Section 1
Body-Mouth Connection: Relevant Pathologies Affecting Dental Treatment, Guidelines, Prevention, and Necessary Precautions

Chapter 1 Body Weight, Diet, and Periodontitis

Jean-Pierre Dibart, MD

BODY WEIGHT

Introduction

The body mass index relates body weight to height. Body mass index, or BMI, is defined as the weight in kilograms divided by the height in meters squared. Obesity is defined as a body mass index greater than 30 kg/m^2; BMI between 25 kg/m^2 and 30 kg/m^2 defines overweight people, the normal weight being between 19 kg/m^2 and 25 kg/m^2. Obesity is a chronic disease with many important medical complications. The main cause of obesity is an imbalance between energy intake and energy expenditure.

The necessary treatment includes

- a calorie-restricted diet,
- increased physical activity, and
- nutritional modifications, with reduction of fat and sugar intake

The prevalence of obesity has increased in Western countries. It is a metabolic disease that predisposes to many medical complications such as cardiovascular disease, cancer, arthrosis, and diabetes, and it has also been implicated as a risk factor for chronic health conditions such as periodontitis. Obesity is associated with periodontal disease because the adipose tissues secrete some cytokines and hormones that are involved in inflammatory process. A high body mass index is associated with a systemic low-grade inflammatory state. Tumor necrosis factor-α, a proinflammatory cytokine, is produced in adipose tissues and is responsible for lowered insulin sensitivity, called insulin resistance which is responsible for elevated plasma glucose levels.

Periodontitis is characterized by alveolar bone loss, which is the consequence of bone resorption by the osteoclasts. Bone-forming cells (osteoblasts) and bone-resorbing cells (osteoclasts) are under hormonal control; the bone formation is negatively regulated by the hormone leptin, produced from adipocytes.

Health education should encourage better nutritional habits to reach normal weight and prevent obesity, and also to promote better oral hygiene to prevent periodontal disease (Alabdulkarim et al. 2005; Dalla Vecchia et al. 2005; Ekuni et al. 2008; Khader et al. 2009; Lalla et al. 2006; Linden et al. 2007; Nishida et al. 2005; Reeves et al. 2006; Saito et al. 2001; Saito et al. 2005; Wood, Johnson, and Streckfus 2003; Ylostalo et al. 2008).

Body Mass Index

High body mass index is a risk factor for periodontitis. There is a 16% increased risk for periodontitis per 1 kg/m^2 of increased body mass index. Body mass index is also significantly associated with the community periodontal index score (Ekuni et al. 2008). Total body weight is associated with periodontitis. Adolescents aged 17 to 21 years old have a 1.06 times increased risk for periodontal disease per 1 kg increase in body weight (Reeves et al. 2006). There is a significant correlation between body mass index and periodontitis, with a dose-response relationship (Nishida et al. 2005). Obesity is a risk factor for periodontitis; there is an association between high body weight and periodontal infection (Ylostalo et al. 2008). High body mass index is significantly associated with periodontitis, with an odds ratio of 2.9 (Khader et al. 2009). Obesity with a body mass index greater than 30 kg/m^2 is significantly associated with periodontitis, with an odds ratio of 1.77 (Linden et al. 2007).

Obese patients are 1.86 times more likely to present periodontitis according to the following groups:

- For patients older than 40 years of age, the odds ratio is 2.67.
- For females, the odds ratio is 3.14.
- For nonsmokers, the odds ratio is 3.36 (Alabdulkarim et al. 2005).

There is a positive correlation between body mass index and periodontitis, with a significantly higher prevalence in females. Obese females are significantly (2.1 times) more likely to have

Practical Osseous Surgery in Periodontics and Implant Dentistry, First Edition. Edited by Serge Dibart, Jean-Pierre Dibart.

periodontitis (Dalla Vecchia et al. 2005). Obesity is also associated with deep probing pockets. High body mass index and body fat are significantly associated with the highest quintile of mean probing pocket depth (Saito 2005). There is a positive and significant association between high body mass index and the number of teeth with periodontal disease; this may be explained by obesity being responsible for a systemic low-grade inflammatory state (Lalla et al. 2006). People with higher categories of body mass index and upper body abdominal fat have a significantly increased risk of presenting with periodontitis (Saito et al. 2001).

