54

Nutrition and Oral Medicine

Dominick P. DePaola, Connie Mobley, and Riva Touger-Decker

Introduction

In the Spring of 2000, the Surgeon General of the United States released the first-ever report on "Oral Health in America." The intent of this landmark report is to alert the American people to the full meaning of oral health and its importance to general health and well being. The report has five major themes:[1]

1. Oral health means much more than healthy teeth.
2. Oral health is integral to general health.
3. Safe and effective disease prevention measures exist that everyone can adopt to improve oral health and prevent disease.
4. General health risk factors, such as tobacco use and poor dietary practices, also affect oral and craniofacial health.
5. There are significant oral health disparities among racial and ethnic minority population cohorts.

The overlying theme is that the etiology and pathogenesis of diseases and disorders affecting the craniofacial structures are complex and multifactional, involving an interplay and interaction among genetic, environmental, and behavioral factors. The major environmental factor in this interplay is diet and nutrition during development of the craniofacial complex, the maintenance of craniofacial structure integrity, and fending off subsequent microbial challenge. In fact, the two classic dental diseases, caries and periodontal disease(s), both have vital nutrition and dietary components. Caries is intimately linked to adequate nutrient intake during development of teeth and salivary glands, and to the frequent ingestion of fermentable carbohydrates post-eruption. Periodontal disease is generally considered to be caused by bacterial plaque residing on the tooth structure, but the inflammatory response can be modulated by adequate systemic nutriture. In terms of craniofacial disorders, cleft lip and palate — among the most common birth defects affecting humans — are linked, in part, to adequate folate nutriture during critical periods in craniofacial development, much the same way that neural tube defects are linked to

folate nutriture. Importantly, the diet and nutrition relationship to oral, dental, and craniofacial diseases is much more extensive than those classic illustrations. For example, systemic disease resulting from infectious oral microbes is generally recognized to occur in patients with immunological and nutritional deficiencies such that individual host defenses are compromised, allowing oral microbes to gain systemic access.[2] In turn, the oral, dental, and craniofacial tissues are the sites of signs and symptoms of about 120 systemic diseases.[1] Additionally, changing demographics suggest that an aging population will increasingly present medically significant oral problems.[1]

This section reviews the relationship between nutrition and oral, dental, and craniofacial diseases and disorders, the nutrient-tissue interplay, and, where appropriate, prevention, treatment, or intervention strategies using diet and nutrition. The section begins with an illustration of the burden of oral disease and proceeds to discuss chronic oral infectious disease (caries, periodontal disease, others); selected systemic diseases; neoplastic diseases; craniofacial-dental-oral birth defects; and health promotion, health education, and behavioral change.

The Burden of Oral Disease

Dental, oral, and craniofacial diseases and disorders are among the most common of human maladies, with widespread tooth loss due to caries and periodontal disease. Dental caries, in particular, disproportionately affects low socioeconomic populations and some racial/ethnic minorities. Additionally, oral and pharyngeal cancer results in over 12,000 deaths per year and has one of the worst morbidity and mortality rates of any cancer. Birth defects, particularly cleft lip and/or palate, are highly prevalent, as are a variety of chronic and disabling diseases and disorders, the oral complications of systemic diseases, and the oral complications of those interventions and medications consequent to treating systemic disease. Figure 54.1 illustrates the burden of disease according to the most recent NIDCR data.[1,3]

Chronic Oral Infectious Disease

Dental Caries

In spite of a substantial reduction over the last 20 years, dental caries continues to be a major problem for adults and children worldwide. For example, a 1988 to 1991 survey of the U.S. population showed that over 45% of children and adolescents in the 5- to 17-year age group had carious teeth.[4] In adults, over 93% had evidence of caries, with an increasing incidence of root caries with age.[5] The impact of dental caries on pain and suffering remains profound. The Surgeon General's Report estimates that 51 million school hours are lost each year to dental related illness, and in adults more than 164 million work hours are lost each year for dental related illness or treatment.[1]

The etiology of dental caries is well documented and results from the interplay of dental plaque present on the tooth surface with ingested fermentable carbohydrates. Many studies have documented that a demineralization-mineralization equilibrium occurs at the tooth-plaque-saliva interface, where the equilibrium balance favors demineralization

Birth Defect
- Cleft lip/palate births estimated at 1 in 600 for whites and 1 out of 850 for African Americans

Dental Caries
- 5-10% of pre-school age children have early childhood caries; rates are substantially higher in low income families and some racial/ethnic minorities
- 19% of all young children age 2-5 have untreated caries in primary teeth; the rate is 30% for children living in poverty
- 45% of school children and 94% of adults have experienced caries in permanent teeth
- 5x more common than asthma and 7x more common than hay fever

Dental Orthopedics
- 20% of adults and 18% of children have mild to severe malocclusions requiring orthodontic treatment

Candidiasis
- Candidiasis is the most common oral fungal infection in patients with immunodeficiency disorders, such as HIV infection

Edentulism - Total Loss of Teeth
- 11% of adults 25 and older have lost all natural teeth; the rate is 17% for adults living in poverty
- 30% of adults 65 and older are toothless; the rate is 46% for those in poverty

Head and Neck Cancer
- 12,300 deaths and 40,400 new cases per year for oral, pharyngeal and laryngeal cancers combined
- Five years after diagnosis, half of all patients with oral and pharyngeal cancers survive; less than a third of African American male patients survive that long

Osteoporosis & Oral Bone Loss
- Osteoporosis is a major health threat for 28 million Americans, 80% female
- Oral bone loss is also found in patients with osteoporosis

Salivary Gland Dysfunction
- Between 1 and 4 million Americans are estimated to have Sjögren's syndrome
- Cystic fibrosis affects about 30,000 Americans. More than 500 prescription and over-the-counter drugs have xerostomic effects

Trauma
- 19.8 Million emergency room visits are estimated for craniofacial injuries each year
- Motor vehicle crashes are leading cause of unintentional injury deaths in children, with majority resulting from head injuries
- 25% of people age 6-50 show evidence of anterior tooth trauma

Pain
- 22% of adults report some form of orofacial pain in last 6 months
- 6% of adults report pain symptoms commonly associated with temporomandibular joint disorders (TMD)
- Orofacial pain is a major component of Bell's palsy, trigeminal neuralgia, fibromyalgia, and diabetic neuropathy

Periodontal Diseases
- 90% of people 13 and older show evidence of periodontal problems
- Maternal periodontal infection appears associated with increased risk of spontaneous pre-term birth and low birth weight
- Periodontal infection appears to be a risk factor for cardiovascular diseases and stroke

Oral Complications of Cancer Therapy
- Each year an estimated 490,000 patients undergoing cancer therapy suffer from such complications as painful mouth ulcers, mucositis, rampant caries, fungal infections, impaired taste, and salivary gland dysfunction

Older Adults
- About 30% of adults 65 years and older are edentulous
- 23% of 65- to 74-year-olds have severe periodontal disease
- Most older Americans take medications which have oral side effects, especially dry mouth

FIGURE 54.1
The burden of disease. From: NIDCR (www.nih.nidcr.gov) 1999 and Surgeon General's Report, 2000.

