

Dent Clin N Am 47 (2003) 319-336

THE DENTAL CLINICS OF NORTH AMERICA

Nutrition and dental caries

Connie C. Mobley, PhD, RD

Department of Community Dentistry, University of Texas Health Science Center at San Antonio Dental School, Mail Code 7917, 7703, Floyd Curl Drive, San Antonio, TX 78229-3900, USA

Dental caries is a diet-dependent infectious disease primarily attributed to the presence of oral bacteria. The prevalence and progression of this disease are further influenced by secondary factors including saliva, fluoride, and the anatomic integrity of the tooth enamel [1]. Prevention and management of dental caries are dependent on a clear understanding of the dynamics of the multifaceted variables that determine not only the initiation of the disease but also its course over an extended period of time.

Dental professionals have had for some time a clear understanding of the biologic activity associated with dental caries [2]. Certain oral bacteria are known to readily produce organic acids from metabolism of fermentable dietary carbohydrates in the oral cavity. Bacteria synthesize insoluble plaque matrix polymers or extracellular dextran that serves to perpetuate bacterial colonization on the surface of the tooth. The resulting acidic environment or low pH in dental plaque is an ideal environment for these bacteria. Thus, the organic acids contribute to the demineralization of the tooth surface proportionally to the bacterial colonization and activity [3]. Eating patterns can enhance or promote the caries process or interfere with and depress this activity. The purpose of this article is to examine those dietary factors involved in dental caries promotion and those that are involved in caries prevention. Dietary and nutrition education appropriate for dental settings are an essential component of guidelines or standards of practice that determine successful management of dental caries and the patient's quality of life across time.

Synergy between diet and dental caries

Nutrition systemically influences teeth during the pre-eruptive stage, including prenatal, perinatal, and postnatal periods. Malnutrition, in the

E-mail address: mobleyc@uthscsa.edu

form of deficiencies of protein energy and vitamins D and A, has been associated with enamel hypoplasia and related increases in the susceptibility of the tooth to caries lesions [4,5]. One episode of mild-to-moderate malnutrition in the first year of life was associated with increased incidence of caries in deciduous and permanent teeth later in life [4]. These deficiencies were also associated with salivary gland atrophy and reduced saliva that is known to alter dental plaque acid activity [5]. Diet more specifically describes foods and foodstuff (alone or in combinations) and the pattern of consumption over a given period of time. It is the local or topical effect of the diet on the tooth surface that is of greatest interest in examining caries, a pathologic disease process resulting from bacterial and dietary interactions [6]. Therefore, caries is primarily associated with dietary choices but may be secondarily influenced by nutritional status that may vary over the course of the life span.

Formation of a salivary pellicle on the tooth surface supports the initiation of dental plaque, the first overt oral clinical evidence of the interaction between diet and oral bacteria. Over time, fermentable carbohydrate metabolism supports the colonization of the bacteria on the tooth surface and orchestrates changes in an acidic-basic milieu [7]. More specifically, it is bacteria from the mutans streptococci and lactobacilli species that are the most prolific producers of the lactic acid responsible for freeing calcium and phosphate ions from the tooth enamel or dentin, leading to caries lesions [3]. The proportions and numbers of acid-base-producing bacteria can alter the impact of what is the demineralization-remineralization equilibrium of the tooth surface [3]. At a critical pH of approximately 5.5, demineralization occurs, especially when this acidic environment is maintained over time. When the pH exceeds 5.5 for a significant time, remineralization of the enamel can repair or reverse the initial surface damage but at a much slower rate than that of demineralization. Specific food groups, food ingredients, and dietary patterns may influence the strength of the direction that this dynamic process maintains over time. As shown in Fig. 1, other factors including saliva, fluoride, anatomic tooth structure, oral hygiene practices, time, aging, and changes in health status may also manipulate the balance that can be sustained. These variables are not discussed in detail in this article.

Saliva provides lubrication, promotes oral clearance of fermentable carbohydrates and acids, and is a source of antimicrobial components and buffering agents [8,9]. Furthermore, saliva encourages tooth remineralization by serving as a source of calcium, phosphate, and proline-rich proteins active in recrystalization of the tooth surface [8]. Aging, health status, and iatrogenic changes can alter saliva production and composition and, thus, caries-risk status [9]. Xerostomia, or dry mouth, is associated with increases in the number of cariogenic bacteria and increased caries [9]. Topical sources of fluoride further enhance remineralization by facilitating the adsorption of minerals while reducing rates of demineralization by plaque acids [10–12].

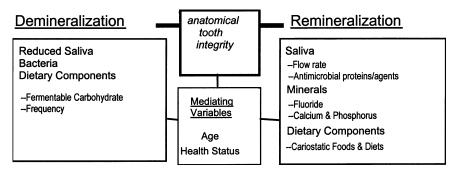


Fig. 1. Factors in demineralization and remineralization that are dynamic in the caries process.

In summary, the balance between demineralization and remineralization is dependent on not just one factor but on multiple factors [13]. A deficiency in base formation associated with inadequate saliva production can be as important in dental caries development as excessive formation of acid from fermentable carbohydrate associated with the diet [3]. An oral environment enriched with fermentable carbohydrate from the diet can change the microflora of the mouth from a noncariogenic state to a cariogenic state. Both the right substrate and the right bacteria must be present to produce high levels of acid required for caries initiation and progression.

