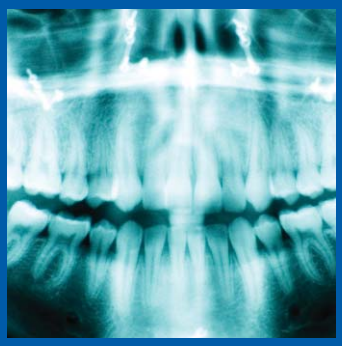
ILSI North America

Healthy Mouth, Healthy Body

What we know about the connection between oral and system health

Denis Kinane, DDS, PhD
Christine Ritchie, MD, MSPH
Cheryl Toner, MS, RD

Healthy Mouth, Healthy Body



With today's increasing attention being paid to disease prevention, the connections between oral health and other health issues become all the more important to understand.

New information is emerging on the importance of keeping our mouth clean and healthy to ensure total health and quality of life.

First published in the
United States of America
in 2009 by ILSI North America
1156 Fifteenth Street, NW
Suite 200
Washington, DC 20005 USA

ILSI North America
1156 Fifteenth Street, NW
Suite 200
Washington, DC 20005 USA

+1.202.659.0074 Tel
www.ilsina.org

The North American branch of the International Life Sciences Institute (ILSI North America) is a public, non-profit scientific foundation. ILSI North America advances the understanding and application of scientific issues related to the nutritional quality and safety of the food supply.

The organization carries out its mission by sponsoring relevant research programs, professional education programs and workshops, seminars, and publications, as well as providing a neutral forum for government, academic, and industry scientists to discuss and resolve scientific issues of common concern for the well-being of the general public. ILSI North America's programs are supported primarily by its industry membership.

Table of Contents

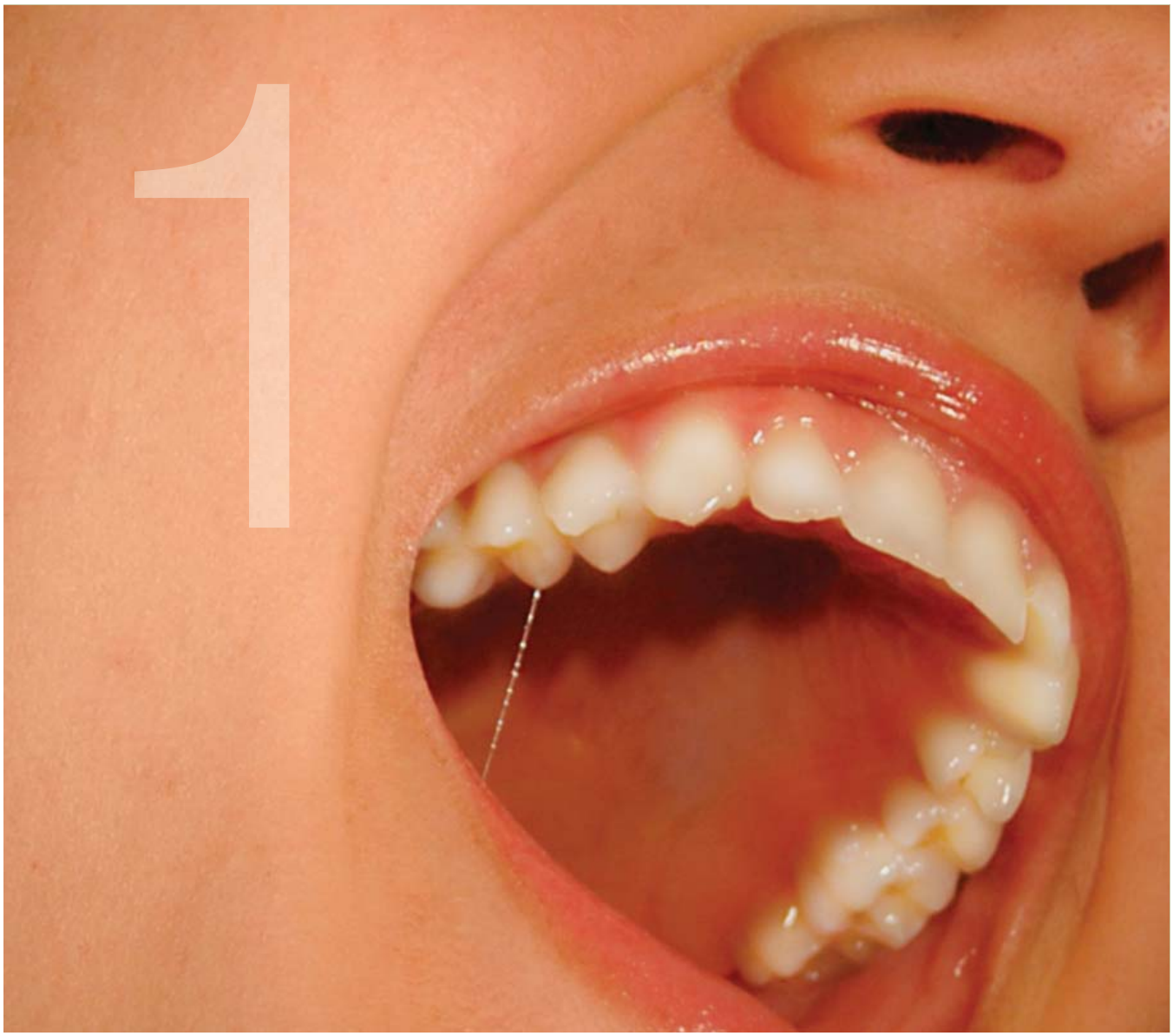
Introduction

Cavities, gum disease, and "bite" problems are often the only conditions commonly associated with oral health. However, the mouth has significant roles in maintaining health through immune and digestive functions, and promoting well-being and self-esteem through communication and aesthetics. The mouth is also related to many systemic health disorders, such as diabetes, cardiovascular disease (CVD), obesity, adverse pregnancy outcomes, and human immunodeficiency virus (HIV) infection.

With ever-increasing attention to prevention and wellness, oral health has emerged as both a reflection of overall health and a potential avenue for reducing other health risks. As this exciting area of research develops, there is mounting evidence for the important role of a clean and healthy mouth in total health and quality of life.

This monograph is designed to give readers an introduction to the connections between the health of the mouth and the health of the body. Mouth anatomy and physiology will be reviewed. Building upon the better-known functions of digestion, mucosal defense, and communication, the significance of oral functions on systemic health will be examined. Dental and gum diseases have been shown to be associated with a number of chronic health issues, including CVD, diabetes, and preterm birth. While this frontier is exciting, limitations in the current body of evidence will be discussed and potential avenues for future scientific discovery identified.

Perhaps the most empowering aspect of oral health for the public is the reality that most oral diseases are preventable, particularly with recent advances in dental treatment as well as the availability of effective products to enhance self-care. Attention to the actions that consumers can take today is critical, while medical and scientific research continues the quest for a greater understanding of the mouth-body connection.



1

Structures and Functions of the Mouth

A healthy mouth, often taken for granted, is more than a portal for food or the expression of a pleasant smile. Digestion is initiated in the mouth, or oral cavity. Communication is enriched. A brief review of the proper functioning of the mouth in this chapter will lay the groundwork for understanding oral health and its impact on systemic health.

Structures of the Mouth

The mouth, or oral cavity, is a small anatomical structure with a highly complex physiology. In addition to the lips, border surfaces (hard and soft palates, cheeks, uvula), connective tissues, teeth and bone, the mouth is home to structures and systems that play integral roles in digestion, communication, and immune response (see Figure 1).

The entire mouth, with the exception of the teeth, is covered by a layer of mucosa. The tongue is a muscular organ covered by a specialized mucous membrane, forming small bumps called papillae, and taste buds. The salivary glands and certain lymph tissues reside within the cheeks and floor of the mouth, and the tonsils are found just behind the oral cavity in the pharynx.