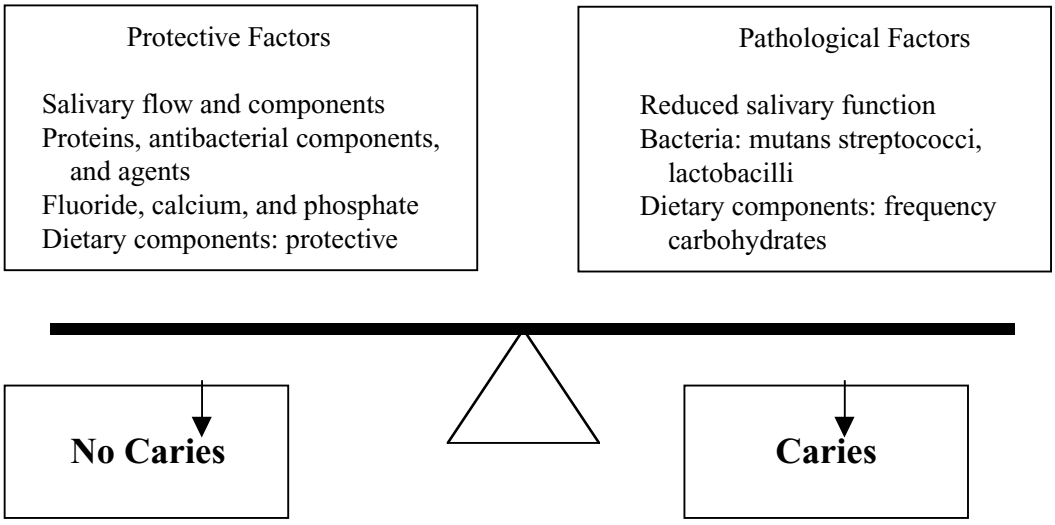

FIGURE 54.2
The caries balance: a schematic diagram of the balance between pathological and protective factors in the caries process. From: Featherstone, J.D.B., *J. Amer. Dent. Assoc.* 131: 887; 2000. With permission.

when the plaque pH drops, such as when carbohydrates (sugars) are fermented by plaque bacteria to form organic acids.[6] Mineral flows back when the pH is neutralized mostly due to the presence of salivary buffers and mineral ions, particularly when supplemented with fluoride.[6] Fluoride, when ingested at optimum amounts during tooth development (about 0.7 to 1.0 ppm), makes the enamel hydroxapatite crystal less soluble. Additionally, individuals with hyposalivation or xerostomia due to use of specific medications, head and neck irradiation, or chronic diseases like Sjögren's syndrome, lack appropriate salivary buffering capacity and thus have increased risk for caries.[7]

In a recent review, Featherstone illustrated that caries balance is dependent on the interaction of protective and pathological factors (Figure 54.2).[6] Dental caries represent an excellent example of how understanding the complex etiological agents of this multifactorial disease can have health promotion, intervention, and treatment consequences that can effect not only the disease itself, but the intricate interactions between health and nutritional status. As shown in Figure 54.3, the balance between health and disease in the oral and craniofacial complex is dependent on food choices and dietary patterns interwoven with nutritional and oral health status. Furthermore, there is a synergy between these two measures of health, (nutriture and oral status) that has a significant impact on general health and thus individual risk for many contemporary chronic and disabling diseases. If dietary intake leads to poor nutritional status, chronic disease is more likely to occur in the presence of additional risk factors that might include other lifestyle, environmental, and genetic factors.

Periodontal Disease(s)

Periodontal disease is an infection with local and systemic inflammatory effects. The initiation and progression of periodontal disease is markedly affected by risk factors. The relationship between nutrition and periodontal disease is multifaceted, where environmental and host risk factors contribute to its pathogenesis. Some of the host factors related to diet and nutrition include presence of other systemic or chronic disease, lifestage (preg-

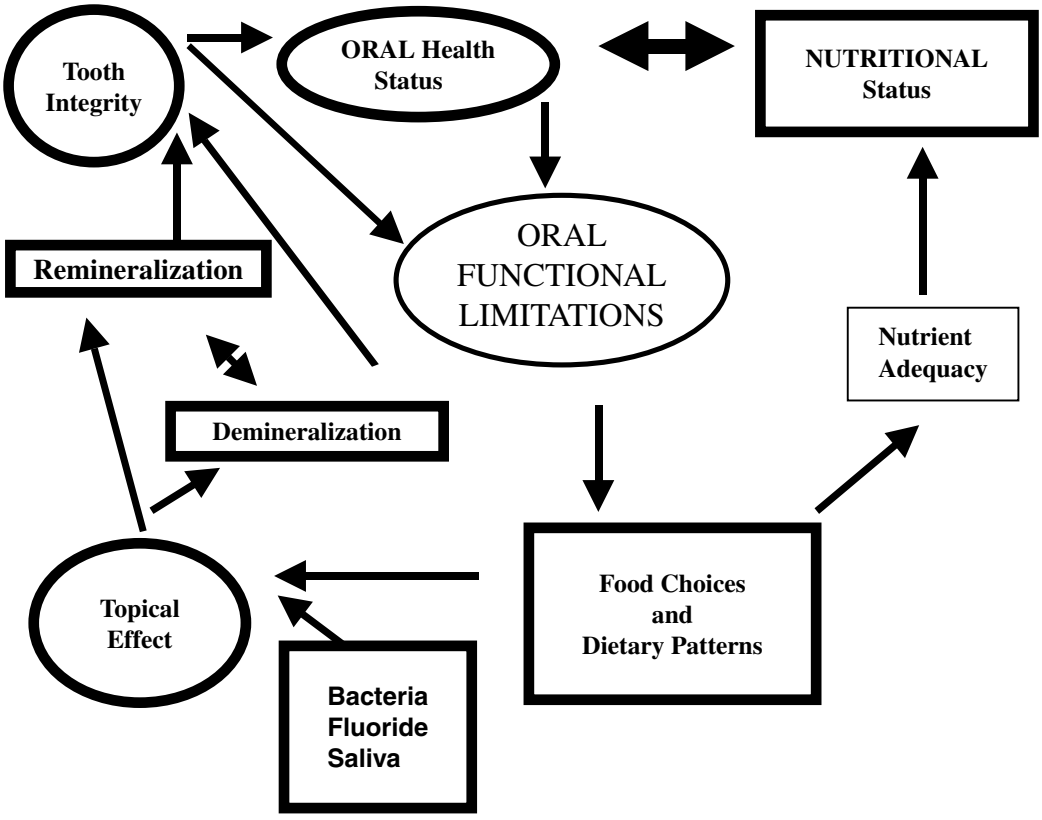

FIGURE 54.3
Diet and dental health.

nancy, menopause, elderly, etc.), osteoporosis, medications, and nutrition status. The current knowledge of nutrition and periodontal disease can be viewed in one of three ways:

1. Known relationships between periodontal disease, nutrition status, and immune response
2. Relationships of periodontal disease with nutrients that have been demonstrated in select populations
3. Unknown and yet to be tested relationships between periodontal disease, individual nutrients, and select host defense and health status variables

Known relationships include the impact of malnutrition on inflammation and infection, and the associations between calcium and periodontal disease. A superb overview of the new paradigm for the pathogenesis of periodontitis was provided recently by Page and Kornman, and is depicted in Figure 54.4.[8]

Nutrient deficiencies can compromise the system's response to inflammation and infection and increase the kcalories and protein needs necessary for adequate wound healing.[9] In this manner, poor nutritional status can impact host response to the inflammatory process and infection imposed by periodontal disease. A balanced diet along with good nutrition status is the appropriate nutrition management strategy for nutritional wellbeing

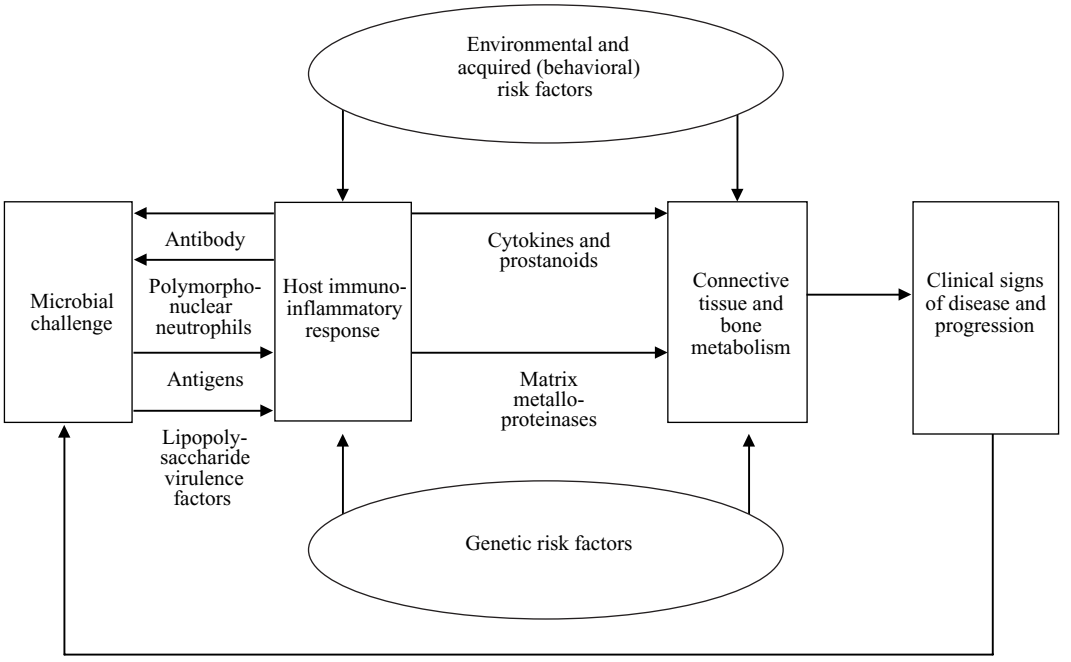

FIGURE 54.4
A new paradigm for the pathobiology of periodontitis. From: Page and Kornman, 1997, Periodontology 2000, 14: 9; 1997, with permission. Reprinted in Surgeon General's Report.

to reduce risk of malnutrition-associated compromises in immune and inflammatory responses and wound healing.