Dental caries is a major cause of tooth loss in the United States. Nearly 20% of young children, almost 80% of young adults, and approximately 95% of older adults have experienced dental decay [14,15]. There is an erroneous impression that by just admonishing "sugar" from the diet and inculcating oral hygiene and optimum fluoride exposure, teeth will be protected from bad dietary habits [16]. Comprehensive caries management not only can include fluoride therapy and oral hygiene but must also address dietary behaviors that alter the availability of fermentable carbohydrate substrate to acidogenic bacteria and simultaneously enhance satisfactory saliva production [1,12,17].

Dietary factors in dental caries promotion

Specific foods, their unique forms, and the frequency of intake are dietary factors associated with caries.

Specific foods

Sugars [18,19] and other fermentable carbohydrates listed in Table 1 are fundamental to dental caries generation, whereas other food components have varying ancillary effects. Evidence from both observational and intervention human studies [20–27], animal studies [28], and laboratory Table 1

Caries-promoting potential (CPP) of sugars, other fermentable carbohydrates, and high-intensity sweeteners

Categories and names	Dietary examples	CPP	Details
Simple sugars Disaccharides = 2 monosaccharides Sucrose = glucose + fructose Maltose = glucose + glucose	Dextrin, corn syrup, fruit sugar, levulose, table, brown, and powdered sugar, turbinado (raw sugar), high-fructose corn syrup, molasses, honey	Yes	Carbonated and bottled drinks, fruits, vegetables, and processed foods with added sugars (eg, cakes, cookies, pies, sweetened canned foods, candies) are sources of simple sugars.
Lactose = glucose + galactose	Milk sugar	Low	Galactose has not been shown to have cpp.
Other fermentable carbo Polysaccharide = starch	ohydrates Cooked potatoes, rice, legumes, grains, cornstarch, and bananas	Yes	Gelatinized starch results from manipulation and processing of these whole-food examples.
Other nonfermentable c Fiber Polyolsaccharides (sugar alcohols)	arbohydrates Cellulose, pectin, gums Sorbitol, mannitol, xylitol Lactitol, isomalt, maltitol Hydrogenated starch hydrolysates	No	Grains, fruits, and vegetables are good sources of fiber. Sugar alcohols are 30%–90% as sweet as sucrose and, by weight, are a source of fewer kilocalories.
High-intensity sweetener Nutritive	rs Aspartame	No	Chemical compounds that are used as food additives in desserts, beverages, and confections that are 200–700 times sweeter than sucrose.
Nonnutritive	Saccharin, acesulfame, sucralose		

studies [29,30] of oral bacteria and dietary substrates have collectively supported the role of dietary sugars in the etiology of dental caries. Sugars consumed alone are readily fermented by oral bacteria to acids and can cause a rapid drop in dental plaque pH. The classic Stephan curve exemplifies the rapid drop in plaque pH in response to a 10% sucrose solution, followed by at least a 40-minute period before returning to a neutral pH of 7 [31]. Simply put, if fermentable carbohydrates were never present in the oral cavity, then there would likely be no caries. Bacteria are unable to replicate and generate acid without adequate substrate; however, investigators have shown that the amount of acid produced does not correlate linearly with the fermentable carbohydrate content of foods [32]. A complexity of the multiple variables in this process prevents concise interpretations that include all the factors.

Associations between total sugar consumption and dental caries in both developed and developing countries are inconsistent [33]. Using population data on per capita consumption of sugar, some investigators have identified positive correlations with the prevalence of caries and others have data that refute these findings [34,35]. In the United States, total sucrose consumption decreased between 1970 and 1996, but total added-sugar consumption increased because of dramatic increases in supplies of corn sweeteners or high-fructose corn sweeteners [36]. Soft drinks or sodas are the major source of added sugars in the American diet [37]. The classic Vipeholm Dental Caries Study [38], conducted between 1945 and 1954, reported that increased sugar consumption resulted in increased caries incidence. More specifically, consumption of retentive forms of sugar (8 to 24 toffee candies per day) between meals rather than with meals was associated with caries. Sweetbread and/or sugar solutions (30 g to 300 g carbohydrate per day) consumed at mealtime up to a maximum of four times a day had little effect on caries increments when snacking between meals was not allowed. Conclusions were that the combined quantity and frequency of sugar consumption contributed to caries risk and that consistency of the sugarcontaining foods was important [39].

A recently conducted systematic review of scientific articles examining sugar and caries risk [22] noted sugar consumption as a moderate risk factor for caries in most people with consistent exposure to fluoride. Studies were primarily cross-sectional in nature, had poor study designs in some cases, and used a variety of narrowly defined dietary assessment methods that did not identify sugar exposures over time. Based on these data, the reviewers concluded that for those who do not have regular exposure to fluoride, sugar consumption is probably a more powerful indicator of risk [22].

Starches cannot directly serve as substrate for oral bacterial fermentation [40,41]. Grains and vegetables such as potatoes, wheat, and beans contain starch granules that are damaged when subjected to heat and mechanical forces, leading to the formation of gelatinized starch. Through further hydrolysis by salivary and bacterial amylases, maltose and maltotriose

become available as substrate for plaque bacteria and acid production [40,42,43]. The bioavailability and cariogenicity of food starches in the mouth vary with the basic genetic character and different cooking and food-processing methods (frying, boiling, and so forth) [44]. Starchy foods such as untreated whole grains and raw vegetables have lower caries-promoting potential [45] than heat-processed foods such as white breads, crackers, chips, and dry cereal snacks.

