Sugars and dental caries[1–4]

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ABSTRACT A dynamic relation exists between sugars and oral health. Diet affects the integrity of the teeth; quantity, pH, and composition of the saliva; and plaque pH. Sugars and other fermentable carbohydrates, after being hydrolyzed by salivary amylase, provide substrate for the actions of oral bacteria, which in turn lower plaque and salivary pH. The resultant action is the beginning of tooth demineralization. Consumed sugars are naturally occurring or are added. Many factors in addition to sugars affect the caries process, including the form of food or fluid, the duration of exposure, nutrient composition, sequence of eating, salivary flow, presence of buffers, and oral hygiene. Studies have confirmed the direct relation between intake of dietary sugars and dental caries across the life span. Since the introduction of fluoride, the incidence of caries worldwide has decreased, despite increases in sugars consumption. Other dietary factors (eg, the presence of buffers in dairy products; the use of sugarless chewing gum, particularly gum containing xylitol; and the consumption of sugars as part of meals rather than between meals) may reduce the risk of caries. The primary public health measures for reducing caries risk, from a nutrition perspective, are the consumption of a balanced diet and adherence to dietary guidelines and the dietary reference intakes; from a dental perspective, the primary public health measures are the use of topical fluorides and consumption of fluoridated water. *Am J Clin Nutr* 2003;78(suppl):881S–92S.

KEY WORDS Sugars, dental caries, oral infectious disease, diet, carbohydrate

INTRODUCTION

According to the American Dietetic Association (1), "nutrition is an integral component of oral health. ...". Oral health and nutrition have a synergistic relation. Oral infectious diseases and acute, chronic, and terminal systemic diseases with oral manifestations affect the functional ability to eat as well as diet and nutrition status. Likewise, nutrition and diet may affect the development and integrity of the oral cavity and the progression of diseases of the oral cavity. As stated by the Surgeon General's report *Oral Health in America* (2), diet and nutrition are major multifactorial environmental factors in the etiology and pathogenesis of craniofacial diseases.

This article focuses on sugars and oral infectious disease, with an emphasis on the relation between sugars and dental caries. Terms that are frequently referred to throughout the text of this manuscript are listed in **Table 1**. Throughout the paper, the terms *DMFT* (decayed, missing, filled teeth) and *DMFS* (decayed, missing, filled surfaces) will be used to refer to the dental caries seen in various populations. When in uppercase letters, the terms refer to permanent dentition; in lowercase letters, the terms refer to primary dentition.

RELATION BETWEEN DIET AND NUTRITION AND ORAL HEALTH AND DISEASE

The relation between diet and nutrition and oral health and disease can best be described as a synergistic 2-way street. Diet has a local effect on oral health, primarily on the integrity of the teeth, pH, and composition of the saliva and plaque. Nutrition, however, has a systemic effect on the integrity of the oral cavity, including teeth, periodontium (supporting structure of the teeth), oral mucosa, and alveolar bone. Alterations in nutrient intake secondary to changes in diet intake, absorption, metabolism, or excretion can affect the integrity of the teeth, surrounding tissues, and bone as well as the response to wound healing.

TOOTH EROSION AND ORAL INFECTIOUS DISEASES

The most prevalent oral infectious diseases are dental caries and periodontal diseases. Tooth erosion is not an infectious disease, but the resultant defects impair the integrity of the tooth. The etiology of the diseases differs with the extent to which diet and nutrition are involved. Although enamel defects may be related to nutrition during tooth formation, they are not addressed here.

Tooth erosion is the progressive loss of dental hard tissue by acids in a process that does not involve bacteria or sugars. The intrinsic acids are from vomiting, gastroesophageal reflux, and regurgitation (3). The extrinsic acids are from the diet [eg, sports beverages (4) and citrus products, including citrus fruit, juices, soft drinks, and citrus-flavored candies and lozenges] or from the occupational environment (eg, battery and galvanizing factories) (5). Tooth erosion as a result of eating disorders (bulimia nervosa) (6) and dietary practices involving frequent intake of acidic foods and beverages (7) can weaken tooth integrity.

Dental caries was first described in Miller's chemoparasitic theory in 1890 (8). Caries is caused by the dissolution of the teeth by

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TABLE 1
Definitions of terms

Anticariogenic: foods and beverages that promote remineralization
Added sugars: sugars that are eaten individually or added to processed or prepared foods, including white, brown, and raw sugar, corn syrup (high-fructose corn syrup, and corn syrup solids), maple syrup, honey, molasses, liquid fructose, and other sugar syrups
Cariogenic: foods and drinks containing fermentable carbohydrates that can cause a decrease in plaque pH to <5.5 and demineralization of underlying tooth surfaces
Cariostatic: foods that are not metabolized by microorganisms in plaque and subsequently do not cause a drop in plaque pH to <5.5 within 30 min
Dental caries (decay): an oral infectious disease of the teeth in which organic acid metabolites produced by oral microorganisms lead to demineralization and destruction of the tooth structures
DMFT, DMFS, dmft, and dmfs: decayed, missing, filled, teeth or surfaces; refers to permanent dentition when written in uppercase letters; and to primary dentition when written in lowercase letters
Early childhood caries: rampant dental caries in infants and toddlers. Previously called baby bottle tooth decay or maxillary anterior caries; refers to one or more primary maxillary incisors that is decayed, missing or filled
Fermentable carbohydrate: any carbohydrate that can be hydrolyzed by salivary amylase in the initial stage of carbohydrate digestion and subsequently fermented by bacteria
Periodontal disease: oral disease characterized by inflammation and destruction of the attachment apparatus of the teeth, including the ligamentous attachment of the tooth to the surrounding alveolar bone
Polyols: sugar alcohols, including sorbitol, xylitol, and mannitol
Root caries (decay): progressive lesions found on the root surface or the cemento-enamel junction; most frequently seen on roots of teeth with gingival recession
Sweets: foods that retain some naturally occurring sugars or contain large amounts of added sugars
Sugar: refers to sucrose only (for the purpose of this paper)
Sugars: includes the mono- (glucose, galactose, and fructose) and disaccharides (sucrose, lactose, and trehalose)