Relationships between periodontal disease, osteoporosis, and calcium intake is presented elsewhere in this section. Krall et al. have demonstrated that osteopenia and bone loss are associated with oral bone loss.[10] Treatments for osteoporosis, including hormone replacement therapy and biphosphanides, are associated with reduced oral bone and tooth loss. The relationship between calcium and vitamin D and incidence of periodontal disease remains to be demonstrated in broader gender- and age-related populations. The need for adequate calcium in the diet (either with foods and/or supplements) is important for prevention and management of osteopenia and osteoporosis as well as for periodontal disease.

The relationship between various antioxidants and periodontal disease is a developing area of research. Nishida et al. demonstrated that low dietary intake of vitamin C was significantly (although "weakly") associated with periodontal disease in individuals who currently smoke or have a history of smoking tobacco.[11] However, vitamin C has not been proven efficacious for improving periodontal health or reducing risk of disease in the general population.[11] Several studies have implicated deficiencies in ascorbate and folate with severity of gingivitis. For example, Leggott demonstrated that depletion of vitamin C was associated with increased gingival bleeding but no other markers of periodontal disease.[12]

The relationship between diabetes and periodontal disease is discussed elsewhere. However, the potential relationship between obesity and periodontal disease is a new area for investigation. In a study of obese, healthy Japanese men undergoing evaluation of periodontal status, there was a positive association between degree of obesity, as measured by body mass index, and incidence of periodontal disease.[13] After adjusting for age, sex, oral hygiene status, and smoking, the relative risk of periodontal disease was 1.3 for each

5% increase in body mass index.[13] Although type 2 diabetes is a recognized risk factor for periodontal disease, there was no association in this study between fasting blood glucose or glycosolated hemoglobin values and periodontal disease.[13] Further research is needed to explore relationships between obesity and periodontal disease, particularly in individuals with diabetes, a disease in which obesity can further compromise metabolic control.

Changes in oral tissues associated with aging can also impact periodontal status. Alveolar bone loses approximately 1% per year after age 30, and tooth mobility occurs more frequently in the elderly.[1] Soft tissue changes include thinning of the surface epithelium of the mucus membranes and gingival recession. Decreased salivary flow, most often secondary to medications or disease, is also common. These changes can also impact on nutrition status and eating ability. If severe, diet intake and nutrition status can suffer. The combined changes in the oral cavity increase the risk for nutrient intake compromise and periodontal disease.

Consumption of a balanced diet consistent with the food guide pyramid and inclusive of fruits, vegetables, grains, and adequate protein will provide sufficient vitamins, minerals, phytochemicals, and protein for overall and oral health. At this point, no published scientifically sound evidence exists to support the notion that individuals with periodontal disease need supplemental doses of any individual nutrients or groups of nutrients.

Environmental Oral Health Promotion: Fluoridation

In the early 1900s, Americans could expect to lose their teeth by middle age. With the discovery of the properties of fluoride and the adjustment of the concentration of fluoride in the supply of drinking water in the 1940s, this trend was reversed. Community water fluoridation remains one of the great achievements of public health and health promotion in the 20th century.[14] It is considered an inexpensive means of improving oral health benefits for all residents of a community. In 1992, 56% of the U.S. population was receiving fluoridated water at levels equal to or greater than 0.7 parts per million, either from natural or fluoridated public water systems. Dental caries prevention is effective at 0.7 to 1.2 part per million.[14] Some foods, beverages, and other dental products provide additional sources of fluoride to individuals.

Since the early days of community water fluoridation, the prevalence of dental caries in the U.S. has declined in communities with and without fluoridated water. This has been largely due to the diffusion of fluoridated water to areas without fluoridated water via food/beverage distribution channels and the widespread use of fluoride toothpaste.[15] Although early studies focused mostly on children, water fluoridation also reduces enamel caries in adults by 20 to 40%, and prevents root surface caries, a condition that particularly affects older adults.[1,16]

Health care providers are agents of health promotion at both individual and community levels. In the absence of a water supply with adequate fluoride to prevent dental caries, health promotion practices that support primary prevention through fluoride supplementation are encouraged. The American Dental Association Council on Scientific Affairs[17] and the American Academy of Pediatrics[18] recommend safe levels of daily fluoride supplementation for children living in unfluoridated communities. Table 54.1 describes the recommended supplementation schedule. Dietary fluoride supplements are available as tablets, drops, or lozenges. Supplementation has not been shown to be beneficial during pregnancy.[19,20] Flouridation is an inexpensive means of improving oral health, and benefits all residents of a community.

TABLE 54.1

Supplemental Fluoride Dosage Schedule (mg/day)[a]

Age	Concentration of Fluoride in Drinking Water (ppm)		
	< 0.3	0.3 to 0.6	>0.6
6 months to 3 years	0.25 mg	0	0
3 to 6 years	0.50 mg	0.25 mg	0
6 to 16 years	1.00 mg	0.50 mg	0

[a] ppm = parts per million; 2.2 mg sodium fluoride contains 1 mg fluoride.

From American Dental Association, *J. Am. Dent. Assoc.* 126: 19S; 1995. With permission.

Systemic Diseases

General Observations

The oral cavity is the gateway to the rest of the body, and at times reflects systemic as well as oral health and disease.[1] The mechanisms by which oral manifestations occur as a result of some systemic diseases are not fully known. Systemic diseases which affect the integrity of the oral cavity, and oral diseases themselves, may impact upon nutrition status, as well as the converse. Any condition affecting the functional capacity of the oral cavity (sensory or motor function) and/or the health and integrity of the soft tissue may impact on diet intake. If the integrity of the oral tissues is compromised due to infection, surgery, trauma, or medications, nutrient needs are increased. Unfortunately, this is often in the face of compromised intake due to pain, dysphagia, or anorexia. In these situations, nutrition status is compromised and the risk of malnutrition is increased. Malnutrition in turn can compromise wound healing and integrity of the immune system, further increasing the risk of oral infectious disease.

Emerging areas of research in dentistry in the 21st century include the relationships between oral infections and systemic diseases, and the oral manifestations and complications of systemic diseases and chronic disabling diseases.[2] It is anticipated that when fully appraised of these oral health–systemic health and nutrition interactions, health care professionals will be able to work collectively to identify dental and nutrition patient management strategies.

It is well documented that nutritional status may influence disease progression and recovery from illness, infection, and surgery. Malnutrition and individual nutrient deficiencies can affect tissue integrity and muscle function. A selected list of chronic diseases with recognized oral manifestations affecting nutrition status, and diseases with associated oral infections are listed in Table 54.2. Nutrition status may be affected via two primary mechanisms, functionally and metabolically, both of which can effect the sense of taste. Additionally, functional integrity of the oral cavity is critical for optimal smell, mastication, and swallowing; negative impacts on any of these functions can influence food and fluid intake, and consequently nutrition status. Metabolic impacts also include altered nutrient metabolism, typically increased catabolism of protein stores for energy, as well as increased protein and kcalorie needs due to infection. The end result of either a functional or metabolic mechanism(s) is often compromised nutrition status, increased risk of secondary infection, and altered response to disease and treatment. Weight loss of 10% or more, which can occur as a result of these diseases, increases morbidity and mortality.