Unique forms of food

The term *sugar* is technically a term to describe two classifications of carbohydrate (see Table 1): monosaccharides, or simple sugars (glucose, fructose, galactose), and disaccharides, or two simple sugars linked together (sucrose, lactose, maltose). Nutritive (sources of energy) sweeteners such as table sugar, honey, molasses, high-fructose corn syrup, dextrose, and sugar alcohols (alcohol forms of monosaccharides) are extrinsic and added to foods and beverages [18]. Acidogenicity and potential cariogenicity of sucrose, glucose, fructose, and maltose are similar; however, lactose is less cariogenic [42]. Grains, fruits, and vegetables that occur naturally are sources of intrinsic sugars encapsulated in foods that include other protein, nondigestible fiber, and fatty acid constituents [46]. There is lack of evidence to suggest these intrinsic sugars support the caries process.

Since the mid-1980s, little has been done to explore the cariogencity of foods. Artificial scientific methods to measure fall in dental plaque pH after exposure to food substrates are the basis of studies to define acidogenicity of foods and their potential cariogenicity. These measures do not truly represent the complexity of the human diet but provide references about specific foods. Edgar et al [47] found considerable difference in the acidogenicity of 54 snack foods. Bibby and Mundroff [32] tested 180 foods and reported that those snacks with high sugar concentrations did not destroy as much tooth enamel in a test situation as did low-concentration sugary foods in combination with starch (breads, cookies). Processed highstarch snacks produce as much acid in dental plaque as sucrose alone, but at a slower rate [48,49]. In conducting measurements of food retention, others investigators [32,43] indicated that high-sugar foods (caramels, chocolate bars) clear the mouth more rapidly than high-starch foods (crackers, potato chips, cookies). When sucrose is added to a cooked starch food, the starch brings sucrose into closer contact with the tooth surface for a longer period than if the food were only a sucrose food [50]. These and other experimental studies with various snack foods have led to conclusions that cariogenicity is dependent on food composition, texture, solubility, retentiveness, and rate of salivary clearance rather than on sugar content alone [32,51]. Hence, "several variables in food components, as well as those in oral biology make it unlikely that any single type of food or food component can be named as the exclusive determinant of caries activity" [32].

Categorizations of foods into noncarigenic and cariogenic groups assist dental practitioners in delivery of practical nutrition education messages to decrease caries risk. Caution should be exercised, however, in presenting these data. Based simply on acidogenic potential identified with rat studies, the following foods were listed as nonacidogenic or as having lower-to-greater acidogenicity [52]: raw vegetables<nuts<milk<corn chips<fresh fruit<ice cream<freench fries<dried fruit. This information used in isolation would imply choosing corn chips over dried fruit; however, in reality, dried fruit is more nutrient dense and can be included in the diet as part of a complete meal, with minimal impact on caries risk. Because frequency of intake, food combinations, nutrient adequacy, and individual needs are as relevant as the potential cariogenicity of a food, nutrition messages for dental patients require context for meaning and accuracy.

Frequency of dietary intake

Despite the overwhelming evidence showing the correlation between sugar consumption and dental caries, increased frequency of eating is considered to be the primary dietary factor associated with the caries process. Most people eat four to six times daily. Exceeding this with frequent nibbling and sipping of sugary/starchy, highly processed foods and beverages for prolonged periods (>30 minutes) increases caries risk [53–55]. Thickened dental plaque, food debris, anatomic tooth abnormalities, and frequent consumption of cooked starches, simple sugars, or processed sugarstarch combinations will sustain an oral environment that promotes demineralization in preference to remineralization. With increased eating frequency, there is expected increased total fermentable carbohydrate intake. Thus, the two are highly associated. Reported caries scores from a number of studies conducted primarily with children are significantly higher in those who report four or more intakes of sugar or three or more between-meal snacks per day [56-58]. These data highlight that both frequency and amount of sugar intake are related to dental caries and control of either is likely to affect the influence of the other.

Dietary factors in caries prevention

Just as there are food components and whole foods that promote the caries process, there are others that are protective and act to enhance the remineralization process. These foods are referred to as being "anticariogenic" or "cariostatic." They act to neutralize acids, restore enamel, or stimulate salvia, with its beneficial effects.

Dairy products

Although milk is associated with early childhood caries in infants because of its lactose (5% sugar) content and linked infant-feeding practices that

allow for prolonged contact with tooth surfaces [59], milk has beneficial implications for caries management. Investigators have conducted human and animal studies demonstrating the effectiveness of milk in reducing the cariogenic potential of sugary foods [60–62]. Calcium and phosphorous bound to casein protein in milk are believed to be responsible for a protective effect on tooth enamel [58,63,64].

Likewise, cheese is considered to be an excellent anticariogenic food [65–67]. When consumed after a sugary food, it is a strong stimulate of salivary flow, resulting in both a buffering effect and neutralization of plaque acids [66,67]. Like milk, cheese contains casein phosphopeptides that appear to reduce demineralization and enhance remineralization. A cube of cheese eaten after sugary meals or snacks reduces the demineralization process.