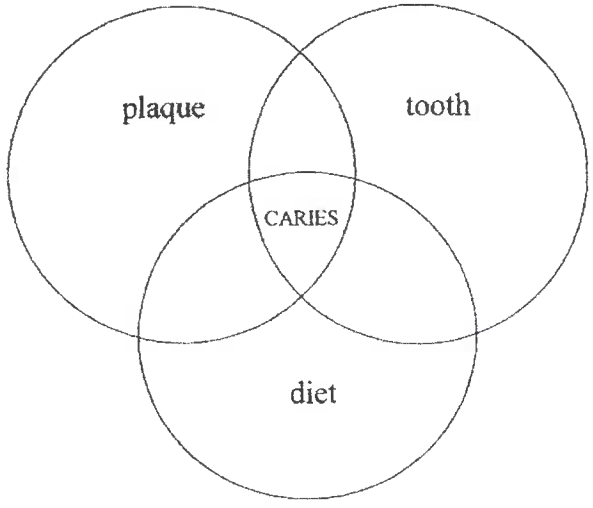

FIGURE 1. The 3 prerequisites for caries development, as described by Keyes and Jordan (9).

acid produced by the metabolism of dietary carbohydrates by oral bacteria. The 2 primary bacteria involved in caries formation are mutans streptococci and lactobacilli. In the 1960s the caries theory was depicted as 3 circles representing the 3 prerequisites for dental caries: the tooth, the diet, and dental plaque (**Figure 1**) (9). Since then, many modifying factors have been recognized, resulting in a more complex model that includes saliva, the immune system, time, socioeconomic status, level of education, lifestyle behaviors, and the use of fluorides. An important breakthrough in the understanding of dental caries was the recognition of the remineralization process as a result of plaque fluid and saliva at pH levels above a critical value being highly saturated with calcium and phosphates. The caries process can be described as loss of mineral (demineralization) when the pH of plaque drops below the critical pH value of 5.5; the critical value for enamel dissolution is 5–6, and an average pH of 5.5 (8, 9) is the generally accepted value. Redisposition of mineral (remineralization) occurs when the pH of plaque rises. The presence of fluoride reduces the critical pH by 0.5 pH units, thus exerting its protective effect (10). Whether a lesion develops is the outcome of the balance between demineralization and remineralization, in which the latter process is significantly slower than the former.

Diet and nutrition may interfere with the balance of tooth demineralization and remineralization in several ways. The diet provides sugars and other fermentable carbohydrates, which are metabolized to acids by plaque bacteria (**Figure 2**). The resultant low pH favors the growth of the acidogenic and aciduric bacteria (mutans streptococci). In contrast, a diet lower in added sugars and fermentable carbohydrates and high in calcium-rich cheese may favor remineralization. Sucrose facilitates the colonization of teeth by mutans streptococci and their outgrowth (12–14). In rat caries experiments, the regrowth of mutans streptococci after suppression by intensive chlorhexidine therapy was enhanced by a sucrose-containing diet as compared with a sucrose-poor diet (15).

Nutrition may affect both the anatomy and function of salivary glands (16, 17). Chronic malnutrition may reduce the secretion rate of saliva and the buffer capacity of stimulated saliva but not that of unstimulated saliva (18). Malnutrition can adversely affect the volume, antibacterial properties, and physiochemical properties of saliva.

Periodontal disease is an inflammatory response to bacterial products in dental plaque; it is an oral infectious disease that affects the supporting structures of the teeth. Select deficiencies of nutrients (notably calcium, folate, and vitamin C) can compromise the associated inflammatory response and wound healing, which alters nutrient needs (19–21). A balanced diet is important in diminishing the severity of periodontal disease, although of limited value when combined with good oral hygiene (22). No substantive data support a relation between intake of dietary sugars and risk of or progression of periodontal disease.

EPIDEMIOLOGY OF CARIES

Caries incidence in Europe

Caries are as old as mankind, and the prevalence of caries is reported to increase temporarily in relatively affluent periods. In Europe, for example, there was an increase in caries during the Roman occupation, probably as a result of the increased use of cooked foods. These early increases were minor compared with the dramatic increase that started from the time that sucrose was

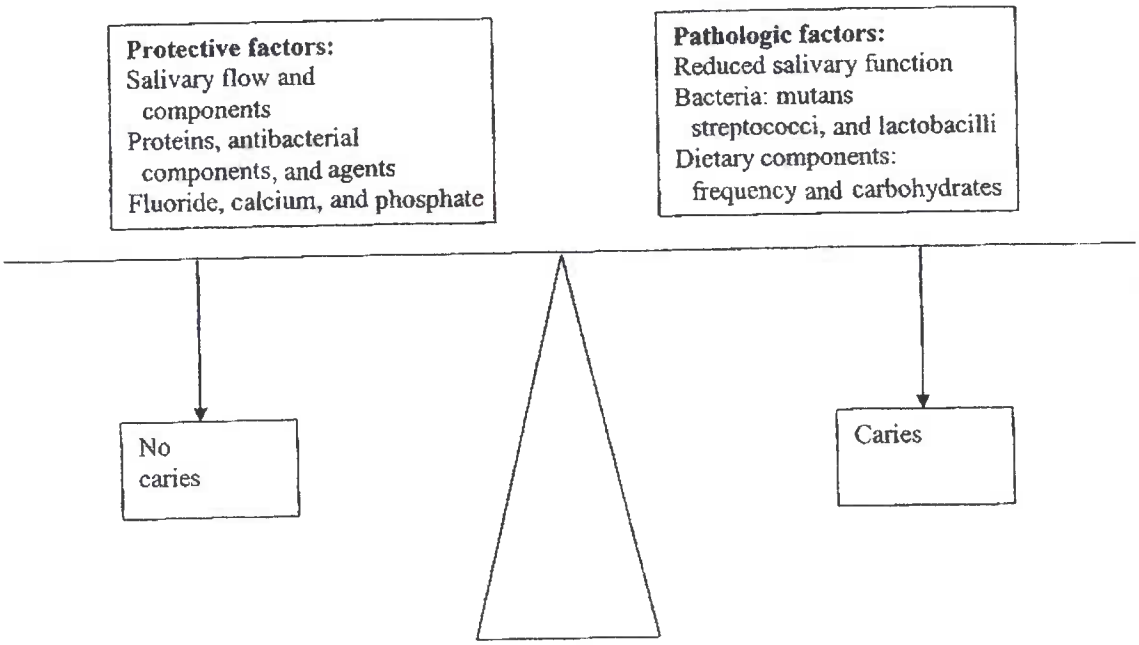

FIGURE 2. Schematic diagram of the balance between pathologic and protective factors in the caries process (11).

imported from the Caribbean islands to Europe. This increase continued until the 1960s, by which time dental caries were considered rampant. At that time, in nonfluoridated European countries like the Netherlands, 5- to 6-y-old children had 18 dmfs and 12-y-old children had 8 DMFT (23).