TABLE 54.2

Selected Systemic Diseases with Potential Oral Manifestations Affecting Nutrition Status

Arthropathies	Autoimmune disorders
Cancers	Chronic orofacial pain
Cardiovascular disease	Diabetes
End-stage renal disease	HIV infection/AIDS
Inflammatory bowel disease	Liver disease
Megaloblastic anemia	Oral-facial pain syndromes
Osteoporosis	Pulmonary disease
Vesiculobullous diseases	Herpes Zoster

TABLE 54.3

Medications with Associated Oral Manifestations

Anticonvulsants	Antipsychotics
Antidepressants	Corticosteroids
Diuretics	Immune suppressing agents
Opiates	Serotonin uptake inhibitors
Tricyclic antidepressants	Protease inhibitors
Anti-anxiety agents	

Medications used to treat chronic diseases may also have oral sequellae that in turn compromise food intake and nutrition status. Xerostomia may occur independently, as a consequence of another systemic disease, or as a side effect of medication. More than 400 prescribed and over-the-counter medications are associated with xerostomia.[1] Xerostomia can affect sense of taste and swallowing ability as well as caries risk. Food texture and temperature may need to be modified for individuals with moderate to severe xerostomia. Drugs may also alter appetite and nutrient metabolism. Antidepressant medications and steroids can increase appetite, thus leading to increased risk of weight gain and subsequent obesity. Steroids increase catabolism of protein and carbohydrate, and lead to calcium losses. Individuals on long-term steroids are at risk for type 2 diabetes, and require calcium supplementation and added protein in their diets to reduce risk of osteoporosis and malnutrition, respectively. Classes of medications with oral manifestations are found in Table 54.3. Individual drugs are not listed; practitioners should check the Physicians Desk Reference (PDR) for individual drugs and their associated oral manifestations.

Table 54.4 addresses clinical symptomatology with associated oral clinical and systemic disorders and nutritional implications. Clinical features may represent one or more local and systemic diseases. It is imperative that clinicians look beyond the symptom to discern the actual cause of the symptomatology so as to treat the symptoms and the primary cause or disease. Although select clinical features may have an associated nutritional etiology, all other causes must be considered, and treatment aimed at the root cause and associated symptoms.

HIV-AIDS

As of December 1999, there were 33.6 million individuals with HIV/AIDS.[21] Estimates indicated 5.6 million new cases alone in 1999 and 2.6 million deaths worldwide from AIDS.[21] In the U.S., combination antiretroviral therapies have contributed to a decline in death due to AIDS as well as a reduction in the incidence of complications. Individuals with immunosuppressive diseases including HIV infection and AIDS are at increased risk

TABLE 54.4

Abnormal Oral Findings: Associated Local and Systemic Diseases

Clinical Feature	Associated Finding	Associated Disorders	Nutritional Considerations
Xerostomia	Excessive dental caries, candidiasis, dysphagia, dysgeusia, burning mouth/tongue	Drug-induced xerostomia, head and neck irradiation, Sjögren's syndrome, diabetes	Increase fluids, minimize cariogenic foods, modify food consistency and flavoring, evaluate glucose control in diabetes
Burning mouth/tongue	May be associated with mucosal erythema/atrophy, glossitis	Anemia, diabetes, candidiasis	Determine etiology of anemia (iron, folate, B_{12}), check riboflavin, modify food consistency and flavoring, evaluate glucose control
Angular cheilitis	Dry, cracked, fissured corner of the mouth	Dehydration, anemia, ill-fitting dentures (drooling)	Determine etiology
Candidiasis	White and/or red removable patches on the oral mucosa	Immunodeficiency, diabetes	Determine etiology
Difficulty chewing	Partial or complete edentulism, poor occlusion, ill fitting dentures	Cranial nerve disorders	Determine etiology, referral for medical nutrition therapy

Adapted from Touger-Decker R, Sirois D. *Support Line* 3: 1; 1996.

of oral complications and malnutrition due to the disease and associated metabolic, oral, GI, immune, psychosocial, and pharmacological sequellae. Macronutrient metabolism is often altered; women lose more fat tissue, further contributing to malnutrition, while men tend to lose lean tissue, again contributing to malnutrition and increased needs.[21] The psychological and physiological stress associated with HIV and AIDS contributes to alterations in nutrient intake and subsequent nutrition status.

Poor oral hygiene, malnutrition, and lack of dental care are key factors in the development and severity of oral lesions in this population. Candidiasis occurs in 80 to 100% of HIV patients with AIDS upon onset of the disease.[3] Periodontal disease including necrotizing ulcerative periodontitis (NUP) is common in patients with HIV. Cancers of the oral cavity, Kaposi's sarcoma, and lymphoma may also occur. Thus, oral challenges, combined with common nutrition problems such as wasting syndrome, visceral and somatic protein depletion, maldigestion and malabsorption, altered nutrient and energy needs, polypharmacy, and reduced oral food intake secondary to anorexia, nausea, or compromised oral health status further contribute to the malnutrition observed in this population.

The possible causes of anorexia in this population can be seen in Table 54.5. Medical Nutrition Therapy (MNT) by the Registered Dietician (RD) and physician is warranted to prevent further nutritional depletion, altered taste perception, odynophagia/dysphagia,

TABLE 54.5

Possible Causes of Anorexia in HIV/AIDS

Polypharmacy	Dysgeusia
Odynophagia/dysphagia	Nausea/vomiting
Depression	Weakness/lethargy
Oral infections/lesions	Tooth loss
GI disease	Kaposi's sarcoma

TABLE 54.6

Impact of HIV Infection on Nutrition and Diet in the Upper GI Tract

Location	Problem	Effect	Diet Management
Oral cavity	Candidiasis, KS herpes, stomatitis, apthous ulcers	Pain, infection, lesions, altered ability to eat, saliva; dysgeusia	Increase kcalories & protein Oral supplements Caries risk reduction
	Xerostomia	Caries risk, pain, no moistening power, food sticks, dysgeusia	Moist, soft foods; non-spicy, "smooth," cool/warm, fluids Caries risk reduction
Esophagus	Candidiasis, herpes, KS, Cryptosporidius	Dysphagia, odynophagia	Oral supp 1st, 2nd = NG using silastic feeding tube or PEG
	CMV CMV + ulceration	Dysphagia, food accumulation	Percutaneous endoscopic gastrostomy

nausea/vomiting, psychosocial considerations, weakness/lethargy, and the expanded use of other medications.

Wasting syndrome is characteristic of later stages of HIV and is defined as "a greater than (>)10% unintentional weight loss from usual body weight in the presence of diarrhea or fever for greater than (>) 30 days that is not attributable to other disease processes."[22] Wasting Syndrome is also defined as "a process of decline characterized by pathological alterations in body composition, weight loss, fatigue and loss of strength and a decrease in quality of life."[23] The pathogenesis of wasting can be attributed to several factors including decreased energy intake, anorexia, GI dysfunction, and deranged metabolism including increased insulin sensitivity, altered carbohydrate metabolism, and increased protein turnover. During acute phases of the disease, drugs and medical nutrition therapy are critical to manage the disease and preserve lean body mass. As individuals progress to lifelong drug management with combinations of antiretroviral therapies, nutrition status is challenged by the associated drug side effects. These side effects may include food-drug interactions, oral lesions, and infection as well as alterations in appetite and intake.

Table 54.6 outlines the impact of HIV infection in the GI tract. Common HIV-associated oral infectious disorders include those of fungal, viral, and bacterial origin, detailed in Table 54.7.[24] Oropharyngeal fungal infections may cause a burning, painful mouth and dysphagia. The ulcers found with viral infections such as herpes simplex and cytomega-

TABLE 54.7

Common HIV-Associated Oral Disorders

Infection	
Bacterial	Linear erythematous gingivitis, necrotizing ulcerative periodontitis, syphilis, caries
Fungal	Candida, cryptococcus, histoplasmosis
Viral	Herpes simplex, hairy leukoplakia, human papiloma
Neoplasm	
Fungal	Kaposi's sarcoma
Viral	Non-Hodgkin's lymphoma
Other	
Fungal	Parotid disease, xerostomia, apthous ulcers
Viral	Pain syndromes, necrotizing stomatitis

Adapted with permission from D. Sirois, *Mt. Sinai J. Med.* 65: 322; 1998.