Teeth are a major anatomical feature of the mouth (see Figure 2). Each tooth consists of a visible portion called the crown and a hidden portion below the gumline called the root. The surface of the crown is covered by enamel, a thin layer of extremely hard calcified tissue. The surface of the root is covered by cementum, a thin layer of softer calcified tissue. The periodontal ligament, a connective tissue, binds the cementum to the bone that holds the tooth in the jaw. Beneath the enamel and cementum lies the body of the inner tooth, or dentin. Dentin is softer than enamel, but harder than bone. It surrounds the pulp chamber within the crown and its extension into the root of the tooth is called the root canal. Within these spaces are the pulp, an area where nerve endings and blood vessels sensitize and nourish the tooth.

The gingival tissue, or gum, surrounds the root of the tooth. The gums represent a vulnerable spot in the oral mucosa, as the gingival epithelial tissue lining, or gingival sulcus (groove between tooth and surrounding gingival tissue), does not have a protective keratin layer. Additionally, the gingival sulcus is home to one of the highest concentrations of bacteria found anywhere in the body.

Figure 1. Illustration Of Mouth Anatomy

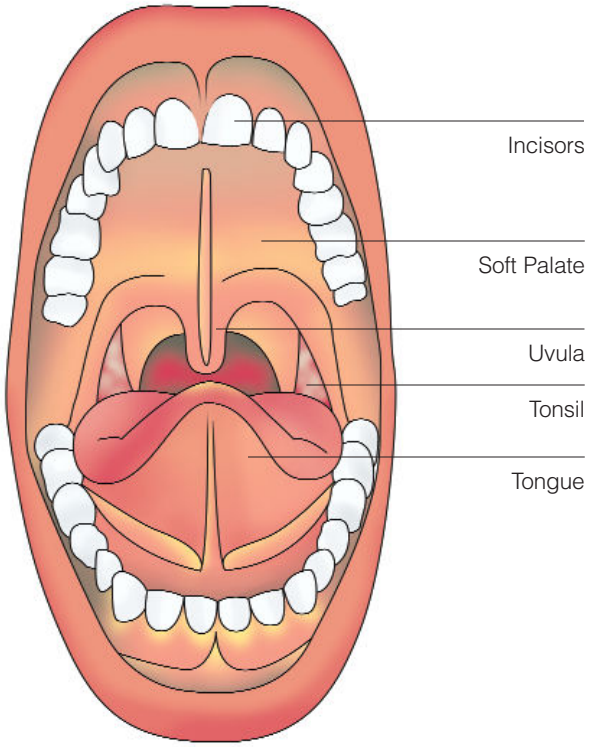
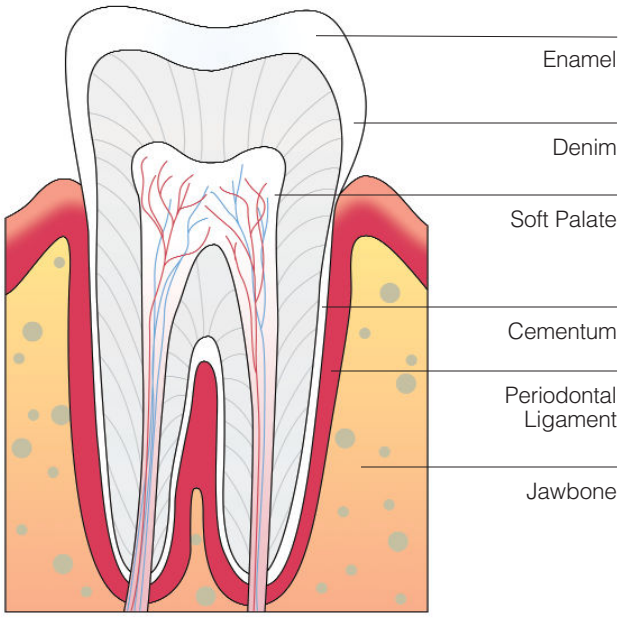


Figure 2. Illustration Of Tooth Anatomy



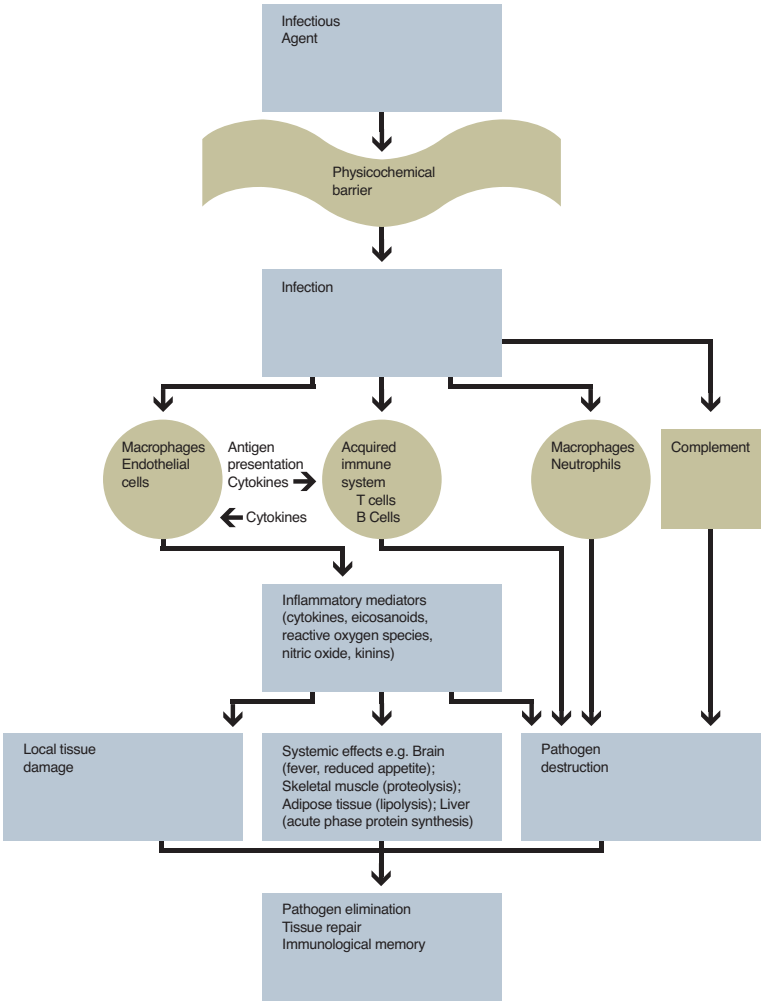
Adapted from *Merriam-Webster's Collegiate® Encyclopedia©2000* by Merriam-Webster, Incorporated (www.Merriam-Webster.com).

Normal Functions of the Mouth

Digestion is initiated in the mouth, with the obvious functions of ingesting, chewing, and swallowing food, as well as the first exposure of food to the digestive components in saliva. Food is chewed and mixed with saliva to form a mass called a bolus. The bolus is collected on the back of the tongue and swallowed. There is a brief suspension of breathing as the nasal passages and larynx are closed off to prevent choking or aspiration.

Of course, the taste buds in the mouth provide the sense of taste, which works synergistically with the sense of smell to allow enjoyment of foods and beverages, and stimulate saliva secretion. The taste function of the mouth helps to ensure adequate and safe nourishment from food, although in recent generations it is determined more by culture and habit than survival instincts. Still, foods that require more chewing or provide gustatory stimulation (e.g., sour or tart flavors) can, in fact, stimulate saliva production (Touger-Decker et al. 2007).

Figure 3. Normal Immune Function



Source: J. Nutr. (2007; 137, 773S-780S), American Society for Nutrition

Although the digestive and communication functions of the mouth are front and center, the oral immune surveillance system works to detect antigens entering the body through the mouth. Detection of these foreign substances can trigger both innate and acquired (or adaptive) immune responses (Calder 2007; See Figure 3). The oral and the entire gastrointestinal mucosa are an important part of the innate immune system, serving as a barrier to foreign substances, or antigens. Saliva complements the mucosal barrier function, mediating bacterial adhesion and reducing demineralization by coating the teeth with a thin film called pellicle. In a healthy mouth, saliva directly fights microbial invaders with enzymes and other innate components and buffers oral tissues from acidic foods, beverages, and bacterial byproducts which effectively counteract cariogenic demineralization.