Sugar substitutes and alternative sweeteners

Nutritive sugar alcohols including sorbitol, mannitol, and xylitol have 40% to 75% of the caloric content of sucrose and have been recognized as having a low caries-producing potential. Xylitol, which is derived from birch trees, corn cobs, oats, strawberries, and bananas, has received the greatest attention by dental professionals [68]. Studies conducted with human subjects evaluating the effect of sorbitol or xylitol or the combination of both on the incidence of caries demonstrated consistent decreases of between 30% and 60% in subjects compared with controls [69]. Xylitol users in the studies experienced the highest caries reductions. Although polyols have biologic properties that effect microbial growth and metabolism, it is thought that the chewing process itself has the greatest role in caries prevention, relative to saliva production [70]. Xylitol chewing gums and mints following a meal or snack are generally effective in reducing caries.

Nonnutritive intense sweeteners used as sugar substitutes are noncarigenic. Approved products on the market include saccharin, aspartame, acesulfame K, sucralose, and cyclamate [18]. Newer products and formulated foods including these products as ingredients will continue to be submitted for government approval. The United States Food and Drug Administration authorized the use of the terms *does not promote, useful in not promoting*, or *expressly for not promoting dental caries* on food labels for those containing one or more sugar alcohols [71].

Plant foods

Plant foods such as grains and vegetables have natural protective factors that act as anticariogenic agents. These are prevalent in unrefined foods and include organic phosphates, inorganic phosphates, polyphenols, phytate, and other nondigestible fibers. Although these factors may act independently and as food additives, there is not sufficient evidence to suggest that they are effective naturally in foods [72]. Fibrous foods, however, stimulate salivary flow and, as part of a healthy diet, contribute to oral health.

Other foods

Green, oolong, and black teas contain fluoride and polyphenols or flavonoids that suppress oral bacterial growth in vitro and reduce the acidogenic potential of sucrose [44,73,74]. Studies in animal models indicate these in vitro effects can translate into caries prevention and that regular tea drinking may reduce the incidence and severity of caries [75]. If substantiated with future significant clinical trials, this could offer a very economical public health intervention.

Other food components have been evaluated for their protective characteristics. Oleic and lenolic fatty acids in cocoa bean husk have shown bactericidal activity against *Streptococcus mutans* in laboratory studies [76,77]. These results indicate that chocolate possesses some anticariogenic potential, but its anticaries activity is not strong enough to suppress significantly the cariogenic activity of the sucrose in chocolate foods [78]. Licorice candies made of glycyrrhizinic acid have been shown to increase plaque-buffering capacity and inhibit bacterial metabolism but can cause enamel staining [79]. Peanuts high in monounsaturates promote mechanical stimulation and salivary flow and are characterized as having a low caries potential [80]. In many cases, either the fermentable carbohydrate or the high fat content in these foods overrides their beneficial effect.

Dietary recommendations

Despite the risk associated with some of the protective foods, the opportunity to fashion dietary messages that put good nutrition into practice exist. Terms like *cariogenic* or *caries promotion* used in descriptions of both foods and diets associated with dental caries conjure avoidance. The role of diet in dental caries incidence and prevalence is reflected in dietary patterns that are a combined consequence of food choices, combination of foods eaten, and frequency of dietary intake in a time period [16].

Assessment of food choices will help the dental professional develop strategies for suggesting the combining of caries-promoting and cariostatic foods [81]. For example, sugary foods can be part of a meal and, when combined with proteins and limited amounts of fats, may be less caries promoting. Fermentable carbohydrates eaten alone stimulate a rapid drop in plaque pH; however, if a nonsugary item that stimulates saliva is eaten immediately before, during, or after this challenge, then the pH will rise [82]. Potential for remineralization is enhanced when calcium-rich and fluoriderich foods are present in the oral cavity. Inclusion of milk or nonfat yogurt with a meal or snack will encourage a cariostatic effect. Elderly subjects who ate cheese frequently had fewer root caries [83]. Foods eaten in sequence within an eating occasion can affect the magnitude of plaque pH. Eating fermentable carbohydrates with greater acidogenic potential sequentially (crackers, cola, caramels, raisins, and cookies) one after the other over a significant period of time will enhance the demineralization process [53,54]. Eating chewy foods (peanuts) combined with those that have minimal effect on salivary flow (apple juice) and eating acidogenic foods like crackers (fermentable carbohydrate) combined with more basic foods like tuna fish (a high-quality protein) can alter the oral environment to enhance maintenance of tooth integrity.

Finally, the overall nutrient adequacy of an individual's diet may be the best indicator of caries risk [16]. Study outcomes report that those who snack frequently and have a high proportion of total energy intake from a variety of fermentable carbohydrates and a low intake of protein foods have more caries [84]. Other investigators report that those who eat diets higher in vegetables and milk products have less caries [85,86]. Diets that promote variety and moderation are going to contribute to both dental and general health.

Dietary screening and education in the dental practice

Dental professionals should routinely screen patients to assess the role of diet in caries risk and management [16]. Dentists are not trained to conduct a complete nutritional assessment that includes anthropometrics and biochemical data but they can use dietary screening, assessment, and analysis to provide nutrition and dietary education and referral to registered dietitians for more in-depth nutrition counseling [81]. Screening activities should include assessment of determinants of dietary intake and behaviors that are associated with dental health status and caries risk [16]. Rather than labeling foods as "allowable" or "to be avoided," nutrition messages need to be offered that promote health but respect the strong influence that taste has on food choices.