TABLE 54.8

Oral Manifestations of Diabetes

Gingivitis	Altered taste
Periodontal disease	Burning mouth
Reduced saliva (with resultant xerostomia)	Increased thirst
Salivary hyperglycemia	Neuropathies
Increased risk of infectious disorders and complications	
Slowed wound healing	

lovirus cause pain and reduced oral intake. Oral and esophageal candidiasis results in painful chewing, sucking, and swallowing, consequently reducing an already compromised appetite and intake. Kaposi's sarcoma compromises oral intake and increases nutrient needs. The oral disorders found with HIV and AIDS increase nutrient demands on the body for healing, and compromise eating ability. MNT is critical to healing and maintenance of lean body mass. Oral diets with or without nutritional supplements should be tried first, followed by tube feedings if needed. The dental professional should observe patients for changes in intake, weight, and overall nutritional wellbeing, and refer individuals to an RD and/or physician early in the process of disease management.[25,26]

Diabetes

Over 11 million individuals in the U.S. have diabetes; another 13 million have impaired glucose tolerance.[27] Diabetes is the seventh leading cause of death in the U.S. Oral sequellae occurring with diabetes are usually a result of poorly controlled diabetes, or hyperglycemia. Oral cell metabolism, immune surveillance, and vascular integrity as well as salivary chemistry may be altered in individuals with diabetes, particularly when uncontrolled. Over 90% of individuals over age 13 with diabetes have some periodontal problems relative to diabetes.[28] While there is limited evidence that periodontal infection affects glycemic control,[29] any infection can contribute to adverse alterations in glycemic control. Other oral manifestations may occur, impacting diet intake and nutrition status. These can be seen in Table 54.8.

Diabetes, particularly when poorly controlled, is associated with a higher prevalence of all infections including oral infections, as compared to non-diabetics.[28] The susceptibility to periodontal disease in diabetes is likely directly related to impaired defense mechanisms. Micro- and macrovascular circulation are altered, along with wound healing, collagen metabolism, neutrophil chemotaxis, and proteolysis. Pathologic tissue destruction contributes to periodontal tissue destruction. Increased salivary glucose increases bacterial substrate and plaque formation. Microangiopathies, altered vascular permeability, and metabolic alterations may lead to an altered immune response and precipitate periodontal disease progression. The oral complications associated with diabetes are often referred to as the sixth major medical complication of diabetes.

The relationship between oral manifestations in diabetes and diet/nutrition is a complex two-way street. While uncontrolled diabetes is partly due to poor diet management, oral manifestations challenge and ultimately compromise eating ability and consequent dietary intake. Taste in patients with diabetes is often altered due to salivary chemistry, xerostomia, and/or candidiasis. Management of burning mouth/tongue requires a determination of the cause. When due to a nutrient deficiency, augmentation of the diet with the appropriate nutrient(s) or supplements will treat the cause and subsequent symptomatology. When a physical or biochemical abnormality cannot be found, the symptoms of burning mouth can be improved using tricyclic antidepressant medications. However, the unfortunate

side effect of tricyclic antidepressants is xerostomia, which may compound any existing alterations in saliva, and increase risk of candidiasis and dental caries.

Diet management is the cornerstone of diabetes care. Proper diet control, in kcalorie and macronutrient distribution throughout the day, is critical to glycemic control, particularly in type 1 diabetes. In both type 1 and type 2 diabetes, a diet consistent with the food guide pyramid consisting of 50% carbohydrate, 20% protein, and 30% fat is recommended with attainment and maintenance of desirable body weight. In most states, MNT by an RD with several follow-up visits is a benefit covered by all third party payers and Medicaid. The oral health professional should work closely with the patient's physician and refer patients, as appropriate, to an RD, reinforce the need to adhere to the diabetic diet, integrate oral hygiene into daily routines, and modify diet consistencies as needed to manage oral conditions and surgeries.

Crohn's Disease

Crohn's disease can present with apthous ulcers, angular stomatitis, and/or glossitis. Oral lesions present on the lips, gingiva, and buccal mucosa may precede gastrointestinal symptoms and have a dramatic impact on oral function. The size of the lesion may not coincide with the intensity of pain reported or degree of compromise in food and fluid intake. Ability to eat may be hampered by pain; topical anesthetic agents (or a 1:1 mixture of Milk of Magnesia and Benadryl as a rinse and spit) prior to meals may temporarily relieve pain and allow more comfort in eating. Pharmacological management is critical; steroids are often needed. The impact of steroids on nutritional wellbeing has been addressed in other sections of this handbook.

Autoimmune Disease

Autoimmune diseases such as Pemphigus Vulgaris (PV) increase nutrition risk by virtue of the oral and facial sequellae of the disease and the medications used to manage it. Steroids are often used in the management of vesiculobullous diseases of the oral cavity including PV. PV can impact diet intake and nutrition status by virtue of the disease process as well as the medications used to manage it. Much like other diseases with oral lesions, PV affects appetite and eating ability due to associated pain. In addition to the medications used for the disease, topical anesthetics, as well as a rinse-and-spit 1:1 mixture of Benadryl and Milk of Magnesia immediately prior to eating, provides a topical coating, allowing more comfort during mealtime. Other autoimmune diseases including Sjögren's Syndrome and rheumatoid arthritis (RA) can affect the oral cavity and subsequent nutrition status. Oral and nutritional implications of select diseases in this category are outlined in Table 54.9. While diet modifications are addressed, particular attention needs to be paid to the individual's stage of disease. During disease exacerbation, eating ability may be severely compromised and a liquid diet using oral supplements (meal replacement formulas) such as Sustacal or Boost (Mead Johnson) or Ensure+ (Ross) may be required to meet energy and protein needs. During remission, individuals may be able to liberalize diets considerably.

Osteoporosis

Osteoporosis is the most common bone disease.[30] Osteoporosis is clinically defined as reduced bone mineral density.[30] The majority of individuals with osteoporosis are women

TABLE 54.9

Autoimmune Disorders with Associated Oral and Nutritional Side Effects

Disease	Oral Manifestation	Nutrition Implications
Rheumatoid arthritis	TMJ ankylosis Limited mandibular opening	Pain upon eating Modify diet consistency
Erythema multiforme	Oral mucosal lesions, often ulcerative	Pain upon eating Increased needs for healing Modify diet, often liquid consistency with straw during exacerbation If treated with steroids: increase calcium, protein
Pemphigus Vulgaris	Oral mucosal lesions	Eating painful and difficult If treated with steroids: increase calcium, protein Modify diet as needed in temperature, consistency
Sjögrens syndrome	Xerostomia; mucosa dry, erythematous; fissures; more susceptible to trauma; increased risk of caries; fissured tongue; periodontal disease; cervical caries	Increased fluids with and between meals, increase fluidity of foods, soft, temperate foods, non-spicy
Systemic lupus erythematosus	Ulcerations of mucosa	Manage side effects of steroids and medications, diet as per Sjögrens

(80%); however it is a risk for up to 28 million Americans, and is often known as a "silent killer."[1] One in two caucasian women will have an osteoporotic fracture in their lifetime.[30] Nonmodifiable and modifiable causes of osteoporosis are listed in Table 54.10. Since dental health care professionals see patients on a regular basis and since alveolar boss loss is associated with osteoporosis, the dental professional is in an ideal situation to access patients at risk for osteoporosis. Simple screening in the dental office can include asking patients about calcium intake in the form of dairy products (milk, cheese, yogurt), functional foods or foods fortified with calcium (orange juice, cereal), and calcium supplements. Other individuals at risk for osteoporosis (see Table 54.10) should be referred for a dexascan to determine risk for or presence of osteopenia or osteoporosis.