Acquired humoral immune function, which involves secreted antibodies, is also active in the mouth (Calder 2007). Saliva, tonsils, lymph nodes, and lymph tissues in the palate and tongue (see Figure 1) detect and respond to immune threats by producing and delivering secretory immunoglobulin A (IgA) antibodies via saliva to protect against specific antigens.

Potential Barriers to Oral Functionality

While the mouth's role in digestion ends after swallowing is completed, it is clear that poor oral health can be a significant barrier to proper digestion. Missing teeth may also cause other teeth to shift into undesirable positions that lead to malocclusion (improper positioning of teeth and jaw), thereby further interfering with chewing. Denture and partial denture wearers typically experience chewing and speech difficulties during a period of adaptation, which is typically more difficult for older patients. Ultimately, pain and difficulty associated with eating may cause the individual to reduce food intake or narrow food choices, thereby risking malnutrition.

Inadequate saliva production may cause difficulty in swallowing and increase risk of aspiration (entry of secretions or foreign material into the trachea and lungs), as well as reduce the antimicrobial and buffering capacity normally provided by saliva. Dry mouth, or xerostomia, results from decreased saliva production and may be related to diseases such as Sjögren's syndrome, HIV infection, sarcoidosis, amyloidosis, or depression. Treatments, such as radiation therapy to the head and neck area, or certain medications may also impact saliva production. Special attention to oral care is vital with xerostomia, including periodic professional cleaning, the use of artificial saliva, and aids in self-care such as prescription fluoride dentifrices and gels and antimicrobial chlorhexidine rinses. In Sjögren's syndrome, new medications may be beneficial in replacing saliva or in stimulating better salivary flow.

As discussed in greater detail later, oral disease may increase the risk for aspiration pneumonia both through interference with adequate chewing and swallowing, and the increased presence of pathogenic bacteria in the oral cavity. Because aspiration pneumonia is caused by the dislocation of bacteria from the oral cavity to the lungs, good oral hygiene, including rigorous tooth brushing and professional prophylaxis of the teeth, can reduce risk of pneumonia, especially in elderly and/or debilitated patients.

Whatever the causes may be of infection, pain, dysfunction, or anatomical changes in the oral cavity, the resulting challenges can be extensive, including malnutrition, social isolation, and depression. However, the importance of a healthy mouth is illustrated even more clearly when the impact of poor oral health on the rest of the body is considered.

Oral Health: More than Just a Pretty Smile

Oral health can have significant direct and indirect social implications. The mouth is vital for interpersonal communication, including speaking and nonverbal facial expressions. Therefore, poor oral health may directly interfere with communication by causing difficulty or pain associated with speaking or forming particular expressions. Changes in physical appearance and/or alterations in speaking or chewing abilities may lead to poor self-confidence and social avoidance behaviors. The individual may avoid eating, speaking, laughing, or smiling with others. The potential for a cascade of physiological and psychological effects of avoiding social interaction is significant (DHHS 2000).

2. Diseases of the Teeth and Gums

Orofacial pain afflicts more than 20 percent of the adult population (DHHS 2000). Oral pain can result from caries (tooth decay or cavities), periodontal (gum) disease, or nerve, bone, or joint problems. Pain and the functional barriers created by poor oral health can have a negative impact on quality of life and pose a significant economic cost to society through missed work and school days.

Understanding the etiology of dental and periodontal diseases improves prevention and treatment efforts, and illustrates the potential mechanisms through which oral health affects other body systems.

Dental Caries

Dental caries (tooth decay), the most common chronic disease in children and young adults, affects more than 90 percent of adults and nearly 60 percent of adolescents (CDC 2007). In children, caries affects 42 percent of the primary teeth and 21 percent of the permanent teeth in children six to eleven years old. In fact, while decreasing for most Americans, cavities are increasing in young children (two to five years old), particularly in the primary teeth (CDC 2007). The pain, infection, and tooth loss that result from untreated dental caries have far-reaching health impacts.

Tooth decay is influenced by the interaction of four factors: the level of salivary flow; acid produced by bacterial flora; the presence of fermentable carbohydrates; and time. The mouth, like the rest of the gastrointestinal tract, is host to a complex bacterial population. The gastrointestinal tract is initially colonized at birth, when maternal bacteria are passed to the newborn infant. Only a few of the hundreds of bacterial species found in the mouth are cariogenic (cavity-forming). Anaerobic (oxygen-avoiding), bacteria attach to the tooth surface in layers referred to as biofilm. Although the makeup of oral bacteria is highly variable from person to person, a few common species are likely to lead to formation of caries. These cariogenic anaerobic bacteria are protected from oxygen by additional layers of biofilm made up of aerobic (oxygen-seeking) bacteria. The many layers of biofilm can become encrusted with minerals and form a hard dental plaque. The bacteria in the biofilm use fermentable carbohydrates from food as a source of energy to produce acids, which subsequently cause demineralization of the tooth enamel.

In a healthy mouth, there is a balance between enamel demineralization and remineralization. Acid produced by bacteria following a meal or snack containing fermentable carbohydrates starts to attack the enamel just below the surface, resulting in an area of demineralization that is the initial stage of tooth decay. Saliva serves to wash away carbohydrates and acids from the tooth surface, as well as neutralize the bacterial acids and provide calcium and phosphate to remineralize the subsurface lesion. Fluoride helps to promote remineralization and, when incorporated in newly formed tooth structure, enhances resistance to future acid attack.

Poor oral hygiene, individual behaviors (e.g., dietary patterns, smoking), and certain systemic health conditions can tip the scale in favor of demineralization. The first evidence of a cavity is typically a white spot indicating demineralization beneath the tooth surface. At this stage, the tooth surface is still intact and under favorable conditions the demineralized region can be remineralized and the carious process reversed. If demineralization continues to progress, the surface collapses and a cavity is formed that requires professional treatment. The cavity may deepen and eventually reach the pulp resulting in painful lesion.

Tooth decay is influenced by the interaction of four factors:

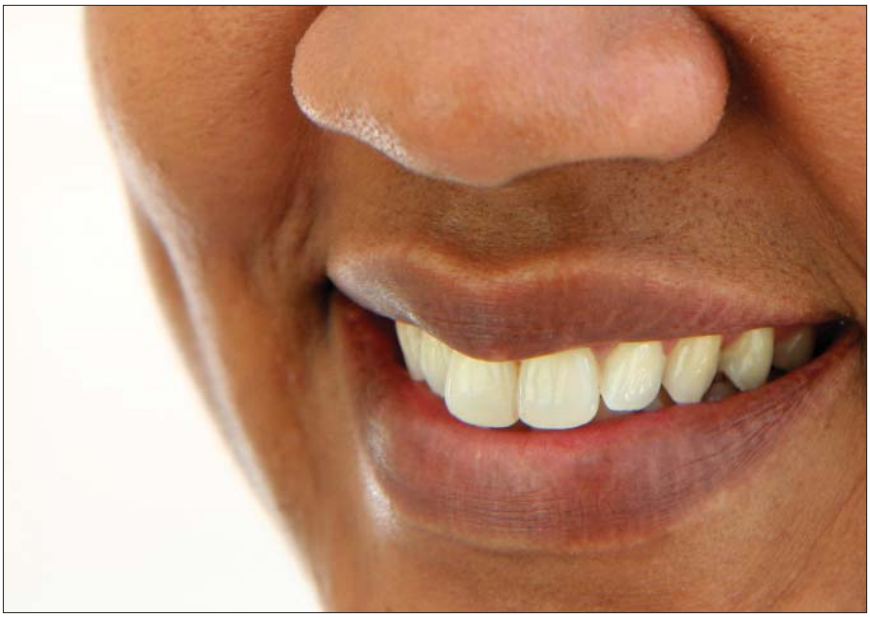
Host:	*Amount of salivary flow*
Agent:	*Presence of acid- producing bacteria (Composition of bacterial flora)*
Environment:	*Fermentable carbohydrates that feed the bacteria*
Time:	*Tooth exposure time to acid*

Periodontal Diseases

Gingivitis and periodontitis, the periodontal diseases, are distinct disorders resulting from a chronic inflammatory response to bacterial plaque. Gingivitis affects the gums, while periodontitis affects the gums and the bone and soft tissues that anchor the teeth. Without adequate oral hygiene, bacteria can quickly settle on and colonize clean tooth surfaces. If the resulting plaque is not controlled, clinical signs of gingivitis may appear within a few days. It has been estimated that as many as 500 different species of bacteria can be found in the mouth, many of which can be found in the dental plaque that forms at the gumline. A number of these bacteria produce toxins and other substances that can directly destroy gingival tissue and indirectly affect gingival tissues by activating an immune response. The activated immune system responds by sending neutrophils to attack the offending bacteria. In the course of attacking the bacteria, substances are released from the neutrophils that also break down gingival tissues. This initial gum tissue inflammation, called gingivitis, is characterized by redness, swelling and bleeding. If adequate oral hygiene is resumed quickly, inflammation of the gums can be resolved.