Since the 1970s, a dramatic decrease in the prevalence of dental caries has occurred in developed countries (**Figure 3**). During the 1990s in the Netherlands, the mean dmfs in 5-y-old children was only 4, whereas >50% of these children were cavity free (25). In this same population, the DMFT for the 12-y-old children was only 1.1%, and 55% of the children were cavity free. The distribution of the children according to their caries experience is skewed, and 60–80% of the decay is found in 20% of the population in both Europe and the United States. However, evidence indicates that the favorable trends in dental caries have stabilized (26).

Caries incidence in the United States

Dental caries is one of the most common childhood diseases in the United States (2). It is 5 times more common than asthma and 7 times more common than hay fever and its prevalence increases with age throughout adulthood (2). Of children aged 5–9 y, 51.6% have had ≥1 filling or caries lesion; of those aged 17 y, the proportion is 77.9%; 85% of adults aged >18 y have had caries (2). The poor have greater proportions of untreated teeth with caries than do those who are not poor. Among adult ethnic groups, poor non-Hispanic white adults have an incidence of 27% untreated decayed teeth as opposed to 8.6% of nonpoor adults. Among Mexican Americans, the percentages are 46.9% and 21.9%, respectively, for the poor and nonpoor; among non-Hispanic blacks, the values are 46.7% and 30.2%, respectively, for the poor and nonpoor. However, in the last quarter of the 20th century, the percentage of adults with no decay or fillings increased slightly from 15.7% to 19.6% in those aged 18–34 y and from 12% to 13.5% in those aged 35–54 y. Reasons for the decline are partly attributed to increased use and availability of fluoride (2, 11). These trends, however, were not found in older adults during this period; in the older adult population, the percentage of teeth free of caries and restorations declined from 10.6% to 7.9% in those aged 55–64 y and from 9.6% to 6.5% in those aged 65–74 y (2).

One of the health objectives for the United States in *Healthy People 2010* (27) is the further reduction of dental caries in all age groups through public health initiatives and improved access to care. The goal for children is to reduce the incidence of decay to 11%; for untreated caries, the goal for children and adults is to reduce the incidence to 9% of the population. During the 1988–1994 baseline period, 52% of children aged 6–8 y and 61% of adolescents had dental caries. The incidence of dental caries by culture and education level (of the head of household) in the United States for children and adolescents is shown in **Table 2**. These health disparities appear to be greatest in minorities and in economically disadvantaged populations.

ROOT CARIES

Older people are at risk of root caries as a result of the exposure of the root surface to the oral environment. This exposure may be related to physiologic retraction of the gingiva or to damage related to oral hygiene habits and periodontal diseases or treatment. In the United States and in Europe, the prevalence of root caries is increasing as the populations are aging (28, 29).

CONFERENCES ON THE DECLINE OF CARIES

Petersson and Bratthall (30) reviewed the primary conferences on the decline of caries held after 1982 and concluded that the authors at these conferences found that the use of fluorides contributed significantly to the decline in dental caries prevalence. Other factors and hypotheses were also posed and included

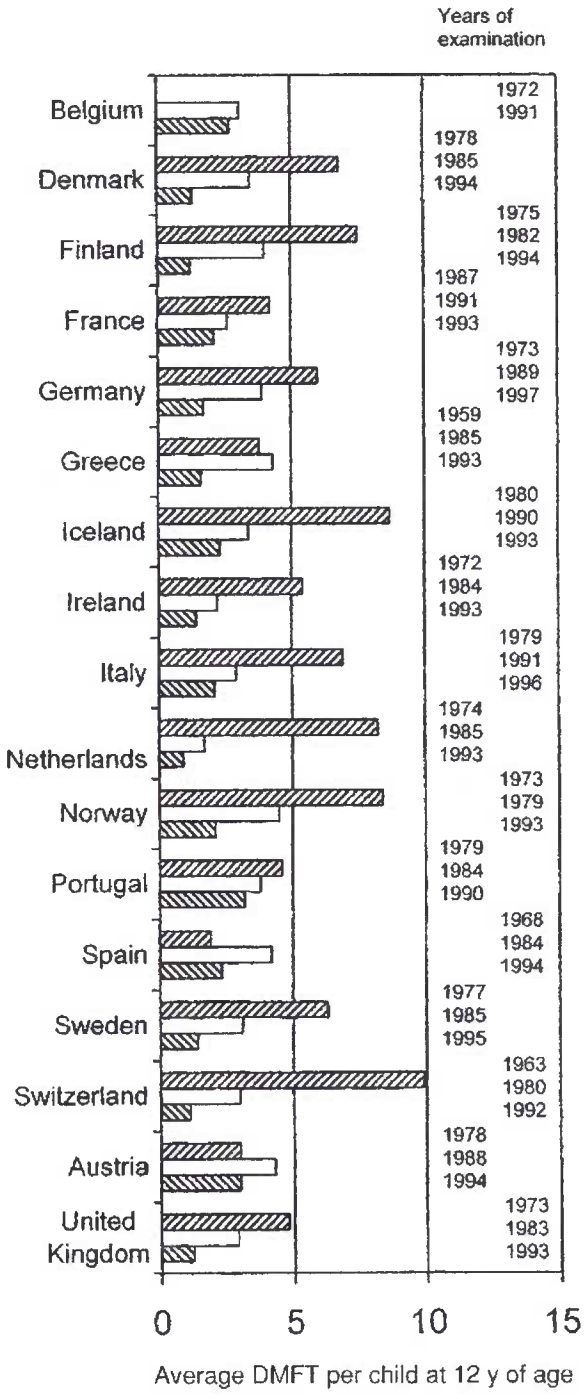

FIGURE 3. Decline in dental caries in European countries (24): average decayed, missing, and filled teeth (DMFT) per child at age 12 y.

increased dental awareness, increased availability of dental resources, decreased sucrose consumption, introduction of dental health education programs, improved preventive approaches in dental practices, changed diagnostic criteria, and other factors.