Bone loss is a common denominator for both periodontal disease and osteoporosis. The relationship between the two diseases remains to be fully determined.[31] However, an association between periodontal disease and systemic osteopenia and osteoporosis has been documented in adults.[31,32] The relationship between calcium intake and risk of periodontal disease has also been demonstrated; lowered dietary intakes in adults have been found to be associated with increased periodontal disease risk.[11] While a serum calcium level may not be associated with actual calcium intake, decreasing levels of intake below the recommended dietary intakes is associated with increased risk and incidence of osteoporosis.[33] Tezal et al. pose "four possible pathways" for the relationship between osteoporosis and severity of periodontal disease, including systemic loss of bone mineral density, modified local tissue response to infection as a result of "systemic factors of bone remodeling", genetics, and lifestyle factors.[31] The final pathway, lifestyle factor, links the issues of hygiene, diet, and exercise with local and systemic disease. Osteoporosis has also been linked to the development of tooth loss, particularly in post-menopausal women.[10] Please refer to Section 61 for a thorough description of osteoporosis and other bone diseases and disorders.

TABLE 54.10

Risk Factors for Osteoporosis

Nonmodifiable
History of fracture as an adult
Caucasian or Asian race
Advanced age
Female gender
Dementia
Poor health/frailty
Prolonged use of glucocorticoids, phenytoin
Estrogen deficiency:
Menopause/early menopause (<age 45) or bilateral ovariectomy; prolonged premenopausal amenorrhea (>1 year)
Prolonged immobilization, i.e., spinal cord injury
Potentially Modifiable
Current cigarette smoking
Low body weight (<127 lbs)
Low calcium intake (lifelong)
Alcoholism
Impaired eyesight despite adequate correction
Recurrent falls
Inadequate physical activity
Poor health/frailty
End-stage renal disease

From National Osteoporosis Foundation, *Physicians Guide to Prevention and Treatment of Osteoporosis,* 1998. With permission.

Neoplastic Diseases: Oral Cancer

Oral cancer develops from a precancerous lesion most commonly on the tongue, lips, and floor of the mouth. White leukoplakia or reddish erythroplakia are usually induced by tobacco use alone or in combination with alcohol abuse.[34,35] It is the sixth most common cancer in males living in the U.S. Over 90% of oral cancers are squamous cell carcinomas — cancers of the epithelial cells. Tobacco components act as promoters of carcinogenesis, and alcohol may act as a solvent to facilitate penetration of the tobacco carcinogens into oral tissue.[36] Viruses, including the herpes simplex type 1 and human papilloma virus, have both been implicated in oral cancer. Of the known oncogenes, many have been implicated in oral and pharyngeal cancer.[37] Both disarmament of the cell's DNA repair mechanisms and mutation of tumor suppressor genes have been linked to smoking and alcohol use, and play a major role in oral cancer development. Likewise, nicotine stimulates negative changes in immune cells that can promote tumor growth.

Ecologic and case-control studies have suggested that nutrients may play an important role in the prevention and management of early oral cancer and precancerous leukoplakia. Likewise, several studies have shown that smokers have lower plasma levels of vitamin E, C, beta-carotene, and other antioxidants due to both low intake and increased metabolic use.[38] Total fruits and vegetables, fresh fruits, green leafy vegetables, other vegetables, total bread and cereals, and whole grain breads and cereals which are excellent sources of multiple antioxidants have been shown to be associated with decreased risk of oral

cancer.[39] More specifically, citrus, dark yellow and other fruits were more consistently associated with decreased risk than were estimated intake of specific nutrients including carotene, vitamin C, fiber, folate, thiamine, riboflavin, niacin, vitamin E, and iron. A case-control study conducted in Italy illustrated that the more a micronutrient, such as vitamin C, carotene, or vitamin E, was correlated to total vegetable and fruit intake, the stronger was its protective effect against oral cancer.[40] Thus, it appears that total fruit and vegetable intake may offer greater risk reduction than singular antioxidant nutrients in supplemental doses. Fioretti et al. reported that even in the absence of tobacco use, if subjects reduced alcohol and saturated fat intake and increased fruit and carrot consumption, there appeared to be a favorable effect on oral cancer risk among subjects who participated in a large case-control study.[41]

Nutritional care of the patient with oral cancer will vary based on treatment modalities and side effects that can include changes in weight, sore mouth or throat, xerostomia, mucositis, dental caries, gingival infection, changes in sense of taste and smell, nausea, vomiting, and fatigue. Nutritional management strategies will require the services of oncology health care professionals.

Craniofacial-Oral-Dental Birth Defects

Nearly two infants in every 1000 live births have some type of craniofacial birth defect.[42] These defects can occur in isolation or as a component of a larger birth defect syndrome caused by genetic influences, environmental disturbances, or the interplay between gene and environment. A number of congenital oral-dental-craniofacial birth defects can be prevented by reducing risk factors associated with human craniofacial malformations. Among the common environmental risk factors are alcohol (associated with fetal alcohol syndrome); smoking (associated with risk of cleft lip with or without cleft palate); anti-convulsant medications, such as phenytoin and other teratogens (associated with a variety of birth defects involving the face, teeth, and jaws); retinoic acid analogues (associated with severe craniofacial and oral clefts and limb defects); and vitamin deficiency, particularly folate (associated with increased risk of cleft lip with or without cleft palate).[42]

Available data from the NIDCR revealed that about 20% of craniofacial-oral-dental birth defects are either genetic or familial.[42] The largest majority are caused by the defined risk factors noted above or unknown causes. From a nutrition perspective, there are a number of important discoveries that could be applied to oral-dental-craniofacial defects. One application relates to the preventive effects of micronutrients. A growing body of evidence strongly suggests that the use of multivitamin supplements and folic acid result in the prevention of cleft lip and/or cleft palate in much the same way that these micronutrients work to prevent neural tube defects such spina bifida. As these studies progress, it will become important to carefully titrate the dose of the micronutrient provided to an expectant woman that will result in a maximum protective effect. This is vital because there is a possibility that excessive amounts of some nutrients, such as vitamin A or retinoic acid, can result in the opposite effect; that is, they could be teratogenic.[42,43] Retinoic acid-induced embryopathy includes defects in craniofacial, skeletal, cardiac, thymic, and central nervous system structures.[42,43]

The protective molecular mechanism for retinoic acid-induced embryopathy relates to the binding of retinoic acid to cognate receptors that, in turn, bind to unique regions within the promoter region of structural and/or regulatory genes.[44] Indeed, the expression

of the homeobox gene, Hoxb-1, is highly responsive to vitamin A. Since Hoxb-1 regulates embryonic axial patterning, excess vitamin A induces altered pattern formation with apoptosis.[44] Thus, folic acid and vitamin A represent excellent examples of the exquisite sensitivity of embryogenesis, and the craniofacial complex in particular, to nutrients. A critical message for the oral health practitioner is the necessity to work closely with the nutritionist and physician to educate prospective and expectant parents regarding nutrition and oral health.

Importantly, following the birth of a "cleft" child, depending on the extent and severity of clefting, a series of physiological, psychological, medical, dental, and social issues emerges. The establishment of a craniofacial anomaly or cleft team to manage patients with clefting and/or other craniofacial defects is ideal. This multidisciplinary team assesses the child and his/her various medical, nutritional, social, and psychological needs. Often a nutritionist/dietician is an integral part of such a team, since one of the major issues to overcome for such children is the ability to ingest adequate amounts of food and nutrients consistent with increased nutrient requirements of the early developmental years. These nutrition requirements are exacerbated by multiple surgical interventions and the extent of the defects. Therefore, craniofacial anomalies present two challenges for the health care professional and the nutrition community in particular. One is to identify women at risk for birth defects and use appropriate interventions to prevent craniofacial anomalies. The second is to work closely in an interdisciplinary team to mitigate the effects of the anomaly.

Health Promotion, Health Education, and Behavior Change

Health promotion is a term used to describe not only heath education but also organizational, economic, and environmental channels that provide support for enabling people to increase control over and improvement of their health.[45,46] Dental nutrition is an integral part of general health promotion and disease prevention.