There are significant correlations between body composition and periodontal disease. Body mass index and abdominal visceral fat are significantly associated with periodontitis (Wood, Johnson, and Streckfus 2003). Only 14% of normal-weight people have periodontitis; although 29.6% of overweight people and 51.9% of obese people present with periodontitis. High percentage of body fat, which is a person's total fat divided by that person's weight, is significantly associated with periodontal disease, with an odds ratio of 1.8 (Khader et al. 2009).

Physical Activity

There is an inverse linear association between sustained physical activity and periodontal disease: increased physical activity induces an improvement in insulin sensitivity and glucose metabolism. Periodontitis risk decreases with increased average physical activity. Compared with men in the lowest quintile for physical activity, those in the highest quintile have a significant 13% lower risk of periodontitis. Physically active patients present with significantly less average radiographic alveolar bone loss (Merchant et al. 2003).

Waist-to-Hip Ratio and Waist Circumference

High waist-to-hip ratio is a significant risk factor for periodontitis. Upper-body obesity as measured by the waist-to-hip ratio or the waist circumference is related to visceral abdominal adiposity. Because of induced systemic inflammation and insulin resistance by adipose tissue, it represents a risk factor for type 2 diabetes and cardiovascular diseases. Patients with a high waist-to-hip ratio present a significantly increased risk for periodontitis (Saito et al. 2001). Periodontitis is more frequent among patients with high waist circumference and high waist-to-hip ratio; high waist circumference is significantly associated with periodontitis with an odds ratio of 2.1 (Khader et al. 2009). Adolescents aged 17 to 21 years old have an 1.05 times increased risk of periodontal disease per 1-cm increase in waist circumference (Reeves et al. 2006). Waist-to-hip ratio, which characterizes abdominal visceral fat, is statistically significantly associated with periodontitis. There are significant correlations between body composition and periodontal disease, waist-to-hip ratio being the most significant element associated with periodontitis (Wood, Johnson, and Streckfus 2003). High waist-to-hip ratio is also significantly associated with the highest quintile of mean probing pocket depth (Saito et al. 2005).

Adipokines

Adipocytes produce cytokines, or adipokines, which are responsible for the association between obesity and other disease. Adipocytes in the adipose tissues of obese people produce large quantities of leptin, which regulates energy expenditure and body weight (Nishimura et al. 2003). Adiponectin and resistin are adipokines, which are responsible for systemic inflammation and insulin resistance in obese people. Serum resistin levels are higher in patients with periodontitis than in healthy subjects. Periodontitis patients with at least one tooth with a probing pocket depth greater than 6 mm have two times higher serum resistin levels than subjects without periodontitis (Furugen et al. 2008). Periodontitis is significantly associated with increased resistin levels. Resistin and adiponectin are secreted from adipocytes, and resistin plays an important role in inflammation (Saito et al. 2008).

Experimentation

Experimental calorie-restriction diet may have anti-inflammatory effects. A low-calorie diet results in a significant reduction in ligature-induced gingival index, bleeding on probing, probing depth, and attachment level. Periodontal destruction is significantly reduced in low-calorie-diet animals (Branch-Mays et al. 2008). After oral infection with *Porphyromonas gingivalis*, mice with diet-induced obesity present a significantly higher level of alveolar bone loss, with 40% increase in bone loss 10 days after inoculation. Accompanying the increase in bone loss, obese mice show an altered immune response with elevated bacterial counts for *P. gingivalis* (Amar et al. 2007).

The Metabolic Syndrome

Metabolic syndrome is characterized by the following:

- Central visceral obesity
- Hypertriglyceridemia and low levels of high-density lipoprotein cholesterol
- Hypertension
- Insulin resistance

Abdominal visceral obesity is characterized by an increased waist circumference.

Atherogenic dyslipidemia is defined by raised triglycerides and low concentrations of high-density lipoprotein cholesterol, elevated apolipoprotein B, small high-density lipoprotein cholesterol particles, and small low-density lipoprotein cholesterol particles.

Hypertension is characterized by chronic elevated blood pressure.

Insulin resistance or lowered insulin sensitivity is associated with high risk for cardiovascular disease and diabetes.

A proinflammatory state is generally present with the elevation of serum C-reactive protein because adipose tissues release inflammatory cytokines, inducing the elevation of C-reactive protein.