If gingivitis is allowed to persist, it can progress to periodontitis. There are several distinct forms of periodontitis, which vary according to the age at which they develop the distribution of involved teeth, and the rate of disease progression. Periodontitis is similarly triggered by the damage inflicted by bacterial toxins and characterized in the early stages by neutrophil and antibody responses. When neutrophils are unable to control the bacterial infection, however, the immune system releases macrophages to engulf the bacteria and cellular debris. Numerous immune cells are activated, including prostaglandin E2 and the cytokines, interleukin-1, tumor necrosis factor alpha (TNF-α), and interferon gamma, which work to regulate immune responses, bone resorption, and connective tissue damage. Kornman 2008 provides a current model for the pathogenesis of periodontitis.

In periodontitis, the space between the gum and tooth deepens as the supporting bone is lost to form periodontal pockets. These pockets are colonized by a wide variety of bacteria that are inaccessible to usual oral hygiene measures. With time, the walls of the periodontal pockets become ulcerated allowing bacterial products access to the general blood circulation. As will be discussed below, it is these accumulations of bacteria and their products which have been associated with a number of diseases and conditions elsewhere in the body.



Risk Factors for Incidence and Progression of Oral Diseases

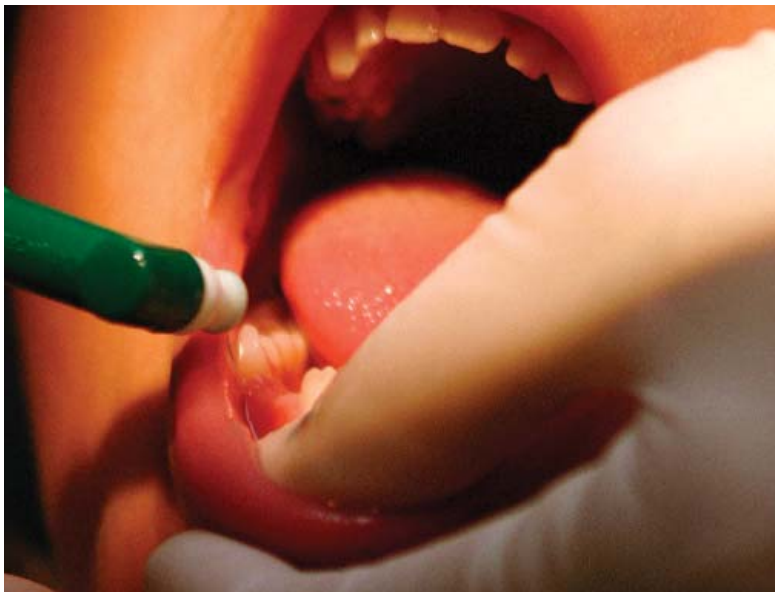
Although fluoride and other dental advancements have been very effective in reducing the prevalence of untreated tooth decay, caries is still a significant problem throughout the world (WHO 2007). Several factors have been associated with increased risk for caries and gum diseases. Some have led to effective interventions for prevention and treatment, such as fluoridation of public water systems (CDC 2008). Others may prove to be useful in the future. Individual risk is highly variable, however. Teasing out factors that are "guilty by association" is important, particularly since socioeconomic correlations have been inconsistent from one study to another. Still, according to the Surgeon General's report on "Oral Health in America" and the Centers for Disease Control and Prevention, epidemiological evidence indicates that there are disparities in oral health that must be addressed through not only education regarding self care, but also public health interventions (DHHS 2000, Dye et al. 2007).

Risk for both caries and gum disease is associated with lower socioeconomic status, and varies within population subsets by gender, age, ethnicity, and race (DHHS 2000, Dye et al. 2007). In general, risk increases with male gender, increasing age, Mexican-American ethnicity, and non-Hispanic black race. There are variations within each of these subpopulations, however, suggesting significant behavioral and/or genetic influences. For example, lower income is often found to be a stronger predictor of risk than a particular ethnicity. And while risk increases with age, caries incidence is generally on the decline for all ages except for in the primary teeth of children who are 2 to 5 years old. Regarding gender, estrogen has been debated as a potentially protective factor (Genco and Grossi 1998); however males are also less diligent with professional dental care (DHHS 2000). It is notable that lifestyle factors associated with caries and gum disease risk, including tobacco use (See Impact of Smoking on Oral Health), dietary patterns, oral hygiene, and professional dental care, are also correlated with socioeconomic status, race, and ethnicity.

The particular characteristics of dietary habits that are known to contribute to caries risk include the nutrient composition of a food or combination of foods, physical properties of the food(s), frequency of eating, the order in which foods are consumed, and the overall quality of the diet (Touger-Decker et al. 2007). The presence of fermentable carbohydrates, including sugars and cooked starches (e.g., breads, cereals, pastas, rice, pretzels, crackers, and some vegetables) are essential for the production of acid by oral bacteria.

Reducing the length of exposure to cariogenic foods is important. Therefore, the physical characteristics of the food are an important consideration. A food that adhere to teeth and gums, particularly if consumed frequently throughout the day when brushing and flossing is delayed, creates favorable conditions for bacterial growth and production of acids and toxins. Frequent snacking, including beverages sipped between meals, increases exposure time to fermentable carbohydrates and thus increases caries risk. Children who are provided with anything other than water in a bottle at bedtime or in a "sippy" cup throughout the day have an increased risk of caries due to prolonged exposure to sugars.

In addition to dietary habits that may increase caries risk, nutritional deficiencies may compromise appropriate immune response and increase periodontitis risk (Touger-Decker et al. 2007). In a state of malnutrition, additional calories and protein are needed to promote healing. The composition of saliva can be adversely affected by inadequate nutrition. Adequate calcium and phosphorus are essential for strong teeth and for preserving the tooth mineralization balance. In general, a diet that is adequate in essential nutrients will ensure tissue integrity and normal immune function, and promote healing as needed. In particular magnesium, zinc, calcium and vitamins A, C, and E are critical for immune function, and iron and vitamins B12, B6, and C protect the gums.



The risk of gum diseases is increased by diseases and conditions, including type 1 and type 2 diabetes mellitus, HIV, and osteoporosis. Hormonal changes, such as those seen during puberty and during pregnancy, result in exaggerated signs and symptoms of gingivitis. In addition, some medications such as nifedipine (a calcium channel blocker used for high blood pressure), phenytoin (used to control epilepsy), and cyclosporin (an immunosuppressive drug) can result in gingival overgrowth.

Although detection of infectious bacteria in the oral cavity was once hoped to be a potential indicator of periodontitis risk, development of the disease is now known to be more complex than the presence of infectious bacteria. It is widely accepted that susceptibility to periodontitis varies greatly between individuals who harbor the same disease-causing bacteria in their respective oral cavities (DHHS 2000). In fact, those who develop periodontitis seem to have a unique host response to certain bacteria and plaque, emphasizing the interplay between the genetics, environment, and behavior. Current research is evaluating the potential for genetic polymorphisms to serve as markers for oral disease risk. Although there is potential for significant advancement in this arena, the body of research is not yet conclusive for any particular marker (Kornman et al. 1997, Huynh et al. 2007, Sumer et al. 2007).