TABLE 2
Percentage of children and adolescents with dental caries experience at selected ages, 1988–1994 (unless noted otherwise)[1]

	Ages 2–4 y (n = 18)	Ages 6–8 y (n = 52)	Age 15 y (n = 61)
		%	
Race and ethnicity			
American Indian or Alaska Native	76[2]	90[2]	89[2]
Asian or Pacific Islander	DSU	DSU	DSU
Asian	34[3]	90[3]	DSU[3]
Native Hawaiian and other Pacific Islander	DNC	79[4]	DNC
Black or African American	24	50	70
White	15	51	60
Hispanic or Latino	DSU	DSU	DSU
Mexican American	27	68	57
Not Hispanic or Latino	17	49	62
Black or African American	24	49	69
White	13	49	61
Sex			
Female	19	54	63
Male	18	50	60
Education level (head of household)			
Less than high school	29	65	59
High school graduate	18	52	63
At least some college	12	43	61
Select populations			
Third-grade students	NA	60	NA

[1] Adapted from Healthy People 2010 (27). DNC, data not collected; DSU, data statistically unreliable; NA, not applicable.
[2] Data are for Indian Health Services service areas, 1999.
[3] Data are for California, 1993–1994.
[4] Data are for Hawaii, 1999.

The contribution of decreased sucrose consumption to the decline in caries prevalence is often discussed because, in many European countries, sucrose consumption did not decline (**Table 3**) but the incidence of caries did (31–33).

In the ensuing discussion, the authors did not differentiate between sugars consumed as sucrose and as other monosaccharides and disaccharides. Ruxton et al (34) used data from Sreebny (35) and Woodward and Walker (36) to inventory sugar availability and dental caries in >60 countries in the 1970s and 1980s to assess the relation between differences in caries rates and the sugar supply. In 18 countries, both DMFT and the sugar supply declined, whereas in 25 countries DMFT declined and sugar consumption increased. In another 18 countries, the incidence of caries and the sugar supply increased. In the 29 industrialized countries examined by Woodward and Walker (36), there was no evidence of a sugar-caries relation. However, it may not be the amount of sugars consumed but how they are eaten—particularly the frequency of consumption, the consistency of the food, and oral hygiene practices—that determines cariogenicity (11, 37–39).

ORAL HYGIENE, FLUORIDE, SALIVA, AND OTHER INFLUENCES ON CARIES RISK

"A clean tooth will not decay," stated J Leon Williams (1852–1931), first president of the American Dental Association. He suggested that oral hygiene is sufficiently effective to prevent dental caries. Stephan and Miller (40) provided evidence for this

TABLE 3
National data on sugar disappearance in Europe in the 1980s and 1990s[1]

Country	Sugar disappearance		
	1980–1984	1985–1989	1990–1994
	$kg \cdot y^{-1} \cdot capita^{-1}$		
Austria	39	35	—
Belgium	39	40	—
Croatia	—	17	
Czech Republic	38	40	—
Denmark	42	40	37
Finland	42	38	38
France	—	—	38
Germany, former East	40	41	37
Germany, former West	37	35	37
Hungary	38	34	38
Iceland	50	52	55
Ireland	40	38	37
Italy	31	28	22
Netherlands	39	39	39
Norway	35	43	42
Poland	41	46	—
Portugal	31	30	29
Russia	44	47	—
Slovak Republic	38	40	—
Spain	31	—	—
Sweden	43	45	43
Switzerland	43	43	43
United Kingdom	38	37	35

[1] Adapted from Marthaler et al (26). The authors did not differentiate between sugars consumed as sucrose or as other monosaccharides and disaccharides.

statement after they conducted the first pH measurements in dental plaque. Within 3 min after human teeth were rinsed with a sucrose solution, the pH of plaque dropped from 6.5 to 5.0 and remained such for 40 min. After the teeth were cleaned, no decrease in pH was registered. However, although consumers can be instructed on proper brushing technique and frequency, it is unrealistic to assume that simple brushing alone will prevent dental caries (41, 42).

Clinical trials with fluoridated toothpastes have shown that caries can be prevented by adequate oral hygiene with the use of fluoridated toothpaste. The effect of fluoridated toothpaste on enamel demineralization was studied in relation to a varied frequency of carbohydrate consumption (43). Participants wearing dental appliances in which slabs of enamel were fixed on teeth consumed 500 mL of a 12% sucrose solution with and without the twice daily use of sodium fluoride (1450 ppm fluoride) or fluoride-free toothpaste. The solutions were consumed at once or 3, 5, 7, or 10 times/d for 5 d. When the subjects used the fluoride toothpaste, net demineralization of the enamel slabs was evident only after consumption of the solutions at a frequency of 7 and 10 times/d, although it was not statistically significant (**Figure 4**). When the subjects did not use the fluoride toothpaste, statistically significant demineralization was observed when the consumption frequency of the sucrose solution was ≥3 times/d. This study (43) showed the importance of brushing the teeth with fluoride toothpaste in reducing the risk of caries with the frequent consumption of cariogenic foods. Other studies have confirmed that the effect of sugars consumption on caries development depends partly on the cleanliness of teeth (44–46).

CULTURAL INFLUENCES ON CARIES RISK

Because the behavior of individual persons is an important determinant of caries and caries risk, it is clear that cultural and

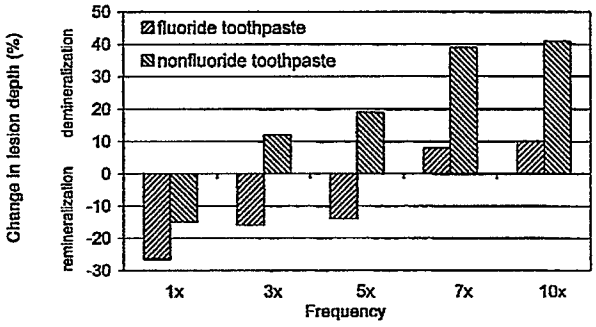

FIGURE 4. Effect of frequency of sugar rinses on lesion depth when fluoride toothpaste or a nonfluoride toothpaste is used (43).

social influences play a role as well. When the prevalence of caries increased in the 16th century, the wealthy upper class was affected first because they could afford and used sucrose. A comparable pattern is now seen in African countries, where the prevalence and severity of dental caries tends to be higher in affluent urban areas, where sugars are more available than in rural communities. In newly industrialized countries, the incidence of caries increases when people switch from a dependence on traditional starchy staple foods to a dependence on refined carbohydrates. In most industrialized countries, persons with a relatively high risk of caries are found in the lower socioeconomic and immigrant groups (Table 2), although differences seem to diminish in older age groups. The risk of caries in young children according to the country of birth and education level of the mother is shown in **Table 4** (25). In the Netherlands, children of Turkish or Moroccan mothers had a higher incidence of caries than did the native Dutch children. Cultural influences diminished as the children grew older.