Prothrombotic state is characterized by raised serum plasminogen activator inhibitor and high fibrinogen (Grundy et al. 2004). The metabolic syndrome is associated with severe periodontitis; these patients are 2.31 times more likely to present with the metabolic syndrome. The prevalence of the metabolic syndrome is

- 18% among patients with no or mild periodontitis,
- 34% among patients with moderate periodontitis, and
- 37% among patients with severe periodontitis (D'Aiuto et al. 2008).

NUTRITION

Omega-3 Polyunsaturated Fatty Acids

Sources of omega-3 polyunsaturated fatty acids (eicosapentaenoic acid and docosahexaenoic acid) can be found in animals and especially in fish such as salmon, tuna, and mackerel. They are also present in many vegetables and nuts (alphalinolenic acid), such as walnuts and almonds. They are capable of reducing proinflammatory cytokine levels (Enwonwu and Ritchie 2007).

Fish oil rich in omega-3 polyunsaturated fatty acids, especially eicosapentaenoic acid and docosahexaenoic acid, may protect from bone loss in chronic inflammatory diseases, such as rheumatoid arthritis or periodontitis.

A fish oil-enriched diet inhibit alveolar bone resorption after experiemental *P. gingivalis* infection. *P. gingivalis* infected rats fed omega-3 polyunsaturated fatty acids have the same alveolar bone levels as do the healthy animals. Omega-3 polyunsaturated fatty acid dietary supplementation can modulate inflammatory reactions leading to periodontitis, with reduction of the alveolar bone resorption (Kesavalu et al. 2006).

Free Radicals, Reactive Oxygen Species, and Antioxidants

Free radical-induced tissue damage and antioxidant defense mechanisms are important factors present in inflammatory diseases. High levels of reactive oxygen species activity combined with low antioxidant defense can lead to inflammatory diseases such as periodontitis.

Oxidative stress is an imbalance between excess production of reactive oxygen species and low antioxidant defense. The reactive oxygen species are

- superoxide anions,
- hydroxyl radicals, and
- peroxyl radicals (Nassar, Kantarci, and van Dyke 2007).

P. gingivalis induces the release of inflammatory cytokines such as interleukin-8 and tumor necrosis factor-α, leading to an increased activity of polymorphonucleocytes. After the stimulation by bacterial antigens, activated polymorphonucleocytes produce the reactive oxygen species (Sculley and Langley 2002).

Systemic inflammation accelerates the consumption of antioxidants such as vitamins and minerals. Increased production of reactive oxygen species necessitates more antioxidant elements such as zinc, copper, and selenium. Selenium has oxidation-reduction functions, and selenium-dependent glutathione enzymes are necessary for reduction of damaging lipids (Enwonwu and Ritchie 2007). In periodontitis, oxidative stress is present either locally and in the serum. Low serum antioxidant concentrations are associated with higher relative risk for periodontitis. Low serum total antioxidant concentrations are inversely associated with severe periodontitis (Chapple et al. 2007).

Lycopene is an antioxidant carotenoid contained in vegetables, particularly in tomatoes. In periodontitis patients, there is a significant inverse relationship between serum lycopene levels and C-reactive protein, and between monthly tomato consumption and white blood cell count. There is also an inverse relationship between monthly tomato consumption and congestive heart failure risk. For moderate monthly tomato consumption, the risk ratio for congestive heart failure is 3.15; for low monthly tomato consumption, the risk ratio is 3.31; and for very low monthly tomato consumption, the risk ratio is 5.1. For people without periodontitis and with moderate serum lycopene level, the risk ratio for congestive heart failure is 0.25 (Wood and Johnson 2004). Peri-implant disease is caused by bacteria infection associated with inflammation and tissue destruction, which is induced by free radicals and reactive oxygen species. In saliva of patients with peri-implant

disease, the total antioxidant status and the concentrations of antioxidants such as uric acid and ascorbate are significantly decreased. On the contrary, total antioxidant status and concentrations of uric acid and ascorbate are higher in healthy people (Liskmann et al. 2007). The total antioxidant capacity of the gingival crevicular fluid and plasma is significantly lower in chronic periodontitis. Successful periodontal therapy increases significantly the total antioxidant capacity of gingival crevicular fluid (Chapple et al. 2007). Gingival crevicular fluid antioxidant concentration is significantly lower in periodontitis. Total antioxidant capacity of plasma is also lower in periodontitis, which can result from excessive systemic inflammation or may induce the periodontal destruction (Brock et al. 2004).