Impact of Smoking on Oral Health

An association between periodontitis and smoking has been observed for nearly 60 years (DHHS 2000). For the 46 million American adults who smoke, studies show an increased risk of severe alveolar bone and tooth loss compared to nonsmokers, even after controlling for other behavioral and socio-demographic risk factors.

Several theories exist regarding the disease-causing effects of smoking on periodontal tissues, including direct tissue damage by toxins, altered plaque microbial composition, and reduced immune and health response due to diminished saliva production, blood flow, and oxygen delivery to tissues (DHHS 2000). Ironically, smoking tends to reduce gingival inflammation through nicotine's effects on vascular constriction. However, nicotine does not stop the disease process. It only masks the symptoms, increasing risk of delayed treatment.

Prevention and treatment of periodontal disease is less effective in smokers (DHHS 2000). Therefore, smoking cessation is crucial to periodontal health and significantly increases an individual's likelihood of tooth retention. Individuals who stop smoking altogether for more than 10 years reduce periodontitis risk to levels enjoyed by non-smokers (Tomar and Marcus, 1998).

Signs, Symptoms, and Risk Factors for Diseased versus Healthy Gums

	Signs and Symptoms	Risk Factors
Healthy Gums	Firm, do not bleed, light pink, fit snugly around teeth	N/A
Gingivitis	Gums are bright red or red-purple and may be swollen, shiny, may bleed easily on brushing or eating, may be tender, bad breath	Dental plaque, certain drugs, hormonal changes (pregnancy), uncontrolled diabetes, misaligned teeth, overhanging dental restorations
Periodontitis/ Advanced Periodontitis	Gums begin to separate and recede from teeth, pockets deepen, teeth loosen and may be lost, bad breath	Dental plaque, smoking, poor nutrition and dietary patterns, alcohol consumption, diabetes mellitus, advanced age, male gender, and lower socioeconomic level

3 Gateway to the Body and Mirror of Health

Advances in biomedical science have led to the understanding that localized symptoms can often be related to dysfunction in more than one body system. Many nutrient deficiencies can be manifested in the mouth. In addition, diseases of the oral mucosa and periodontal tissues can result in inflammation and the intrusion of microbial pathogens into the rest of the body. Evidence has shown associations between periodontal disease and numerous other health problems, including diabetes mellitus, CVD, adverse pregnancy outcomes (Renvert 2003) and obesity. The mouth is, therefore, a critical reflection, as well as gatekeeper, of overall health.

Diabetes

Approximately 20.8 million people in the United States have diabetes mellitus, according to a 2005 estimate of the Centers for Disease Control and Prevention (CDC 2005). Up to one-third of these cases are undiagnosed. Diabetes is an increasingly common disease characterized by a disturbance of glucose metabolism. Normally, glucose is carried into cells, where it is metabolized for energy by insulin, a pancreatic hormone. In type 1 diabetes mellitus, the pancreatic beta-cells that produce insulin are destroyed via an autoimmune process in susceptible individuals. As a result, the body does not produce enough insulin. In type 2 diabetes mellitus, lifestyle and/or coexisting health conditions reduce insulin sensitivity in susceptible individuals. Body cells then do not respond to insulin properly. In both types of diabetes, glucose accumulates in blood and eventually spills over into urine.

If blood glucose is poorly controlled, numerous complications may develop. In fact, because there is no cure for this progressive disease, some degree of comorbidity is expected to develop over the life span of the diabetic individual. Periodontal disease is a potential complication of diabetes, along with CVD, kidney damage (nephropathy), eye damage (retinopathy), nerve damage (or neuropathy),

and pregnancy complications, including birth defects. Severe periodontitis is also associated with increased mortality in individuals with diabetes (Skamagas et al. 2008). Further research is needed to determine if there is a causal relationship between diabetes and periodontal disease (DHHS 2000, Skamagas et al. 2008, Mealey and Rose 2008). However, the evidence for diabetes as a risk factor for periodontal disease prevalence, severity, and extent of symptoms is consistent.

It is possible that diabetes increases risk of periodontitis in a manner similar to other vascular complications of diabetes. Specifically, the accumulation of advanced glycation end products and alterations in the immune-inflammatory response may accelerate gum disease.

At the same time, periodontal disease is associated with a higher risk of poor glycemic control and certain complications of diabetes. One possible explanation is that chronic exposure to bacteria, bacterial acids and toxins, and products of inflammation may decrease insulin sensitivity (Skamagas et al. 2008, Mealey and Rose 2008).

Clinical research examining the effect of periodontal treatment on glycemic control has produced inconsistent results, however. Further research elucidating the relationship between periodontal disease and diabetes is needed so that the potential for improved outcomes in these disorders can be realized.

Cardiovascular Diseases

Heart disease and stroke are, respectively, the first and third leading causes of death in the United States. One in three American adults has one or more types of CVD, including high blood pressure, coronary heart disease, stroke, and congestive heart failure. It is becoming increasingly clear that infections and chronic inflammatory conditions as seen in periodontitis are similar to those which cause and accelerate arterial plaque formation, or atherosclerosis, the precursor to many types of CVD.

Numerous studies have revealed associations between periodontal disease and cardio-, cerebro-, and peripheral vascular diseases. Increased levels of antibodies specific to periodontal pathogens have been associated with coronary heart disease, atherosclerosis, coronary events, and stroke, and periodontal bacteria have been detected in atheromatous plaque.

Periodontitis and atherosclerosis are complex and distinct diseases that nonetheless share common risk factors, such as smoking, poor nutrition, alcohol consumption, diabetes mellitus, advanced age, male gender, and lower socioeconomic level. Although there has been some inconsistency in the data, well-designed, large studies have demonstrated a positive association between periodontal disease and CVD, even after controlling for these potentially confounding factors (Demmer and Desvarieux 2006).

Associations appear to be strongest among younger male subjects and for stroke outcomes. Periodontitis may also be a contributing risk factor for atherosclerosis, in that the physical changes caused by tooth loss, or the pain caused by dental and gingival tissue damage may cause dietary changes, such as reduced intake of fruits and vegetables, that favor atherosclerosis (DHHS, 2000).

It is important to recognize that an association between two conditions does not, in itself, imply that one causes the other. The few intervention trials conducted in the area of periodontal disease treatment and measures of systemic inflammation and CVD were small, not randomized, and poorly controlled. Therefore, more research is needed to understand the nature of the association between these diseases.

Adverse Pregnancy Outcomes

Normal hormonal changes brought about by pregnancy cause an increase in periodontal pocket depth that resolves after birth (Dasanayake et al. 2008). However, pre-existing periodontitis in the mother has been correlated with preterm birth (prior to 37 weeks gestational age) and low birth weight (less than 2500 grams), serious contributors to infant mortality around the world. In one study, mothers with severe periodontal disease had a seven-fold higher risk of delivering a pre-term and low birth weight infant than mothers with healthy gums (Offenbacher et al. 1996, 1998).

The immune system is again at play, responding to the presence of bacteria and bacterial toxins in the blood with the release of cytokines and prostaglandins, components which may cause preterm labor. Prostaglandin levels in the gingival crevicular (sulcus) fluid of mothers of low birth weight infants have been shown to be higher than in controls. Animal studies suggest that the bacterial infection and/or the immune response may suppress fetal weight gain, and have demonstrated an association between periodontitis and unhealthy changes in the amniotic fluid. It is also important to remember the simple fact that the maternal flora is passed along to the infant, along with immune protection.

Gestational exposure to inflammation may have negative consequences for the developing fetus beyond the immediate risks associated with preterm delivery. Poor maternal health may bring about epigenetic changes that affect long term health risk. There may be an increased risk for neurological disorders in the near term and for Crohn's disease, Alzheimer's disease, CVD, and diabetes in adulthood.