A similar study in children aged 18 mo to 4.5 y was conducted in Great Britain (44). This project examined the relation between the intake of dietary sugars, toothbrushing frequency, social class, and caries experience in a cross-sectional study. The children were classified into 4 groups according to social class and toothbrushing habits. The associations between diet and caries were examined for biscuits and cakes, candy, chocolate confectionery, soft drinks, and the percentage of energy from added sugars. The strength of the association between social class and caries was 2 times that between toothbrushing and caries and nearly 3 times that between consumption of sugars and caries; the associations with other dietary variables were not significant. The association of caries with sugars, both in amount and frequency, was present only for children whose teeth were brushed only once per day. Gibson and Williams (44) concluded that twice daily toothbrushing with fluoride toothpaste may have a greater effect on the reduction in caries in young children than does the restriction of foods sweetened with sugars.

Kuusela et al (47) studied odds ratios in 20 European countries, Canada, and Israel for the association between the self-reported family socioeconomic status of 11-y-old children and the consumption of soft drinks and sweets more than once per day. In general, self-reported family socioeconomic status (good compared with poor and average) was positively associated with soft drinks or sweets consumed more than once daily; in the countries where the association was significant, the odds ratios varied between 2.9 and 20.2 for soft drinks and between 1.8 and 5.6 for sweets. These findings do not

TABLE 4
Effect of the consumption of sweet snacks, oral hygiene habits, and mother's educational level and country of birth on the dental caries status of children in relation to a reference child[1]

	dmfs[2]		DMFS,[2]
	Age 5 y	Age 8 y	age 11 y
Reference child[3]	2.1	5.5	1.9
Consumption of sweet snacks			
<1 time/d	−0.2	−1.1[4]	0.0
1–5 times/d	0.0	0.0	0.0
>5 times/d	+5.7[4]	+3.9	+1.0
Oral hygiene habits			
1 (poor)	+1.5[4]	+1.0	−0.5
2	0.0	0.0	0.0
3	−0.5	−0.4	−0.9[4]
4 (very adequate)	−0.3	−2.6[4]	−1.6[4]
Mother's educational level			
1 (low)	+1.4	+1.8	+1.4[4]
2	+1.6	+0.6	+0.6
3	0.0	0.0	0.0
4 (high)	−0.8[4]	−2.0[4]	−0.4
Mother's country of birth			
The Netherlands	0.0	0.0	0.0
Turkey or Morocco	+4.2[4]	+3.5[4]	+0.7
Surinam or Dutch Antilles	+0.8[4]	+1.0	−0.8
Other countries	+2.0[4]	+3.9[4]	−0.1

[1]The values for the nonreference children are relative to the values of the reference child, eg, a 5-y-old child with a mother born in Turkey or Morocco has a dmfs of 2.1 + 4.2 = 6.3.

[2]Decayed, missing, filled surfaces of primary dentition (dmfs) or of permanent dentition (DMFS).

[3]A Dutch child who eats sweets 1–5 times/d, has a moderate oral hygiene score of 2, and has a mother born in the Netherlands with an education level just below university level [adapted from Kalsbeek and Verrips (25)].

[4]Significantly different from the reference child, $P < 0.05$.

support a relation between the consumption of soft drinks and sweets and more caries in the lower socioeconomic class. Freeman et al (48) investigated determinants of reported snack consumption in adolescents in Belfast, Northern Ireland, and in Helsinki. Adolescents in Belfast had significantly higher levels of oral health knowledge, despite higher rates of consumption of snacks sweetened with sugars, than did Helsinki adolescents. In contrast, the adolescents in Helsinki had a more positive attitude toward their oral health. This study showed that knowledge may play a lesser role than attitude as a determinant of oral health behaviors.

RELATION BETWEEN CARIES AND DIET AT THE END OF THE 20TH CENTURY

Reports from the past 2 decades of the 20th century have shown that a small percentage of the variance in caries increase may be explained by dietary components since the introduction and use of fluoridated toothpaste (25, 44, 45, 49–57). The relation between sugars and dental caries is difficult to quantify because of inherent limitations. König and Navia (58) noted that *1*) variability in patterns of sugars consumption affects the duration of exposure of the teeth to sugars, *2*) dietary recalls or food diaries only provide an approximation of actual sugars consumption and food consumption patterns, *3*) patterns of sugars consumption are reported on an annual basis but caries formation can take several years, and *4*) caries prevalence is influenced by several factors that are difficult to control for, including the dietary mineral content (fluoride, calcium, and phosphorus), health care, oral hygiene habits, and education level. The following studies must be considered in light of these concerns.

Rugg-Gunn et al (53) reported an increase in caries of 5 carious tooth surfaces within 2 y in children (initially aged 11–12 y) who consumed >163 g sugar/d, whereas children who consumed less than half this amount (78 g) still developed 3.2 carious tooth surfaces. The only differences between children with no increase in caries development and those who developed ≥7 lesions within 2 y were a lower consumption of sweets (53 compared with 62 g/d) and sucrose-sweetened hot drinks other than tea (75 compared with 115 g/d) in the children with no increase in caries. Burt et al (57) reported a difference in caries increase in children (initially aged 11–15 y) who consumed on average 109 or 175 g sugar/d that was only 0.45 approximal carious tooth surfaces over 3 y. The children with no increase in caries consumed only 8 g less sugar and 11 g less fermentable carbohydrates in snacks than did the children who developed 2 approximal lesions, which was 4 times the group average. Rugg-Gunn et al (53) and Burt et al (57) showed an increase of 0.05–0.13 new caries surfaces per year in children aged 11–15 y for each 20-g (5-tsp) increase in daily sugar intake. Using the data from Burt et al's study, Szpunar et al (59) found that each additional 5-g/d intake of sugars was associated with a 1% increase in the probability of developing caries during a 3-y interval.

In a study by Kalsbeek and Verrips (25), 4% of the Dutch children consumed >5 sweet snacks per day (Table 4). These children had more caries than did the other children; however, the difference was only statistically significant for the primary dentition of the 5-y-old children (as opposed to the permanent dentition of the 8- and 11-y-old children). This study suggests that frequent consumption of sweets is still an important determinant for caries in the primary but not in the permanent dentition. One explanation might be that the caries in the 5-y-old children who snacked frequently had developed before the children had their teeth regularly cleaned with fluoride toothpaste by their parents.