Fusobacterium-stimulated polymorphonucleocytes induce the release of reactive oxygen species, which are responsible for a high degree of lipid peroxidation (Sheikhi et al. 2001). Imbalance between oxidative stress and antioxidant capacity may be responsible for periodontal disease. Lipid peroxidation is significantly higher in periodontitis patients. On the contrary, total antioxidant capacity in saliva is significantly lower in periodontitis patients (Guentsch et al. 2008). Reactive oxygen species are responsible for the destruction of periodontal tissues because of the imbalance between oxidant and antioxidant activity.

In periodontitis, gingival crevicular fluid presents a significantly higher lipid peroxidation level. Saliva shows lower antioxidant glutathione concentration and higher lipid peroxidation level. Periodontal therapy induces a significant decrease of lipid peroxidation and a significant increase in glutathione concentrations (Tsai et al. 2005). Gingival crevicular fluid total antioxidant capacity is significantly decreased in periodontitis patients, presenting lower mean plasma antioxidant capacity. Concentrations of glutathione, which has antioxidant activity, are lower in gingival crevicular fluid because of decreased glutathione synthesis and increased local degradation. In periodontitis plasma and gingival crevicular fluid contain a lower mean total antioxidant capacity (Chapple et al. 2002). Total salivary antioxidant concentrations are significantly lower in periodontitis because of the enhanced action of the reactive oxygen species, which may also predispose to increased effects of reactive oxygen species on periodontal tissues (Chapple et al. 1997).

Superoxide dismutases are antioxidant enzymes that neutralize superoxide radicals. Copper, zinc, and superoxide dismutase are antioxidants that play a protective role against oxidation caused by infections (Balashova et al. 2007).

After stimulation by bacterial antigens, polymorphonucleocytes produce superoxide radicals. The increased number and activity of leukocytes induce an important reactive oxygen species release, with damage to periodontal tissues and to alveolar bone. Ascorbate, albumin, and urate are antioxidant elements of plasma; although urate is the main antioxidant of saliva (Sculley and Langley-Evans 2002). Reactive oxygen species are produced by leukocytes during an inflammatory response. Periodontal destruction is secondary to the imbalance in the antioxidant and oxidant activity in periodontal pockets. Reactive oxygen species are responsible for extracellular matrix proteoglycan degradation because of their oxidant action (Waddington, Moseley, and Embery 2000).

Nutritional Status

Nutrition

Malnutrition impairs phagocytic function, cell-mediated immunity, complement system, and antibody and cytokine production. Protein energy malnutrition is responsible for impaired immunity and multiplication of oral anaerobic pathogens.

Inflammation necessitates the use of increased quantities of vitamins and minerals. Adequate energy and nutrients are necessary for the production of acute phase proteins, inflammatory mediators, and antioxidants.

Calcium and Vitamin D

Calcium and vitamin D are two important elements for bone metabolism. Women with hip osteoporosis have more than three times the alveolar bone loss around posterior teeth than do women without hip osteoporosis. Calcium and phosphorus are major minerals in hydroxyapatite crystals, and vitamin D regulates calcium and phosphorus metabolism and intestinal absorption. Calcium and vitamin D dietary intake is essential for bone health in periodontitis. Calcium and vitamin D medical supplementation is always necessary for osteoporosis treatment and prevention (Kaye 2007).

Whole Grain

Periodontitis may decrease with higher dietary whole-grain intake; four whole-grain servings per day may decrease the risk. Men in the highest quintile of whole-grain intake are 23% less likely to have periodontitis than are those in the lowest (Merchant et al. 2006).

Diet

Patients with metabolic syndrome who undergo 1 year of a nutritional program show the following significant changes in gingival crevicular fluid:

- Reduction of clinical probing depth
- Reduction of gingival inflammation
- Reduced levels of interleukin-1β
- Reduced levels of interleukin-6 (Jenzsch et al. 2009)

Cranberry

A treatment with a cranberry antioxidant fraction prepared from cranberry juice inhibits *Aggregatibacter actinomycetemcomitans*-induced interleukin-6, interleukin-8, and prostaglandin E2 inflammatory mediators production, as well as cycloxygenase-2 inflammatory enzyme expression (Bodet, Chandad, and Grenier 2007).