According to health promotion theoretical models, personal health practices and behaviors that enhance lifestyle lead to reduced morbidity and mortality, improved health status, and improved quality of life. Frequently, oral diseases in children are associated with eating difficulty, general health problems, and even lost school time.[47] In aging populations strong associations exist between oral health status and ability to swallow, chew efficiently, and select a variety of foods.[48] Numerous nutrition and dietary practices enhance positive oral health outcomes throughout the life span. Thus, it is imperative that dietary intake provide adequate nutrients to support oral health and function.

Nutrition education programs focused on changing dietary behaviors should include oral health promotion within the Food Guide Pyramid and U.S. Dietary Guidelines messages.[49] Table 54.11 describes global nutrition messages appropriate for inclusion in oral health promotion programs targeting primary and secondary prevention of dental caries. Table 54.12 identifies messages appropriate for early childhood caries (ECC) prevention. Effort has been made to reduce ECC, particularly in high-risk populations that include minority and low socioeconomic groups. Unfortunately, the nutrition focus has been limited to infant feeding practices, promoting early weaning with transition from bottle to cup and the impression that milk sugar (lactose) is the primary culprit. Sucrose, glucose, and fructose found in fruit juices and drinks as well as sweetened solid foods are probably the main sugars associated with ECC.[55] Due to the casein as well as calcium and phosphate

TABLE 54.11

Global Nutrition Messages Addressing Primary and Secondary Prevention of Dental Caries

Message	Rationale
Eat a balanced diet representing moderation and variety	Focus on positive aspects of healthy eating. Fermentable carbohydrates can contribute to dietary intake and be consumed in moderation.
Combine and sequence foods to encourage chewing and saliva production	Combinations of raw and cooked foods can increase saliva flow. Protein-rich foods combined with cooked carbohydrates, and dairy foods combined with fermentable carbohydrates can alter dental plaque pH. (Rugg-Gunn, 1993)[50]
Space the frequency of eating or drinking fermentable carbohydrates at least two hours apart	It may take up to 120 minutes for dental plaque pH to return to neutral after exposure to fermentable carbohydrate. (Edgar, 1996)[51]
Chew sugarless gum after meals and snacks to increase saliva	The Food and Drug Administration authorized the use of sugar alcohol containing foods to be labeled "does not promote," or "useful in not promoting," or "expressly for not promoting" dental caries if the food does not cause a drop in dental plaque pH below 5.7 when a fermentable carbohydrate is present. (U.S. Food and Drug Administration, 1996)[52]
Drink water to satisfy thirst and hydration needs	A review of fermentable carbohydrate consumption in the U.S. identified carbonated beverages as the major contributor to total intake. (Gibney, 1995)[53]

content, milk formulas (with the exception of some soy-based and protein hydrolyzed formulas), bovine milk, and human milk may indeed be cariostatic and not a source of cariogenic substrate in ECC. However, the nursing bottle can effectively block salivary access to tooth surfaces and may increase the caries-promoting potential of any food that remains in the mouth.[56] Reisine and Douglass have suggested that psychosocial and behavioral issues related to elements of the environment may be greater modulators of ECC than the baby bottle.[57]

Numerous opportunities exist for nutrition messages to be included in oral health promotion initiatives. Table 54.13 lists a variety of health topics that impact oral health outcomes and status. With the advent of The Surgeon General's Report on Oral Health, attention has been drawn to the need for health care professionals to address oral health and systemic health as one entity: health![1] This list in Table 54.13 provides insight into the aforementioned synergy between oral and nutritional status, and offers targeted messages for health promotion in public health and private practice.

Better understanding of the role of behavioral variables in health and disease, including the role of prevention, is important in the delivery of messages that promote both nutritional and oral health status. Practices that attempt to translate the scientific discoveries in nutrition and oral health will provide a basis upon which to plan and execute future health promotion activities.

TABLE 54.12

Nutrition Messages to Integrate into Parenting Practices for Primary Prevention of ECC

Message	Rationale
Birth to 6 Months of Age	
Encourage feeding schedules that encourage breast milk and formula consumption on a regular routine basis rather than continuously on-demand.	The American Academy of Pediatrics (AAP) encourages breastfeeding on demand in response to signs of hunger, described as increased alertness or activity, mouthing or rooting. The American Academy of Pediatric Dentistry (AAPD) recommend that infants not be put to sleep with a bottle and that nocturnal breastfeeding should be avoided after the first primary tooth begins to erupt.
Instruct mothers to avoid introduction of food until the infant doubles the birth weight or weighs at least 13 pounds.	The AAP recommends breastfeeding exclusively for about the first six months after birth, after which time iron-enriched solid foods can be added to complement the breast milk diet. Infants will double their birth weight or reach 13 pounds at between 4 and 6 months of age, or approximately 5 months.
Hold the infant when bottle and/or breast feeding.	This will prevent bottle propping, prolonged exposure to caries-promoting substrate, and allowing infants to drink from bottles on a continuing basis.
6 to 12 Months	
Promote weaning from the bottle in combination with the introduction of a cup and spoon.	The AAPD encourages parents to have infants drink from a cup prior to their first birthday and be weaned from the bottle at 12-14 months of age.
Promote introduction of foods to encourage self-feeding and growth and development as well as dental health.	The AAPD recommends implementation of oral hygiene measures by the time of eruption of the first primary tooth.
1 to 16 Years of Age	
Promote snacking habits that support growth and development and dental health.	The AAPD endorses the Dietary Guidelines for Americans that promote variety, a healthy weight, a diet that includes vegetables, fruits, and grains, and use of sugars in moderation for children and adults.
Advocate discontinued bottle and breast feeding practice.	
Stress the value of mealtime and the importance of variety and moderation.	
Encourage the beginning of routine dental visits for the child.	The AAPD recommends an oral evaluation visit within six months of the eruption of the first tooth and no later than twelve months of age.

American Academy of Pediatrics, *Pediatrics* 11: 1035; 1997.

Journal of The American Academy of Pediatric Dentistry Reference Manual 1996-97. *Pediatr. Dent.* 18: 25; 1996.

TABLE 54.13

Nutrition Messages for Targeted Oral Health Promotion Topics

Oral Health Promotion Topics	Nutrition Messages
Hypomineralized or hypoplastic primary teeth and caries risk	Children who are malnourished pre-, peri-, or postnatally and/or low birthweight are more likely to have this condition. (Alvarez 1995, Lai 1998)[58,59]
Craniofacial development	Causes are attributed to genetic defects often working in concert with environmental factors such as alcohol intake and possibly excessive therapeutic vitamin A. Neural tube defects and risks of cleft lip and palate may be reduced in children if women support dietary folic acid intake with additional supplementation to equal 400 µg. (Bonin 1998)[60]
Bone status	Loss of teeth leads to bone atrophy. Localized diseases like periodontal disease and systemic diseases like osteoporosis may further affect alveolar bone loss. Promotion of diets adequate in calcium and vitamin D to target these effects should be discussed in the context of oral health. (Bhaskar 1991)[61]
Oral soft tissue integrity	Nutritional status can enhance the ability of healthy epithelial tissue to prevent penetration of bacterial endotoxins into gingival tissue. Protein, vitamins A, C, and E, as well as the B-compex vitamins and zinc will help to maintain immune system integrity, but there is a paucity of scientific data to support supplemental use of these nutrients. Prevention of diseases of the soft tissue related to diseases like periodontal disease and systemic diabetes may challenge utilization of nutrients and can increase risk of decreased oral soft tissue integrity. (Touger-Decker 2000)[62]
Salivary output	Saliva and salivary glands provide clues to overall health and disease and function in the mucosal immune system to protect oral tissue integrity. Saliva moistens food and lubricates the bolus for swallowing. Fiber intake and frequency of eating can promote salivary output. Xerostomia (dry mouth) associated with disease and drug therapies may require medical nutrition therapy. (Martin 1999)[48]
Edentulous state	Toothless persons or those who wear dentures need to be encouraged to modify food selection habits and method of preparing foods for easier biting and chewing. One can still achieve good nutritional status important to the maintenance of the oral tissue.
Oral cancer prevention	Promoting five or more servings a day of fruits and vegetables from a variety of sources that include both dark green and yellow sources may decrease risk for oral cancer. Weight management strategies for those interested in smoking cessation can possibly enhance success and should be explored when appropriate. (Jones and Mobley 2000)[63]
Dental erosion	Fruit juices, citrus fruits, acid sweet candies and mints, pickles, and cola drinks can cause loss of tooth enamel. Vomiting and regurgitation associated with gastroesophageal reflux and eating disorders can also cause this dental condition. Encourage dietary practices to neutralize the impact of these products and conditions. (Rugg-Gunn 1993)[50]