At the 2001 National Institutes of Health Consensus Development Conference on Caries, Burt and Pai (60) reported that, of the 69 studies on diet and caries published between January 1980 and July 2000, only 2 showed a strong diet-caries relation. Of the other studies, 16 showed a moderate relation and 18 showed a weak relation. The authors of the 2 strong studies did not differentiate between sugars consumed as sucrose and those consumed as other monosaccharides and disaccharides; they concluded that diets that promote coronal caries also promote root caries (60). Burt and Pai (60) emphasized that the findings of their review differ from sugar-caries studies published in the decades before fluoride use. Although the papers reviewed indicated a decline in caries risk in relation to sugar intake, they attributed the relative decrease to fluoride use. The authors reported that although individual persons eating sugar are more likely to have increased cariogenic bacteria, the relation is not linear and the resultant caries rate differs by individual person. They concluded that "sugar consumption is likely to be a more powerful indicator for risk of caries infection in persons that don't have regular exposure to fluoride" (60).

Harel-Raviv et al (61) introduced the category "inexplicable findings" for studies showing that DMFT scores did not differ significantly despite a higher intake of sugar and greater snack frequency (49–51, 62–64). Cleaton-Jones et al (50, 51) found declining sucrose consumption in rural blacks who had a higher disease prevalence than expected, with few caries-free children.

TABLE 5
Caries-promoting activity and food sources of carbohydrates and sweeteners[1]

Category	Chemical structure	Examples	Caries-promoting potential	Food sources
Sugars				
	Monosaccharide	Glucose, dextrose, fructose	Yes	Most foods, fruit, honey
		High-fructose corn syrup	Yes	Soft drinks
		Galactose	No	Milk
	Disaccharide	Sucrose, granulated or powdered or brown sugar	Yes	Fruit, vegetables, table sugar
		Turbinado, molasses	Yes	
		Lactose	Yes	Milk
		Maltose	Yes	Beer
Other carbohydrates				
	Polysaccharide	Starch	Yes	Potatoes, grains, rice, legumes, bananas, cornstarch
	Fiber	Cellulose, pectin, gums, beta-glucans, fructans	No	Grains, fruits, vegetables
	Polyol-monosaccharide	Sorbitol, mannitol, xylitol, erythritol	No	Fruit, seaweed, exudates of plants or trees
	Polyol-disaccharide	Lactitol, isomalt, maltitol	No	Derived from lactose, maltose, or starch
	Polyol-polysaccharide	Hydrogenated starch, hydrolysates, or malitol syrup	No	Derived from monosaccharides
High-intensity sweeteners				
	Saccharin	Sweet and Low	No	
	Aspartame	Nutrasweet, Equal	No	
	Aceulfame-K	Sunett	No	
	Sucralose	Splenda	No	
Fat replacers made from carbohydrates		Carrageenan, cellulose gel/gum, corn syrup solids, dextrin, maltodextrin, guar gum, hydrolyzed corn starch, modified food starch, pectin, polydextrose, sugar beet fiber, xanthan gum	Unknown	Baked goods, cheese, chewing gum, salad dressing, candy, frozen desserts, pudding, sauces, sour cream, yogurt, meat-based products

[1]Reprinted with permission from reference 75. Sunett (Nutrinova, Somerset, NJ), Nutrasweet (Nutrasweet, Chicago), SweetnLow (Cumberland Packing Co, Brooklyn, NY), Equal (Merisant Co, Chicago), Splenda (McNeil Pharmaceuticals, Fort Washington, PA).

Oral hygiene was found to be the dominant variable. Stecksén-Blicks et al (65) found a mean DMFT score of 5.9 for 13-y-old children in an area where the children used 167 g total sugars/d, whereas in another part of Sweden, where the children consumed 147 g total sugars/d, the mean DMFT was found to be 11.4. The children consumed a mean of 5.5 and 4.9 meals/d, respectively.

SUGARS AND STARCHES IN THE DIET

Intake of sugars and starch

The US *Food Guide Pyramid* (66) and *Dietary Guidelines for Americans* (67) along with the European National Guidelines (34, 68) promote a diet rich in carbohydrates as whole grains, fruit, and vegetables. However, foods within these categories are sources of fermentable carbohydrates. Fruit and select dairy products, vegetables, and starches contain fermentable carbohydrates. A detailed discussion of sources of sugars, dietary guidelines, and terminology regarding sugars may be found in the articles from this workshop by Sigman-Grant and Morita (69) and Murphy and Johnson (70).

Intake of sugars in the United States increased significantly in the latter part of the 20th century; per capita consumption of added sugars increased by 23% from 1970 to 1996 (71, 72). Intake of added sugars increased from 1989–1991 to 1994–1996, representing an increase from 13.2% to 15.8% of total energy intake (73). Despite *The Food Guide Pyramid* (66) and the 2000 *Dietary Guidelines for Americans* (67), which encourage consumers to choose beverages and foods that provide a moderate intake of sugars, intakes of sugars in foods and fluids have increased.

In 2000 the most frequently reported form of added sugars in the US diet was nondiet soft drinks, which accounted for up to one-third of the intake of sugars (70, 74). An increasing availability of added sugars in the diets at home, in restaurants, and even in schools contributes to the rising intake of these foods. The general trend in Europe has been the stable use of sugars (Table 3), $\approx$34 kg $\cdot$ person$^{-1} \cdot$ y^{-1} (16).

Forms of sugars and starch in the diet

Sugars are a form of fermentable carbohydrate. Fermentable carbohydrates are carbohydrates (sugars and starch) that begin digestion in the oral cavity via salivary amylase. Sugars enter the diet in 2 forms: those found naturally in foods (eg, fruit, honey, and dairy products) and those that are added to foods during processing to alter the flavor, taste, or texture of the food (72) (Table 1). Examples of added sugars include white or brown sugar, honey, molasses, maple, malt, corn syrup or high-fructose corn syrup, fructose, and dextrose (**Table 5**) (75). Other disaccharides,

particularly trehalose, threhalose, and isomaltose, have a lower cariogenic risk than does sucrose. Starches are subsequently digested by salivary amylase to oligosaccharides, which may be fermented by the oral microflora. According to Lingstrom et al (76), only the gelatinized starches are susceptible to breakdown by salivary amylase into maltose, maltotriose, and dextrins.