Green Tea

Catechins are antioxidants derived from green tea; they are able to reduce collagenase activity and tissue destruction. Collagenase activity in gingival crevicular fluid of highly progressive periodontitis patients is inhibited by green tea catechins. Among green tea catechins, epicatechin gallate and epigallocatechin gallate have the most important inhibitory effect (Makimura et al. 1993). Green tea catechins may help in the treatment of periodontal disease. Green tea catechins show a bactericidal effect against Gram-negative anaerobic bacteria such as *P. gingivalis* and *Prevotella* spp. After a mechanical treatment and the local application of green tea catechins, pocket depth and proportion of Gram-negative anaerobic rods are significantly reduced (Hirasawa et al. 2002).

Garlic

Garlic has antimicrobial properties against periodontal pathogens and their enzymes. Periodontal pathogens present among the lowest minimal inhibitory concentrations and the lowest minimum bactericidal concentrations of garlic. Garlic inhibits trypsin-like and total protease activity of *P. gingivalis* (Bakri and Douglas 2005).

Onion

Onion extracts may possess a bactericidal effect on some oral pathogens such as *Streptococcus mutans*, *Streptococcus sobrinus*, *P. gingivalis,* and *Prevotella intermedia* (Kim 1997).

Vitamins

Vitamin C

There is a significant association between low vitamin C levels and periodontal attachment loss. Patients with vitamin C deficiency show more attachment loss than subjects with normal serum vitamin C levels (Amaliya et al. 2007). Serum vitamin C level is inversely correlated to attachment loss; clinical attachment loss is 4% greater in patients with lower serum vitamin C level (Amarasena et al. 2005). Low serum vitamin C is inversely associated with periodontitis, especially in severe disease. Higher serum vitamin C concentrations are associated with less-severe periodontitis, with an odds ratio of 0.5 (Chapple, Miward, and Dietrich 2007). Chronic periodontitis patients present significantly reduced plasma vitamin C levels; after 2 weeks of dietary vitamin C intake as grapefruit consumption, the plasma levels rise significantly and the sulcus bleeding index is reduced (Staudte, Sigush and Glockmann 2005). *P. gingivalis* infection is associated with low levels of serum vitamin C; there is a highly significant inverse association between plasma vitamin C and *P. gingivalis* antibody levels. High antibody titers to *A. actinomycetemcomitans* and *P. gingivalis* are inversely correlated with low levels of vitamin C, especially for vitamin C concentrations lower than 4 mg/L (Pussinen et al. 2003).

Vitamin B

Chronic periodontitis patients supplemented with multiple vitamin B medications, show significantly lower mean clinical attachment levels (Neiva et al. 2005).

Alcohol Consumption

There is a significant positive linear relationship between high alcohol consumption and periodontal parameters such as mean clinical attachment loss and mean probing depth (Amaral, Luiz, and Leao 2008).

Deep probing depth is significantly associated with high alcohol consumption with an odds ratio of 7.72 (Negishi et al. 2004). Alcohol consumption is significantly associated with increased severity of clinical attachment loss, with the following odds ratios:

- 1.22 for 5 drinks per week
- 1.39 for 10 drinks per week
- 1.54 for 15 drinks per week
- 1.67 for 20 drinks per week (Tezal et al. 2004)

Alcohol consumption is significantly associated with probing depth and attachment loss. For 15–29.9 g alcohol per day, patients have a significantly higher odds ratio (2.7) of having more than 35% of their teeth with probing depth greater than 4 mm (Shimazaki et al. 2005). People who drink alcohol have a higher risk of getting periodontal disease: it is an independent risk factor for periodontitis.

- For 0.1–4.9 g per day, the relative risk is 1.24.
- For 5–29.9 g per day, the relative risk is 1.18.
- For more than 30 g per day, the relative risk is 1.27 (Pitiphat et al. 2003).

Gamma-glutamyl transpeptidase enzyme serum levels are elevated in case of liver damage by chronic alcohol intake. Severe alcohol use with plasma gamma-glutamyl transpeptidase level greater than 51 IU/L is significantly associated with periodontal parameters such as plaque index, gingival margin

level, gingival index, probing depth, and attachment loss (Khocht et al. 2003).

Elevated levels of reactive oxygen species following chronic alcohol consumption induce an increased periodontal inflammation, high oxidative damage, and elevated tumor necrosis factor-α concentrations. In rats with ligature-induced periodontitis, ethanol feeding decreases the ratio of reduced oxidized glutathione. Alcohol intake increases polymorphonuclear leukocyte infiltration, tumor necrosis factor-α production, and gingival oxidative damage (Irie et al. 2008).

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