Suggested screening questions listed in Table 2 were validated in a clinical setting with multiple dietary records [16] and have proved to be predictors of caries risk among patients who present in dental clinics. Part A of the questionnaire includes those dietary behaviors that were most predictive of caries risk. When responses to these are positive, part B can be initiated to identify additional information specific to understanding underlying issues that can be addressed in a targeted nutrition message. Table 3 describes guidelines for developing targeted messages and presents a rationale for interpretation for patient education. When responses to these questions are combined with assessment of oral health status, xerostomic potential, and dental treatment needs, tailored messages evolve that help patients recognize the importance of oral health within the scheme of achieving overall health status. By way of this approach or one similar, the dental professional is

Table 2 Dietary screening and assessment questionnaire for dental settings

Topic	Sample questions	Response	
Part A			
Frequency of food and beverage intake ^a	Do you eat snacks between meals or instead of meals? Or	Yes No	
-	Do you eat or drink any food or beverage ≥ 5 times/d?	Yes No	
Frequency of fermentable carbohydrate snacking ^a	Do you eat mints, hard or chewy candies, candy bars, donuts, pastries, chips, crackers, or other similar snack foods between meals 3 out of every 6 d?	Yes No	
Frequency of non-diet or regular sugar- sweetened beverage intake ^a	Do you drink non-diet soda, lemonade, fruit aids, sport drinks, or sugar (not sugar substitute)-sweetened tea or coffee between or after meals?	Yes No	
Chewing gum use ^a	Do you chew regular (not sugar-free) chewing gum most days of the week?	Yes No	
		If responses (two or more Yes responses) indicate excessive total sugar intake that exceed guidelines, nutrient adequacy of the diet should be explored to assist patient in making decisions about alternative choices.	
Part B			
Nutrient adequacy of diet	 Do you drink or eat dairy foods at least twice daily? Do you eat fruits and brightly colored vegetables at least 5 times/d? Do you eat whole grain breads or cereals at least 4 times/d? Do you eat proteins like meats, eggs, fish, nuts, or dried beans at least twice daily? Do you drink 6–8 cups of water daily? 	Refer to the guidelines for the development of dental and general health promotion messages listed in Table 3 to assist patient in setting goals for making dietary changes to support caries management.	

^a Questions have been validated for dietary screening in dental settings.

Adapted from Mobley C. Dietary analysis in a restorative practice. In: Duke ES, editor. Proceedings of the 5th Annual Indiana Conference on the Changing Practice of Restorative Dentistry. Indianapolis: Indiana University School of Dentistry; 2002. p. 139–56; with permission.

Guidelines for the dev	elopment of dental and genera	Guidelines for the development of dental and general health promotion messages ^a		
Food group/topic	Guideline	Serving size	Rationale	Reference
Added simple sugars	<12 servings/d or 40 g/d (Food Guide Pyramid)	1 teaspoon = 4 g	Annual sugar consumption data indicates intakes above 55 g/d are associated with increased caries risk.	[34,46,88]
Sugar-free chewing gum	Chew 1 slice after eating for 5–10 min		Sugar alcohols and high-intensity sweeteners in these products do not promote the breakdown of tooth enamel as do sugars. Chewing increases salivary benefits.	[69,70]
Food combinations	Acidogenic with basic/ neutral or chewy foods		An acidic oral environment can be neutralized when basic foods are present or when chewy foods increase saliva production. Examples are cheese and crackers, skim milk and cookies, peanuts and apple slices, bananas and cottage cheese, yogurt and granola, and peanut butter on celery.	[82]
Frequency of meals and snacks	4-5 times/d for adults and 4-6 times/d for children		Eating occasions should be spaced a minimum of 2 h apart to allow time for acidic environments to be neutralized. This behavior may help those interested in weight management to control caloric intake.	[65]
Added fats	<30% total calories/d or with emphasis on unprocessed fats	1 teaspoon $= 9$ g or equivalent of the tip of your thumb	Monounsaturated fats (oleic and lenoleic fatty acid) are not as highly associated with heart disease and cancer risk as other fats and may interfere with adherence to the tooth surface.	[77,88]
Whole grains, cereals, and breads	6-11 servings/d	1 slice or equivalent of one fist of the hand	Unprocessed products are not fermentable, increase salivary flow rates, and increase production of salivary components effective in neutralizing acids.	[44,88]

Table 3 Guidelines for the development of dental and general health promotion messa

330

C.C. Mobley / Dent Clin N Am 47 (2003) 319-336

[16,72,88]	[16,72,88]	[66,88]	
Fresh fruits and vegetables increase salivary flow rate and provide an excellent source of antioxidants important in oral soft tissue health. Cooked and processed forms including juices should be consumed in combination with other neutral foods.	Proteins, vitamins, and minerals essential to healthy soft tissue in these foods act as neutral buffers of acidic oral environments.	Calcium, phosphorus, and casein protein in these products reduce tooth enamel decay; cheeses, in particular, increase salivary flow.	Water increases the rate of oral clearance after eating.
1 small item or cup equal to one fist	3 oz or equivalent of palm of the hand	18 oz cup of milk/yogurt or 1 in cheese cube	18 oz cup
At least 5 servings/d	2-3 servings/d	2-3 servings/d	8 cups/d
Fruits and vegetables	Meat, poultry, fish, eggs, dry beans, and nuts	Milk, yogurt, and cheese	Water

^a Messages should be combined with oral hygiene and other patient self-management instructions.

better prepared to offer nutrition education messages consistent with dental research findings and health-promotion guidelines.

