy. Therefore, dental professionals have been researching whether obesity is a risk factor for periodontal disease and how it affects treatment. In a study done by researcher, Levine (2012), evidence from multiple literature reviews showed a higher prevalence of periodontal disease in overweight and obese groups. Although many studies have shown a correlation between obesity and periodontal disease, there have been studies that found “no significant association between BMI and deepened periodontal pockets” (Levine, R., 2012, p.455). Therefore, more research on the relationship between obesity and periodontal disease needs to be conducted.

 Furthermore, evidence has shown that “adipose tissue secretes chemical mediators of inflammation, including cytokines and hormones,” therefore, with an increase in adipose tissue there will be an increased secretion of cytokines, an inflammation mediator, which can modify the response of periodontal tissues (Levine, R., 2012, P. 455). Additionally, evidence shows a link between “obesity, abnormal fat metabolism, hypertension, insulin resistance, high plasma fibrogen, and elevated C-reactive protein” to periodontal disease, strengthening the argument that certain obesity-related systemic illnesses are related to periodontal disease (Levine, R., 2012, P. 455).

 In addition, a study on the role of diet conducted by RDH, Dianne Watterson (2013), explored the different Western diets and how they influenced periodontal disease. In particular, studies have been conducted on levels of Vitamin C, showing “the greatest clinical effect in smokers who took the lowest levels of Vitamin C” (Watterson, 2013, p.79). Additionally, studies have shown that “a low dietary intake of calcium is associated with severe periodontal attachment loss, and prevalence of periodontal disease decreases with high intake of dairy products,” deficiencies in calcium and Vitamin D also resulted in increased inflammation (Watterson, 2013). Furthermore, the “consumption of at least 55 g of lactic-acid-containing foods per day significantly lowers the prevalence of periodontal disease,” in comparison to no consumption (Watterson, 2013). In a Hisayama study, it was found that “oral lactobacilli suppress the growth of periodontal pathogens in vitro” (Watterson, 2013). When lactic acid is produced by oral lactobacilli through carbohydrate fermentation, it creates a low pH, which could inhibit the growth of anaerobic bacteria. Therefore, more research needs to be done to determine if regularly consuming lactic acid foods can control the overgrowth of periodontal pathogens.

**Medical Approach:**

 Unfortunately, most research conducted and analyzed regarding the relationship between obesity and periodontal disease has been done by researchers in the dental profession. There are multiple links and consistencies between both obesity and periodontal disease risk factors, and although the research is skewed, most leans toward there being a direct link between the two major diseases. Medical professionals should work interprofessionally along side dental professionals to help determine the link between obesity and periodontal disease so that both fields are able to better educate their patient’s on how to maintain a healthy body. Obesity is becoming more prevalent in younger children, and periodontal disease has begun to show up in younger ages as well, therefore, research should be focused on young Americans and how to help them understand overall health does affect oral health.

**Conclusion:**

 In conclusion, numerous studies have shown that obesity can be associated with an increased and severity of periodontal disease. One’s diet plays an extremely important role in the health and wellness of the overall body, as well as one’s oral health. Research has been found that obesity and periodontal disease can be linked together by common pathophysiology. It has been proven that the “adverse effected of obesity on the periodontium are from inflammatory cytokine and adipokines, such as TNF and adiponectin or leptin” (Karabey, Cifcibasi, and Cintan, 2014). Subsequently, the fibroblasts that “promote synthesis of degrading enzymes and stimulation of osteoclasts that activate bone resportion” are exacerbated by high concentrations of TNF, which is present in those who suffer from obesity (Karabev et al., 2014). Therefore, medial and dental professionals need to work together to educate their patients on how the two are linked and set goals for their patients to better their oral health.