References

1. U.S. Department of Health and Human Services. *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, 2000.
2. Slavkin H, Baum BJ. *JAMA* 284: 1215; 2000.
3. National Institute of Dental and Craniofacial Research. *Burden of Disease.* www.nidcr.nih.gov/discover/ctfy2001, accessed July 26, 2000.
4. Kaste LM, Selwitz RH, Oldakowski RJ, et al. *J Dent Res* 75: 631; 1996.
5. Winn DM, Brunell JA, Selwitz RH, et al. *J Dent Res* 75: 642; 1996.
6. Feathersone JDB. *J Am Dent Assoc* 131: 887; 2000.
7. Winston AE, Bhaskar SN. *J Am Dent Assoc* 129: 1579; 1998.
8. Page RC, Kornman KS. *Periodontology 2000* 14: 9; 1997.
9. American Dietetic Association. Position of The American Dietetic Association: Oral Health and Nutrition. *J Am Diet Assoc* 96: 184; 1996.
10. Krall EA, Garcia RI, Dawson-Hughes B. *Calcif Tissue Int* 59: 433; 1996.
11. Nishida M, et al. *J Periodontal* 71: 1215; 2000.
12. Leggot PJ, et al. *J Dent Res* 70: 1531; 1991.
13. Saito T, et al. *N Engl J Med* 339: 482; 1998.
14. Achievement in public health, 1900-1999; *MMWR Weekly* 48: 933; 1999.
15. Horowitz HS. *J Public Health Dent* 56: 253; 1996.
16. Newbrun E. *J Public Health Dent* 49: 279; 1989.
17. American Dental Association. *J Am Dent Assoc* 126: 15; 1995.
18. American Academy of Pediatrics. *Pediatrics* 11: 1035; 1997.
19. Leverett DH, Vaughn BW, Adair SM, et al. *J Public Health Dent* 53: 205A; 1993.
20. Clarkson HB, Fejerskov O, Ekstrand et al. In: *Fluorides in Dentistry.* Fejerskov O, Ekstrand J, Burt BA, Eds, 2nd ed. Copenhagen: Munksgaad; 1996, p 347.
21. American Dietetic Association. *J Am Diet Assoc* 100: 708; 2000.
22. Centers for Disease Control. *MMWR* 41: 1; 1992.
23. Kotler D, Tierney AR, et al. *Am J Clin Nutr* 57: 1; 1989.
24. Sirois D. *Mt Sinai J Med* 65: 322; 1998.
25. Touger-Decker R. *Mt Sinai J Med* 65: 355; 1998.
26. Touger-Decker R, Sirois D. *Support Line* 3: 1; 1996.
27. National Institute of Dental and Craniofacial Research. *Workshop on Oral Disease and Diabetes,* December 6-7, 1999.
28. Baron S. Bacterial Infection in Diabetes. *Workshop on Oral Disease and Diabetes,* National Institute of Dental and Craniofacial Research, December 6-7, 1999.
29. Taylor G, Burt B, Becker M, et al. *J Periodontol* 67: 1085; 1996.
30. Osteoporosis Coalition of New Jersey and the New Jersey Department of Health and Senior Services. *Recommended Practice Guidelines for the Diagnosis and Treatment of Osteoporosis.* Washington Crossing PA: Scientific Frontiers Inc, 1998.
31. Tezal M, Wactawski-Wende J, Grossi SG. *J Periodontol* 71: 1492; 2000.
32. Payne JB, Reinhardt RA, et al. *Osteoporosis Int* 10: 34; 1999.
33. National Osteoporosis Foundation, *Physicians Guide to Prevention and Treatment of Osteoporosis.* Washington DC: National Osteoporosis Foundation, 1998.
34. Mashberg A. *J Am Dent Assoc* 96: 615; 1978.
35. Shklar G. *N Engl J Med* 315: 1544; 1986.
36. Blot WJ, McLaughlin JK, Winn DM, et al. *Cancer Res* 48: 3282; 1988.
37. Spandidos DA, Lamothe A, Field JK. *Anticancer Res* 5: 221; 1985.
38. Handelman GJ, Packer L, Cross CE. *Am J Clin Nutr* 63: 559; 1996.
39. Marshall JR, Boyle P. *Cancer Causes Control* 7: 101; 1996.
40. Negri E, Franceschi S, Bosetti C, et al. *Int J Cancer* 86: 122; 2000.
41. Fioretti F, Bosetti C, Tavani A, et al. *Oral Oncology* 35: 375; 1999.

42. Slavkin H. Meeting the Challenge of Craniofacial-Oral-Dental Birth Defects, *J Am Dent Assoc* 127: 681; 1996.
43. Slavkin H. *Prospects for Dental, Science, Education and Practice in the 21st Century.* 2nd Asia Pacific Congress, Japan, May 2000. www.nidcr.nih.gov/discover/slides.
44. Slavkin H. *Nutrients and Micronutrients: Progress is Science-Based Understanding, Insights on Human Health*, NIDCR; 1-11, 1998. www.nih.nidcr.gov.
45. Green LW, Kreuter MW. *Health Promotion Planning: An Educational and Environmental Approach.* 3rd ed. Mountain View CA: Mayfield Publishing Company, 1999.
46. Epp L. Achieving Health of All: A Framework for Health Promotion in Canada. Toronto: Health and Welfare Canada, 1986.
47. Edmunds M, Coye MJ, Eds. *America's Children: Health Insurance and Access to Care.* Committee on Children, Health Insurance and Access to Care, Division of Health Care Services, Institute of Medicine. Washington: National Academy Press, 1998.
48. Martin WE. Oral health in the elderly. In: *Geriatric Nutrition; The Health Professional's Handbook.* 2nd ed. Chernoff R, Ed, Aspen Publishers, Gaithersburg, 1999, p 107.
49. Nutrition and Your Health: Dietary Guidelines for Americans. 5th ed, Washington, DC, U.S. Depts of Agriculture and Health and Human Services, *Home and Garden Bulletin*, No. 232, 2000.
50. Rugg-Gunn AJ. *Nutrition and Dental Health*. Oxford: Oxford University Press, 1993.
51. Edgar WM, O'Mullane DM. *Saliva and Oral Health*. London: British Dental Association, 1996.
52. United States Food and Drug Administration. Health claims: dietary sugar alcohols and dental caries, *Fed Reg*, 43433, August 23, 1996.
53. Gibney M, Sigman-Grant M, Stanton Jr, JL, et al. *Am J Clin Nutr* 62: 178S; 1995.
54. Journal of The American Academy of Pediatric Dentistry Reference Manual 1996-97. *Pediatr Dent* 18: 25; 1996.
55. Seow WK. *Community Dent Oral Epidemiol* 26: 8; 1998.
56. Bowen WH. *Community Dent Oral Epidemiol* 26: 28; 1998.
57. Reisine S, Douglass JM. *Community Dent Oral Epidemiol* 26: 32; 1998.
58. Alvarez JO. *Am J Clin Nutr* 61: 410S; 1995.
59. Lai PY, Seow WK, Tudehope DI, et al. *Pediatr Dent* 19: 42; 1997.
60. Bonin MM, Bretzlaff JA, Therrien SA, et al. Northeastern Ontario Primary Care Research Group, *Arch Fam Med* 7: 438; 1998.
61. Bhaskar SN. In: *Orban's Oral Histology and Embryology.* Bhaskar SN, Ed, St Louis, Mosby Yearbook; 1991, p 239.
62. Touger-Decker R. In: *Krause's Food, Nutrition and Diet Therapy.* 10th ed. Mahan LK, Escott-Stump S, Eds, WB Saunders: Philadelphia, 1999, p 633.
63. Jones DL, Mobley CC. *Tx Dent J* 26; 2000.