Summary

Promotion of sound dietary practices is an essential component of caries management, along with fluoride exposure and oral hygiene practices. Scientific discoveries have lead to better understanding of the caries process, the ever-expanding food supply, and the interaction between the two. Fermentable carbohydrates interact dynamically with oral bacteria and saliva, and these foods will continue to be a major part of a healthful diet. Dental health professionals can serve their patients and the public by providing comprehensive oral health care and by promoting lifestyle behaviors to improve oral and general health within the time constraints of their practice. Dietary advice given should not contradict general health principles when providing practical guidance to reduce caries risk [87]. The following principles should guide messages:

- Encourage balanced diets based on moderation and variety as depicted by the Food Guide Pyramid and the *Dietary Guidelines for Americans* [88] to provide a sound approach. Avoid references to "bad" foods and focus on "good" diets that include a variety of foods.
- Give examples of how combining and sequencing foods can enhance mastication, saliva production, and oral clearance at each eating occasion. Combining dairy foods with sugary foods, raw foods with cooked, and protein-rich foods with acidogenic foods are all good examples [89]. Suggest that eating and drinking be followed by cariostatic foods such as xylitol chewing gum.
- Drink water to satisfy thirst and hydration needs as often as possible. Restrict consumption of sweetened beverages to meal and snack times when they can be combined with other cariostatic foods.
- When a patient reports excessive dietary intake of a fermentable carbohydrate to the point of displacing other important foods in the diet, identify alternatives that will help the patient maintain or achieve a healthy body weight, oral health status, and a nutrient-dense intake.

References

- [1] König KG. Diet and oral health. Int Dent J 2000;50:162-74.
- [2] Miller WD. The microorganisms of the human mouth. The local and general diseases which are caused by them. Basel, Switzerland: Karger; 1973.
- [3] Kleinberg TM. A mixed-bacteria ecological approach to understanding the role of the oral bacteria in dental caries causation: an alternative to *Streptococcus mutans* and the specificplaque hypothesis. Crit Rev Oral Biol Med 2002;13(2):108–25.

- [4] Alvarez JO. Nutrition, tooth development, and dental caries. Am J Clin Nutr 1995; 61(Suppl):410S-6S.
- [5] Nunn J. Nutrition and dietary challenges in oral health. Nutrition 2001;17:426-7.
- [6] Bowen WH. Do we need to be concerned about dental caries in the coming millennium? Crit Rev Oral Biol Med 2002;13(2):126–31.
- [7] Tanzer JM, Livingston J, Thompson AM. The microbiology of primary dental caries in humans. J Dent Educ 2001;65(10):1028–37.
- [8] Kaufman E, Lamster IB. The diagnostic applications of saliva—a review. Crit Rev Oral Biol Med 2002;13(2):197–212.
- [9] Stack KM, Papas AS. Xerostomia: etiology and clinical management. Nutr Clin Care 2001; 4(1):15–21.
- [10] American Dietetic Association. Position of the American Dietetic Association: the impact of fluoride on dental health. J Am Diet Assoc 2000;100:1208–13.
- [11] Petersson GH, Bratthall D. The caries decline: a review of reviews. Eur J Oral Sci 1996; 104:436–43.
- [12] Stookey GK. Current status of caries prevention. Compendium 2000;21(10A):862-7.
- [13] Featherstone JDB. The science and practice of caries prevention. J Am Dent Assoc 2000; 131:887–99.
- [14] Diagnosis and Management of Dental Caries Throughout Life. NIH Consensus Statement. 2001 March 26–28 18(1):1–30.
- [15] US Department of Health and Human Services. Oral health in America: a report of the Surgeon General. Executive summary. Rockville (MD): US DHHS, NIDCR, NIH; 2000.
- [16] Mobley C. Dietary analysis in a restorative practice. In: Duke ES, editor. Proceedings of the 5th Annual Indiana Conference on the Changing Practice of Restorative Dentistry. Indianapolis: Indiana University School of Dentistry; 2002. p. 139–56.
- [17] Jensen ME. Diet and dental caries. Dent Clin N Am 1999;43(4):615-33.
- [18] American Dietetic Association. Position of the American Dietetic Association: the use of nutritive and nonnutritive sweeteners. J Am Diet Assoc 1998;98(5):580–7.
- [19] Coulston AM, Johnson RK. Sugar and sugars: myths and realities. J Am Diet Assoc 2002; 102(2):351–3.
- [20] Al-Malik MI, Holt RD, Bedi R. The relationship between erosion, caries and rampant caries and dietary habits in preschool children in Saudi Arabia. Int J Paediatr Dent 2001; 11(6):430–9.
- [21] Beighton D, Adamson A, Rugg-Gunn A. Associations between dietary intake, dental caries experience and salivary bacterial levels in 12-year-old English schoolchildren. Arch Oral Biol 1996;41(3):271–80.
- [22] Burt BA, Satishchandra P. Sugar consumption and caries risk: a systematic review. J Dent Educ 2001;65(10):1017–23.
- [23] Garcia-Closas R, Garcia-Closas M, Serra-Majem L. A cross-sectional study of dental caries, intake of confectionery and foods rich in starch and sugars, and salivary counts of *Streptococcus mutans* in children in Spain. Am J Clin Nutr 1997;66(5):1257–63.
- [24] Karjalainen S, Soderling E, Sewon L, et al. A prospective study on sucrose consumption, visible plaque and caries in children from 3 to 6 years of age. Community Dent Oral Epidemiol 2001;29(2):136–42.
- [25] Milgrom P, Riedy CA, Weinstein P, et al. Dental caries and its relationship to bacterial infection, hypoplasia, diet, and oral hygiene in 6- to 36-month-old children. Community Dent Oral Epidemiol 2000;28(4):295–306.
- [26] Rodrigues CS, Sheiham A. The relationships between dietary guidelines, sugar intake and caries in primary teeth in low income Brazilian 3-year-olds: a longitudinal study. Int J Paediatr Dent 2000;10(1):47–55.
- [27] Rugg-Gunn AJ, Hackett AF, Appleton DR, et al. Relationship between dietary habits and caries assessed over two years in 405 English adolescent school children. Arch Oral Biol 1984;29:983–92.