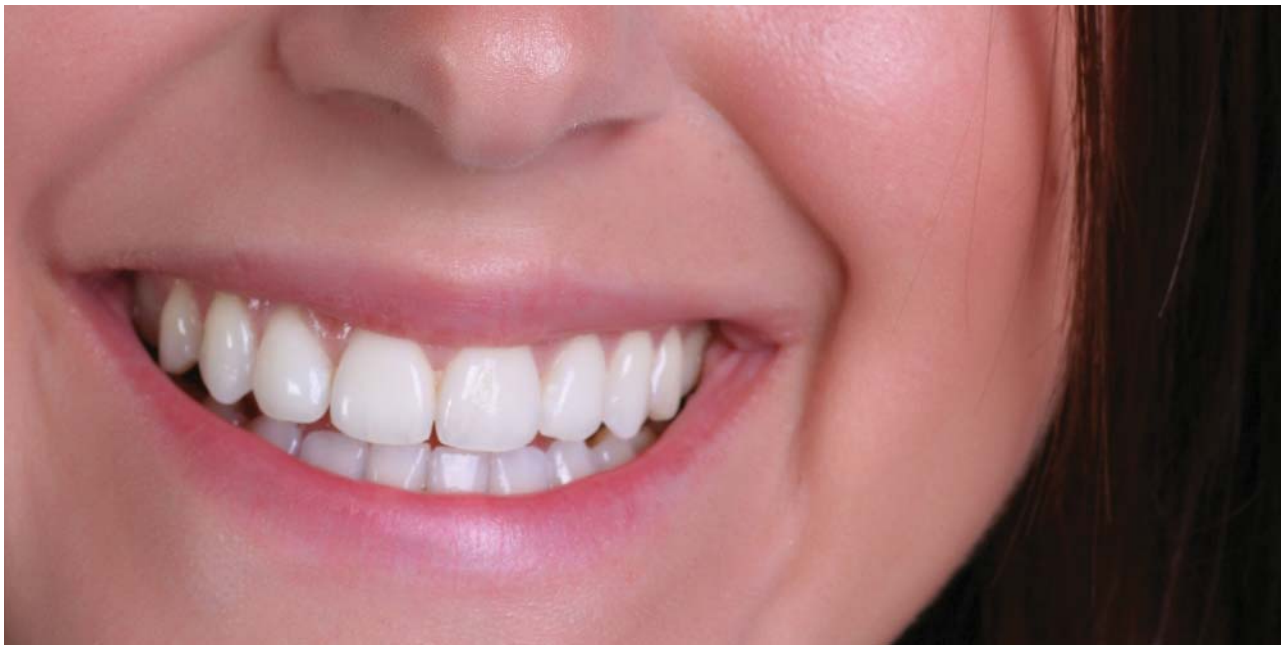
Clinical trials have demonstrated that treatment of periodontal disease prior to pregnancy, or during pregnancy, may reduce preterm delivery risk. The recommendation for oral health care during pregnancy, therefore, is quite clear.

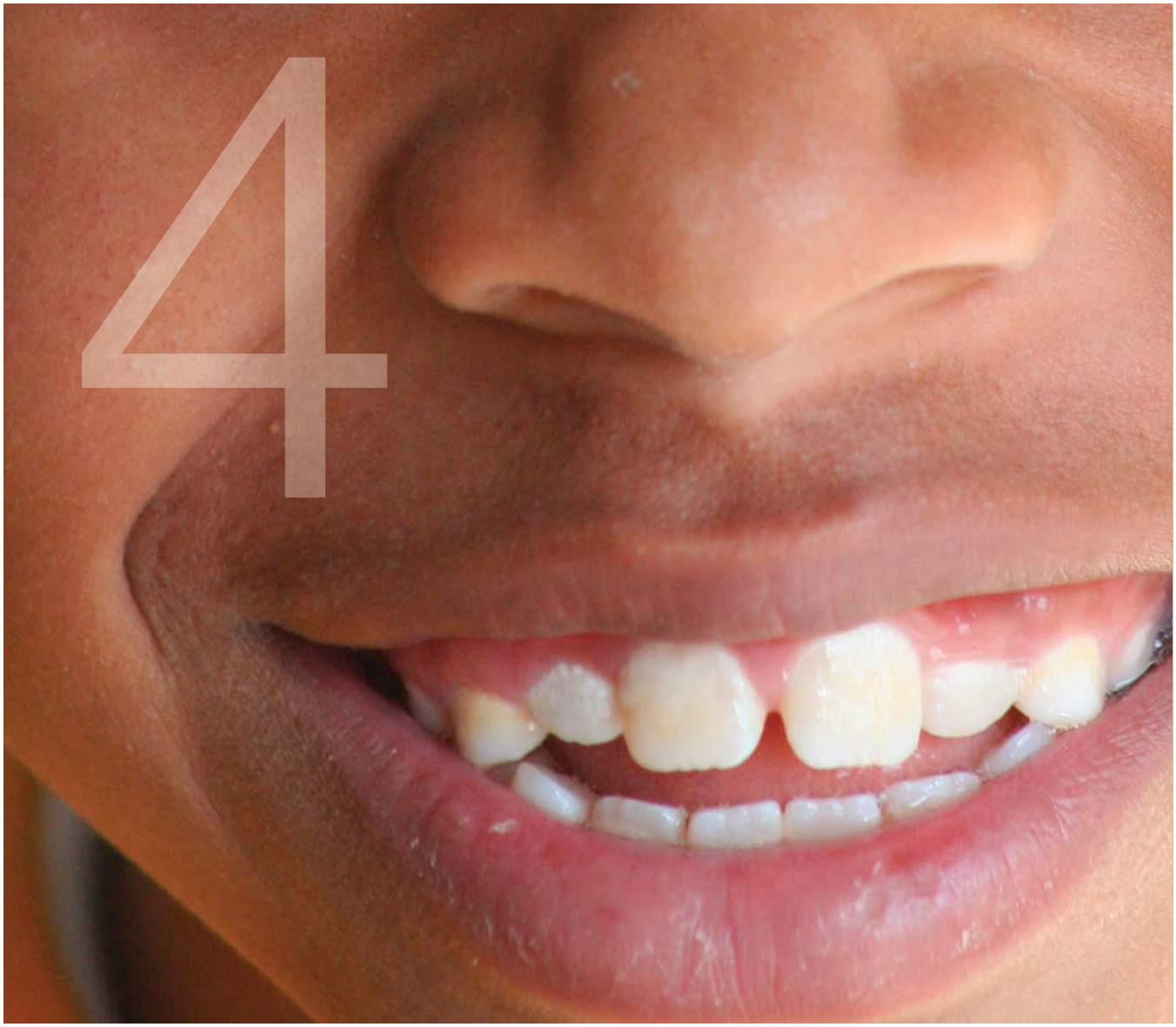
Obesity

Obesity is a growing public health concern around the world. Upper body or visceral obesity (around the waist) is closely associated with glucose intolerance, hyperinsulinemia, hypertriglyceridemia, and hypertension. Obesity is a risk factor for increased risk for morbidity and mortality from cardiovascular disease and diabetes. Fat tissue in the abdominal cavity accentuates free fatty acid levels in hepatic circulation and leads to increased production of triglycerides by the liver. Likewise, production of inflammatory chemical messengers is increased in abdominal obesity.

Obesity has recently been noted in human studies to be a risk factor for periodontal disease. In Japanese adults, increasing body mass index and waist-hip ratio was associated with increasing risk of periodontitis. In the US, a retrospective analysis of data from NHANES revealed that individuals who maintained normal weight, engaged in the recommended level of exercise, and had a high-quality diet were 40 percent less likely to have periodontitis compared to individuals who maintained none of these behaviors (Al-Zahrani et al. 2005).

The nature of this relationship has not been clearly determined. Some evidence suggests that adipose tissue secretes a variety of immune and inflammatory substances that affect metabolism and contribute to low-grade systemic inflammation. Interleukin-6 and TNF-α, in particular, stimulate acute phase reactions in the liver. Therefore, it is possible that increasing body fat may have the potential of modulating the host's immune and inflammatory response, rendering the patient more susceptible to the effects of microbial plaque.





4 Research Needs

The path from oral to systemic health provides multiple, diverse opportunities for research. Much remains to be discovered, including causal risk factors, mechanisms of action, genetic markers, and the interplay between genetics, environment, and behavior.