Cariogenic risks of foods and beverages

The focus of this paper is sugars, but sugars are often eaten in combination with starches and many of the same issues arise in determining the cariogenic risk associated with individual foods. Key issues to consider in determining dietary cariogenic, cariostatic, and anticariogenic properties are food form, frequency of sugars consumption and other fermentable carbohydrates, retention time, nutrient composition, the potential of the food to stimulate saliva, and combinations of foods (37, 76). Caries risk also depends on individual host factors. The presence of any individual characteristics—such as low or high salivary pH, genetic predisposition, prior caries history, use of medications, incidence of systemic or local diseases that affect the immune system, and personal hygiene habits—also play a role in the associated caries risks of particular foods.

The cariogenic potential or associated risk of sugars and other fermentable carbohydrates has been extensively reviewed (37, 60, 76–78). Studies have attempted to estimate the cariogenic potential of foods and beverages on the basis of the decrease in plaque pH caused by food (79). Stephan and Miller (40) published the first research describing the decrease in plaque pH after exposure to fermentable carbohydrates. The cariogenic risk associated with individual foods is challenging to determine in human studies because of the variability in salivary flow and salivary and plaque pH, the eating experience (frequency and food combinations), bioavailability of starch-derived sugars (75), retention time of food in the oral cavity, and potential interactions between starches and sugars. No epidemiologic evidence supports the cariogenic risk associated with select starch products without added sugars, such as rice, potatoes, and bread (80). Luke et al (81), however, showed the caries risk of white bread in the laboratory setting in humans. In a rat study, Mundorff et al (82) showed the potential caries risk associated with French fries. Lingstrom et al (76) concluded that it is premature to consider food starches in modern diets to be safe for teeth.

Food form

The form of the fermentable carbohydrate directly influences the duration of exposure and retention of the food on the teeth. Prolonged oral retention of cariogenic components of food may lead to extended periods of acid production and demineralization and to shortened periods of remineralization. Duration may also be influenced by the frequency and amount of fermentable carbohydrate consumed (76, 77). Liquid sugars, such as those found in beverages and milk drinks, pass through the oral cavity fairly quickly with limited contact time or adherence to tooth surfaces. However, fluid intake patterns can influence the caries risk of the beverages. Holding sugar-containing beverages in the oral cavity for a prolonged time or constant sipping of a sugared beverage increases the risk of caries. Long-lasting sources of sugars, such as hard candies, breath mints, and lollipops, have extended exposure time in the oral cavity because the sugars are gradually released during consumption.

Oral clearance

Oral clearance properties vary by individual person and depend on metabolism by microorganisms, adsorption onto oral surfaces, degradation by plaque and salivary enzymes, saliva flow, and swallowing. Most carbohydrates will be cleared by these simultaneous mechanisms. Luke et al (81) showed this clearance to be relatively slow. Retentiveness of foods is not the same as stickiness. A caramel or jellybean may be sticky, but its retentive properties are fairly low and they are cleared from the oral cavity faster than are retentive foods such as cookies or chips. Luke et al (81) found that after rinsings with 10% solutions, sucrose cleared more rapidly from the saliva than did glucose, fructose, or maltose. Products of sucrose metabolism (ie, glucose and fructose) were not detected after the sucrose rinse in contrast with after the maltose rinse. A test of the salivary clearance of 3 different fermentable carbohydrates (white bread, bananas, and chocolate) showed that the clearance of residual carbohydrates (sucrose, fructose, and maltose) from bananas and chocolate was marginally faster than that from white bread. Carbohydrate residues from the 3 foods were still present in the mouth 1 h after ingestion. Glucose produced by chocolate and bananas was higher initially and cleared more rapidly than that produced by bread, which was initially lower because of the time needed for starch breakdown by amylase.

In comparable studies of salivary carbohydrate, Edgar et al (83) and Bibby et al (84) found that high-starch foods had slow salivary clearance rates. Kashket et al (77) found particles of food with high contents of starch, such as creme sandwich cookies and potato chips, to be retained on teeth in larger amounts than foods that contained little starch, such as milk chocolate, caramels, and jelly beans. In a subsequent study, Kashket et al (78) showed that the starch particles retained on the tooth surface were hydrolyzed to sugars (maltose and maltotriose), depending on the processing of the food starch. Gelatinization of starches by various degrees of heating enhances the ability of salivary amylase to break down the starches and stimulates a decrease in pH (76, 78). Doughnuts and potato chips processed at the highest temperature gave rise to the highest amount of the sugars compared with the other test foods. The study showed that the longer that foods are retained in the oral cavity, the greater the potential the starch has to break down into sugars and contribute to the caries process. The initial content of sugars was not the culprit; rather, it was the type of starch and extent of starch retention time in the oral cavity that determined the relative cariogenic risk of the food (78).

Frequency

The frequency of consumption seems to be a significant contributor to the cariogenicity of the diet (58, 85–88), although Bowen et al (88) concluded that it is not the frequency of ingestion per se that is related to the development of caries but the time that sugars are available to microorganisms in the mouth. The importance of frequency is clear when caries is regarded as the outcome of the alternation of demineralization and remineralization. Higher frequency means more demineralization and less remineralization. The duration of the decrease in pH after intake of a cariogenic food is an important confounder in this relation. Traditionally, and in many educational models, the decrease in pH lasts 30 min (40). pH telemetry measurements, however, show that after plaque is a few days old, the decrease in pH can last for several hours, unless the site is actually cleared by a stimulated salivary flow or by removal of impacted food (89). These data suggest

that local oral factors that influence the accessibility of saliva may modify the cariogenicity of food.

Nutrient composition

Diet and nutrition may favor remineralization when their content is high in calcium, phosphate, and protein. In experiments using processed cheese and sucrose solutions, Jensen and Wefel (90) showed that processed cheese was anticariogenic. Cheese consumption followed by a 10% sucrose solution resulted in a pH of 6.5 compared with a pH of 6.3 for cheese alone and a pH of 4.3 for sucrose alone. When the intakes of sugars and cheese were compared in the Forsyth Institute Root Caries Study, Papas et al (91, 92) showed that, independent of the consumption of sugars, cheese protected against coronal and root caries. These findings, relative to root caries, are particularly important for older adults, many of whom consume a diet rich in simple sugars and are at risk of root caries. Mechanisms proposed to explain the anticariogenic effects of cheeses are as follows: *1*) increased salivary flow and the subsequent buffering effect, which can neutralize plaque acids; *2*) inhibition of plaque bacteria and the effect of that inhibition on reducing the amount of bacteria, thereby reducing acid production; and *3*) intake of increased alkaline substances, calcium, inorganic phosphate, and casein, which decrease demineralization and enhance remineralization (93).