- [28] Shaw JH. Animal caries models-resource paper. J Dent Res 1986;65:1485-90.
- [29] Clarkson BH. In vitro methods for testing the cariogenic potential of foods. J Dent Res 1986;65:1516–9.
- [30] Ten Cate JM. Demineralization models: mechanistic aspect of the caries process with special emphasis on the possible role of foods. J Dent Res 1986;65:1511–5.
- [31] Stephan RM. Changes in hydrogen-ion concentration on tooth surfaces and in carious lesions. J Am Dent Assoc 1940;27:718–23.
- [32] Bibby BG, Mundroff SA. Enamel demineralization by snack foods. J Dent Res 1975; 54(3):461–70.
- [33] Blinkhorn AS, Davies RM. Caries prevention. A continued need worldwide. Int Dent J 1996;46:119–25.
- [34] Sreenby LM. Sugar and human dental caries. World Rev Nutr Diet 1982;40:19-65.
- [35] Woodward M, Walker ARP. Sugar consumption and dental caries: evidence from 90 countries. Br Dent J 1994;176:297–302.
- [36] Kantor LS. A dietary assessment of the U.S. food supply: comparing per capita food consumption with Food Guide Pyramid serving recommendations. Food and Rural Economics Division, ERS, USDA, Agricultural Economic Report No. 772. Washington, DC: US Government Printing Office; 1998.
- [37] Guthrie JF, Morton JF. Food sources of added sweeteners in the diets of Americans. J Am Diet Assoc 2000;100:43–51.
- [38] Gustaffsen BE, Quensel CE, Lanke SL, et al. The Vipeholm dental caries study. The effects of different levels of carbohydrate intake in 436 individuals served for five years. Acta Odontol Scand 1954;11:195–206.
- [39] Krasse B. The Vipeholm dental caries study: recollections and reflections 50 years later. J Dent Res 2001;80(9):1785–8.
- [40] Kashket S, Yaskell T, Mrphy JE. Delayed effect of wheat starch in foods on the intraoral demineralization of enamel. Caries Res 1994;28:291–6.
- [41] Lineback DR. The starch granule organization and properties. Baker's Digest 1984;3: 16–21.
- [42] Imfeld TN. Identification of low caries risk dietary components. Monographs Oral Sci (Basel, Karger) 1983;11:3–195.
- [43] Kashket S, Zhang J, van Houte J. Accumulation of fermentable sugars and metabolic acids in food particles that become entrapped on the dentition. J Dent Res 1996;75:1885–91.
- [44] Lingstrom P, van Houte J, Kashket S. Food starches and dental caries. Crit Rev Oral Biol Med 2000;11(3):366–80.
- [45] Lingstrom P, Holm J, Birkhed D, et al. Effects of variously processed starch on pH of human dental plaque. Scand J Dent Res 1989;97:392–400.
- [46] Sheiham A. Dietary effects on dental diseases [special issue]. Public Health Nutr 2001; 4(2B):569–91.
- [47] Edgar WM, Bibby BG, Mundorff S, et al. Acid production in plaques after eating snacks: modifying factors in foods. J Am Dent Assoc 1975;90:418–25.
- [48] Grenby TH. Snack foods and dental caries. Investigations using laboratory animals. Br Dent J 1990;168(9):353–61.
- [49] Mormann JE, Muhlemann HR. Oral starch degradation and its influence on acid production in human dental plaque. Caries Res 1981;15:166–75.
- [50] Sgan-Cohen HD, Newbrun E, Huber R, et al. The effect of previous diet on plaque pH response to different foods. J Dent Res 1988;67:1434–7.
- [51] Morrissey RB, Burkholder BD, Tarka SM Jr. The cariogenic potential of several snack foods. J Am Dent Assoc 1984;109(4):589–91.
- [52] Palmer CA. Important relationships between diet, nutrition, and oral health. Nutr Clin Care 2001;4:4–14.
- [53] Firestone AR, Schmid R, Muhlemann HR. Effect of the length and number of intervals between meals on caries in rats. Caries Res 1984;18:128–33.