Teasing out Causal Relationships

How chronic illnesses, including oral diseases, interact with one another other remains poorly understood. Basic research is necessary to determine optimal exposure definitions, treatment targets, windows of intervention, mechanisms, biological markers, and appropriate populations in order to design intervention trials to determine whether periodontal infections are a causal factor in the development of CVD or cerebrovascular disease. Does treatment or prevention of periodontal disease lead to fewer cardiovascular events? Is there a threshold of irreversible subclinical CVD after which periodontal treatment or prevention would be ineffective (Demmer and Desvarieux 2006)? Long-term observation of oral health status, inflammatory and hemostatic markers, and CVD risk should also be undertaken, as well as determination of associations between genetic polymorphisms of oral health parameters, heart disease, and stroke (Meurman et al. 2004).

Interaction of Risk Factors

Nutritional factors may play an important role in periodontal disease. Additional research is needed to explore these associations further both at the population and basic science levels. Given the potentially protective role of antioxidants in periodontal disease, the interaction between smoking and antioxidant status should be carefully addressed. Much of the current basic research has tested nutrient mechanisms in chronic disease processes other than periodontal disease. Similar studies should be conducted to apply current understanding of nutrient mechanisms and inflammation to periodontal disease. The impact of adiposity (the amount of body fat) and weight change on inflammation and in periodontal disease is an area needing further investigation.

The rapidly emerging body of evidence regarding the interactions between the oral microbial community and the human host will likely provide answers and solutions to problems of oral and systemic health connections. The genome of the microbial community in the gastrointestinal tract will also likely be of increasing importance.

Treatment And Prevention

No longer is tooth loss an inevitable result of aging. Advances in dental care such as water fluoridation, fluoridated toothpaste, antimicrobial toothpastes and mouthrinses, and preventive periodontal care have made it possible for people to keep teeth and gums healthy and intact.

Collaboration amongst health care professionals is increasingly important with respect to oral health. The dentist can play a critical role in referring patients for evaluation of systemic health issues, based on oral health conditions. Proactive referrals to dental care providers are clearly needed for individuals with diabetes, those who are pregnant, and those who are experiencing challenges with oral functionality. A multi-disciplinary approach is often needed to support efforts to avoid tobacco products.

Of course, professional cleaning and examinations must be sought routinely, as numerous factors can tip the balance in favor of bacterial pathogens or overstimulate the immune response. These interactions with the dentist facilitate accurate and early detection of caries. Follow-up at three- to six-month intervals is critical, and sealants to reduce pit and fissure caries may also be useful.

It is important that the message about the far-reaching benefits of good oral hygiene be delivered by not only the dentist, but also the physician, the dietitian, and the nurse. Brushing, flossing, and rinsing on a consistent basis is the first line of defense against dental caries, periodontal disease, and the systemic effects that can be initiated by oral bacteria making its way into the bloodstream.

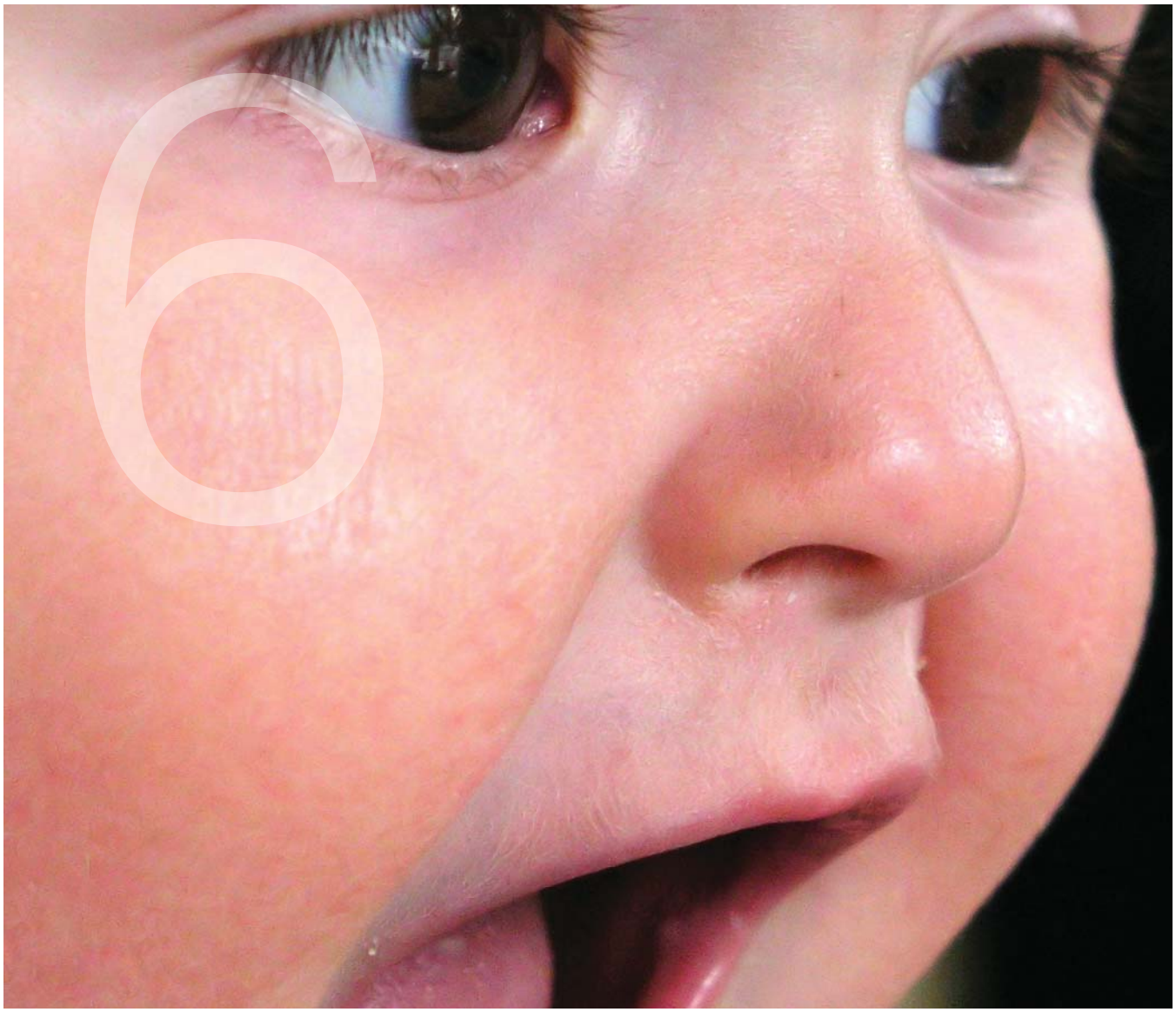
A consistent message from both dietitians and dentists regarding healthful dietary practices is achievable, as the Dietary Guidelines for Americans and MyPyramid provide recommendations that are consistent with oral health. In addition, there are four other strategies to focus on for oral health. First, consumers should be advised that foods high in fermentable carbohydrates should be consumed with protein-rich foods. Second, foods that stimulate saliva production can contribute to both good digestion and immune protection for the mouth.

Third, exposure time to fermentable carbohydrates should be reduced by drinking only water between meals and avoiding grazing and sipping on foods and beverages. Fourth, brush and floss after eating or drinking occasions, or chew a polyol-sweetened chewing gum when brushing is not possible.

Research continues to reveal the intricate connections between all facets of health in various body systems. Although more research is needed to ascertain the oral health: systemic health connection, preventing oral diseases is a well accepted aspect of health care that is relatively easy to educate on and to perform. There is no apparent downside to encouraging better oral health practices, which may have a significant immediate and long-term beneficial impact on our body. All of these reasons should provide motivation for individuals and health care providers to ensure a healthy mouth for a healthy body.