Acid content

The acidity of individual foods can precipitate erosion. The erosive potential, however, depends also on whether the oral buffer systems can neutralize the food. Because the critical pH for enamel dissolution is 5.5, any food with a pH lower than 5.5 may contribute to or stimulate erosion. In persons with adequate saliva and good oral hygiene habits, these fluids and foods pose minimal risk when consumed as part of a balanced diet. Large doses of chewable vitamin C may also cause a decrease in pH because of its citric acid content, which contributes to tooth erosion (80).

Polyphenols

Polyphenols such as tannins in cocoa, coffee, tea, and many fruit juices may reduce the cariogenic potential of foods. In vitro experiments have shown that these polyphenolic compounds may interfere with glucosyltransferase activity of mutans streptococci, which may reduce plaque formation (94, 95). In rat experiments, tea polyphenols reduced caries (95, 96).

Sugar alcohol–based products

Sugar-free gums can stimulate saliva, increasing the clearance of sugars and other fermentable carbohydrates from the teeth and the oral cavity and increasing buffer capacity. Tooth-friendly polyols include sorbitol, xylitol, mannitol, erythritol, and isomalt. However, xylitol—a 5-carbon sugar that oral microflora cannot metabolize—has additional anticariogenic effects attributable to antimicrobial action, stimulation of saliva resulting in increased buffer activity and an increase in pH, and enhanced remineralization (97, 98). Sorbitol-sweetened gums simulate saliva without causing a drop to the critical pH and have been shown to be equal to xylitol gum in terms of caries control (99).

INFLUENCE OF LIFE SPAN ON CARIES RISK

Caries patterns in children, adults, and elderly vary, as do eating patterns. In all age groups, eating frequency, retentive and nutrient properties of food, and oral hygiene habits play a role in determining caries risk. Infants and children are particularly susceptible to early childhood caries (3). Caries in early childhood typically occurs when bedtime or naptime habits include lying with a bottle filled with formula, juice, milk, or another sweetened beverage. Although the content of sugars in the diet plays a pivotal role in caries patterns, adoption of good oral hygiene habits, a balanced diet, and limited intake of high-sugar between-meal snacks will reduce the risk of caries. Root caries are more prevalent in the elderly than in other age groups (91, 92). Papas et al (91, 92) showed that elderly persons whose sugar intakes were in the highest quartile had significantly more root caries than did persons whose sugar intakes were in the lowest quartile. Persons with a sugar intake in the highest quartile consumed approximately twice as many sugars in the form of liquids (sweetened coffee or tea) and sticky sugars (92).

DIETARY RECOMMENDATIONS FOR REDUCING THE RISK OF ORAL INFECTIOUS DISEASE

The primary public health measure for reducing oral infectious disease, from a dental perspective, is the use of topical fluorides (as toothpastes) and water fluoridation at appropriate levels of intake (100). The primary public health measure, from a nutrition perspective, is dietary balance and moderation in the adherence to dietary guidelines, food guides, and dietary reference intakes (101). Dietary habits regarding the consumption of naturally occurring and added sugars, including the frequency of eating, the form of the sugars-containing food, the sequence in a meal, the presence of buffers such as calcium, and the duration of exposure greatly affect caries risk and should be addressed in dietary recommendations. Likewise, the use of fluoridated toothpaste and water greatly affects caries risk. Persons at high risk should be attentive to their consumption patterns, moderate their intakes of sugars (naturally occurring or added) and other fermentable carbohydrates, and use fluoridated toothpaste. A diet void of naturally occurring sugars and fermentable carbohydrates is not feasible, and a diet void of added sugars would be difficult to achieve and maintain. Maintaining a moderate use of added sugars and sweets is a prudent recommendation found in the US *Dietary Guidelines* for *Americans* (67).

A diet history concerning food intake patterns, diet adequacy, consumption of fermentable carbohydrates (including naturally occurring and added sugars), and the use of fluoridated toothpaste is a strategy for health professionals to use to determine the diet-related caries risk habits of persons. Diet recommendations for oral health are as follows (1, 2, 58, 75):

1) eat a balanced diet rich in whole grains, fruit, and vegetables and practice good oral hygiene—particularly the use of fluoridated toothpastes—to maximize oral and systemic health and reduce caries risk.
2) eat a combination of foods to reduce the risk of caries and erosion; include dairy products with fermentable carbohydrates and other sugars and consume these foods with, instead of, between meals; add raw fruit or vegetables to meals to increase salivary flow; drink sweetened and acidic beverages with meals, including foods that can buffer the acidogenic effects.
3) rinse mouth with water, chew sugarless gum (particularly those containing sugar alcohols, which stimulates remineralization), and eat dairy product such as cheese after the consumption of fermentable carbohydrates.

4) chew sugarless gum between meals and snacks to increase salivary flow.
5) drink, rather than sip, sweetened and acidic beverages.
6) moderate eating frequency to reduce repeated exposure to sugars, other fermentable carbohydrates, and acids.
7) avoid putting an infant or child to bed with a bottle of milk, juice, or other sugar-containing beverage.

SUMMARY AND CONCLUSIONS

The relation between sugars and oral health is dynamic. Although sugars, both naturally occurring and added, and fermentable carbohydrates stimulate bacteria to produce acid and lower the pH, several dietary factors affect the caries risk associated with fermentable carbohydrates. Topical fluoride in toothpaste and fluoridated water supplies have had a significant effect on reducing caries risk globally. Eating patterns, nutrient composition, duration of exposure, food form, saliva, and supplemental use of fluoride in drinking water, toothpastes, and other agents all interact and affect caries development. Integration of oral hygiene instruction into diet and oral health education will help to reduce caries risk. Health professionals, particularly dental and nutrition professionals, must recognize the relation between oral health and diet and manage patients accordingly.

Further research is needed to determine anticariogenic strategies to reduce risks posed by sugars and other fermentable carbohydrates, explore the use of sugar alcohols and dairy products to prevent caries, and determine the cariogenicity of different starches and starch-sugar combinations. The effect of sugars on plaque pH and decay patterns in mixed diets merits additional human studies. Longitudinal studies are needed to explore caries risk over time in persons with different sugar and meal-snack consumption patterns. Educational and behavioral research is needed to determine strategies to moderate the frequency of added sugars and to increase compliance with the *Dietary Guidelines for Americans* and the dietary reference intakes.

Sugars and oral health are integrally related. Dietary guidelines for the prevention and management of dental caries provide a framework for consumers and health professionals to use in managing the intake of sugars.

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