- [54] Gatenby SJ. Eating frequency: methodological and dietary aspects. Br J Nutr 1997;77: S7–10.
- [55] Kandelman D. Sugar, alternative sweeteners and meal frequency in relation to caries prevention: new perspectives. Br J Nutr 1997;77:S121–8.
- [56] Holbrook WP, Arnadottir IB, Takazoe I, et al. Longitudinal study of caries, cariogenic bacteria and diet in children just before and after starting school. Eur J Oral Sci 1995; 103:42–5.
- [57] Holt RD. Foods and drinks at four daily time intervals in a group of young children. Br Dent J 1991;170:137–43.
- [58] Jenkins GN, Ferguson DB. Milk and dental caries. Br Dent J 1966;120:472-7.
- [59] Tinanoff N, Palmer CA. Dietary determinants of dental caries and dietary recommendations for preschool children. J Public Health Dent 2000;60(3):197–206.
- [60] Bowen WH, Pearson SK, Wuuyckhuyse BC, et al. Influence of milk, lactose reduced milk and lactose on caries in desalivated rats. Caries Res 1991;25:283–6.
- [61] Reynolds EC, Johnson H. Effect of milk on caries incidence and bacterial composition of dental plaque in the rat. Arch Oral Biol 1981;26:445–51.
- [62] Thompson ME, Dever JG, Pearce EIF. Intra-oral testing of flavoured sweetened milk. N Z Dent J 1984;80:44–6.
- [63] Bibby BG, Huang CT, Zero D, et al. Protective effect of milk against in vitro caries. J Dent Res 1980;59(10):1565–70.
- [64] Weiss ME, Bibby BG. Effects of milk on enamel solubility. Arch Oral Biol 1966;11:49–57.
- [65] Edgar WM, Bowen WH, Amsbaugh S, et al. Effects of different eating patterns on dental caries in the rat. Caries Res 1982;16:384–9.
- [66] Kashket S, DePaola DP. Cheese consumption and the development and progression of dental caries. Nutr Rev 2002;60(4):97–103.
- [67] Moynihan PJ, Jenkins GN, Fletcher E. Cheese-containing meals increase plaque calcium following acidic drink consumption [abstract 3697]. J Dent Res 2000;79:606.
- [68] Makinen KK, Makinen PL, Pape HR Jr, et al. Conclusion and review of the Michigan Xylitol Programme (1986–1995) for the prevention of dental caries. Int Dent J 1996; 46(1):22–34.
- [69] Hayes C. The effect of non-cariogenic sweeteners on the prevention of dental caries: a review of the evidence. J Dent Educ 2001;65(10):1106–9.
- [70] Machiulskiene V, Nyvad B, Baelum V. Caries preventive effect of sugar-substituted chewing gum. Community Dent Oral Epidemiol 2001;29(4):278–88.
- [71] Health claims. Dietary sugar alcohols and dental caries. Fed Register 1996;61:43433-47.
- [72] Moynihan P. Foods and factors that protect against dental caries. Nutr Bull 2000;25: 281-6.
- [73] Bennick A. Interactions of plant polyphenols with salivary proteins. Crit Rev Oral Biol Med 2002;13(2):184–96.
- [74] Linke HAB, Salgado T, Retino M, et al. Effect of black tea on caries formation in hamsters [abstract 3601]. J Dent Res 2000;79:594.
- [75] Hamilton-Miller JM. Anti-cariogenic properties of tea (*Camellia sinensis*). J Med Microbiol 2001;50(4):299–302.
- [76] Bowen WH. Food components and caries. Adv Dent Res 1994;8:215-20.
- [77] Ooshima T, Osaka Y, Sasaki H, Osawa K, et al. Cariostatic activity of cacao mass extract. Arch Oral Biol 2000;45(9):805–8.
- [78] Osawa K, Miyazaki K, Shimura S, et al. Identification of cariostatic substances in the cacao bean husk: their anti-glucosyltransferase and antibacterial activities. J Dent Res 2001;80(11):2000–4.
- [79] Edgar WM. Reduction in enamel dissolution by liquorice and glycyrrhizinic acid. J Dent Res 1978;57:59–64.
- [80] Mundorff SA, Featherstone JD, Bibby BG, et al. Cariogenic potential of foods. I. Caries in the rat model. Caries Res 1990;24(5):344–55.

- [81] American Dietetic Association. Position of the American Dietetic Association: oral health and nutrition. J Am Diet Assoc 1996;96:184–9.
- [82] Geddes DAM. Diet patterns and caries. Adv Dent Res 1994;8:221-4.
- [83] Papas AS, Joshi A, Palmer CA, et al. Relationships of diet to root caries. Am J Clin Nutr 1995;61:423S–9S.
- [84] Burt BA, Eklund SA, Morgan KJ, et al. The effect of sugars intake and frequency of ingestion on dental caries in a three-year longitudinal study. J Dent Res 1988;67:1422–9.
- [85] Petridou E, Athanassouli T, Panagopoulos H, et al. Socio demographic and dietary factors in relation to dental health among Greek adolescents. Community Dent Oral Epidemiol 1996;24(5):307–11.
- [86] Rugg-Gunn AJ. Nutrition and dental health. Oxford, England: Oxford University Press; 1994.
- [87] DePaola DP, Mobley C, Touger-Decker R. Nutrition and oral medicine. In: Berdanier CD, editor. Handbook of nutrition and food. Boca Raton (FL): CRC Press LLC; 2002. p. 1113–4.
- [88] Nutrition and your health: dietary guidelines for Americans (2000). 5th edition. Available at: http://www.health.gov/dieatyguidelines/. Accessed June 2002.
- [89] Mobley C, Dodds MW. Diet and dental health. Top Nutr 1998;7:1-19.