Personal Actions to Prevent Dental Caries and Gum Diseases:

- *Brush with fluoridated toothpaste at least twice a day and after each meal if possible.*
- *Floss every day.*
- *Rinse with antimicrobial mouth rinses after brushing.*
- *Chew sugar-free gum after meals if brushing is not possible.*
- *Visit the dentist for dental examination and cleaning every six months.*
- *Discuss dental sealant, a thin plastic-like coating applied to the chewing surfaces of the molars to prevent plaque accumulation, with the dentist.*
- *Eat a well-balanced diet.*
- *Avoid grazing or sipping on food or beverages that are rich in fermentable carbohydrates.*
- *Replace foods that are rich in fermentable carbohydrates with protein-rich foods.*
- *Avoid tobacco use.*
- *Avoid excessive alcohol consumption.*



Glossary and References

Glossary Of Oral Health Terms

Alveolar Bone – A thin layer of compact bone that forms the tooth socket surrounding the roots of the teeth.

Calculus (tartar) – A tough, crusty tooth deposit consisting of calcified plaque that cannot be removed by brushing or flossing. Tartar can form above or below the gum line and must be removed by professional dental cleaning.

Cementum – The thin layer of calcified tissue covering the dentin of the tooth root. Cementum is one of four tissues that support the tooth in the jaw. It is pale yellow with a dull surface and is softer than dentin.

Craniofacial – Relating to or involving both the skull (cranium) and the face.

Demineralization – Loss of mineral salts, especially from the teeth and bones.

Dental caries (tooth decay) – A destructive infectious disease of the teeth in which bacterial byproducts lead to demineralization and destruction of the tooth structure. Caries is Latin for rotten.

Dental prophylaxis– Professional tooth cleaning to remove plaque and tartar from the teeth.

Dentin – The most abundant dental tissue, dentin determines the size and shape of teeth. Dentin has a unique structure, softer than enamel and cementum, but harder than bone, and functions as the substructure for rigid enamel tissue. This provides teeth with the ability to flex and absorb tremendous pressure without fracturing.

Gingiva – Soft tissue surrounding the teeth. Also called gums.

Gingivitis – Inflammation of the gingiva (gum tissue).

Periodontitis – An infection resulting in inflammation of the and destruction of ligaments and bones that support the teeth; generally preceded by gingivitis.

Plaque – The accumulation of bacteria on tooth surfaces. Plaque is yellow-white and can be removed by daily brushing and flossing. However, if plaque accumulates it can lead to various oral diseases.

Tongue – The movable fleshy organ attached to the bottom of the inside of the mouth used for tasting, licking, swallowing, and speech.

Tooth Enamel –Outer layer of the crown of the tooth. It is a hard, thin, translucent layer of calcified substance that envelopes and protects the dentin (the main portion of the tooth structure) of the crown of the tooth. It is the hardest substance in the body and is almost entirely made up of alcium salts.

Xerostomia – Dry mouth.

References

Al-Zahrani MS, Borawski EA, Bissada NF. 2005. Periodontitis and three health-enhancing behaviors: maintaining normal weight, engaging in recommended level of exercise, and consuming a high-quality diet. Journal of Periodontology, 6:1362-1366.

Calder PC. 2007. Immunological parameters: What do they mean? Journal of Nutrition,137:773S–780S.

Centers for Disease Control and Prevention (CDC). 2005. National Diabetes Fact Sheet: General Information and National Estimates on Diabetes in the United States, 2005. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention, 2005. Available at: www.cdc.gov/diabetes/pubs/pdf/ndfs_2005.pdf (accessed February 2, 2008).

CDC. 2007. Trends in Oral Health Status: United States, 1988-1994 and 1999-2004. CDC National Center for Health Statistics. Available at: http://www.cdc.gov/nchs/data/series/sr_11/sr11_248.pdf (accessed October 27, 2008).

CDC. 2008. Populations Receiving Optimally Fluoridated Public Drinking Water – United States, 1992-2006. Morbidity and Mortality Weekly Report, 57:737-741.

Dasanayak AP, Gennaro S, Hendricks-Munoz KD, Chhun N. 2008. Maternal periodontal disease, pregnancy, and neonatal outcomes. The American Journal of Maternal Child Nursing, 33:45-49.

Demmer RT, Desvarieux M. 2006. Periodontal infections and cardiovascular disease: The heart of the matter. Journal of the American Dental Association, 137:14-20.

Dye BA, Tan S, Smith V, Lewis BG, Barker LK, Thornton-Evans G, et al. 2007. Trends in oral health status: United States, 1988–1994 and 1999–2004. National Center for Health Statistics. Vital Health Stat 11(248).

Genco RJ, Grossi SG. 1998. Is estrogen deficiency a risk factor for periodontal disease? Compendium of Continuing Education in Dentistry Supplement, (22):S23-9.

Huynh-Ba G, Lang NP, Tonetti MS, Salvi GE. 2007. The association of the composite IL-1 genotype with periodontitis progression and/or treatment outcomes: a systematic review. Journal of Clinical Periodontology, 34:305-17.

Kornman KS. 2008. Mapping the pathogenesis of periodontitis: A new look. Journal of Periodontology, 79(8):1560-1568. Internet: http://www.joponline.org/doi/full/10.1902/jop.080213.

Kornman KS, Crane A, Wang HY, di Giovine FS, Newman MG, Pirk FW, Wilson TG Jr, Higginbottom FL, Duff GW. 1997. The interleukin-1 genotype as a severity factor in adult periodontal disease. Journal of Clinical Periodontology, 24:72-7.

References (continued)

Mealey BL and Rose LF. 2008. Diabetes mellitus and inflammatory periodontal diseases. Current Opinion in Endocrinology, Diabetes & Obesity, 15:135–141.

Offenbacher S, Katz V, Fertik G, Collins J, Boyd D, Maynor G, McKaig R, Beck J. 1996. Periodontal infection as a possible risk factor for preterm low birth weight. Journal of Periodontology, 67(10 Suppl):1103-13.

Offenbacher S, Jared HL, O'Reilly PG, Wells SR, Salvi GE, Lawrence HP, Socransky SS, Beck JD. 1998. Potential pathogenic mechanisms of periodontitis associated pregnancy complications. Annals of Periodontology, 3(1):233-50.

Renvert S. 2003. Destructive periodontal disease in relation to diabetes mellitus, cardiovascular disease, osteoporosis and respiratory disease. Oral Health and Preventative Dentistry, 1(Supplement 1):341-357.

Skamagas M, Breen TL, LeRoith D. 2008. Update on diabetes mellitus: prevention, treatment, and association with oral diseases. Oral Disease, 14:105–114.

Sumer AP, Kara N, Keles GC, Gunes S, Koprulu H, Bagci H. 2007. Association of interleukin-10 gene polymorphisms with severe generalized chronic periodontitis. Journal of Periodontology, 78(3):493-7.

Tomar SL, Marcus SE. 1998. Cigarette smoking and periodontitis among U.S. adults. Journal of Dental Research, 77(Spec No B):830.

Touger-Decker R, Mobley CC. 2007. American Dietetic Association. Position of the American Dietetic Association: oral health and nutrition. Journal of the American Dietetic Association, 107(8):1418-28.

US Department of Health and Human Services (DHHS). 2000. Oral Health in America: A Report of the Surgeon General. Rockville, MD: U.S. Department of Health and Human Services, National Institute of Dental and Craniofacial Research, National Institutes of Health.

World Health Organization (WHO). February 2007. Fact Sheet Number 318: Oral Health. Internet: http://www.who.int/mediacentre/factsheets/fs318/en/index.html (accessed April 28, 2008).

Acknowledgements

This monograph was created with the support of the ILSI North America Oral Health Committee. The Oral Health Committee was created to raise awareness of the importance of oral hygiene and good nutrition for oral and systemic health, especially to health professionals outside the dental field.

The committee wishes to express its sincere thanks to Cheryl Toner, MS, RD as the main author of the document. The committee is also appreciative of contributions of committee advisor Dominick DePaola, DDS, PhD of Nova Southeastern University and The Forsyth Institute (Emeritus); committee Chair Doris Tancredi, PhD, Cadbury plc; and committee manager Laura Brockway-Lunardi, PhD.

ILSI North America Oral Health Committee 2008 Membership:
BENEO Group
Kraft Foods, Inc.
Cadbury plc
The Coca-Cola Company
Danisco USA
Wm. Wrigley Jr. Company