Sugar and Dental Caries: A Review of Human Studies

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Summary. Studies of special population groups, epidemiological surveys, controlled longitudinal studies of humans, and longitudinal studies on the effect of sugar substitutes indicate that frequent or high intake of sugary foods predisposes to dental decay. The relation is not always clear-cut, and most studies have important methodological problems and limitations. Longitudinal measurements of caries increments combined with multiple dietary histories are needed to clarify the association between caries and eating habits. The relative cariogenicity of specific foods can be assessed by a combination of in vitro tests, human in vivo tests, and experimental caries in animals. Human diets, however, vary in food items eaten and the frequency and sequence of eating, and these factors can affect the cariogenicity of a food. Therefore, reported correlations must be interpreted with caution.

Dental caries is a pathological process of localized destruction of tooth tissues by microorganisms. There are several different types: pit and fissure caries, smooth surface caries, root caries, and deep dentinal caries. The process of tooth destruction involves dissolution of the mineral phase, consisting primarily of hydroxyapatite crystals, by organic acids produced by bacterial fermentation. Evidence points overwhelmingly to dietary sugar as the major factor in the etiology of coronal caries (pit and fissure and smooth surface caries) (*1*), but the role of diet in root caries is not well understood. In this article, caries will refer to coronal caries only.

Dental caries is a multifactorial disease involving three principal factors: the host, particularly the saliva and teeth; the microflora; and their substrate, the diet. Caries research has been devoted mostly to host factors and the microflora. The caries-preventive benefits of fluoride, whether used systemically in the water and as a dietary supplement or applied topically to the tooth surface, have been thoroughly investigated, and the possibility of immunization against specific cariogenic organisms has received considerable attention.

Research has advanced our understanding of this disease, of the specific oral flora associated with the carious lesion, the transmissibility of the flora, and the mechanisms involved in the adherence of the flora to teeth and the formation of dental plaque. The role dietary sucrose plays in determining the numbers of cariogenic organisms able to colonize on tooth surfaces has been established (*2*). For example, restricting the dietary intake of sucrose limits the numbers of cariogenic *Streptococcus mutans* found in the dental plaque (*3, 4*), because when the intake of sucrose is low, this organism will colonize only in small numbers (*5–7*).

To date studies of the relation between diet and human caries have been limited, but there is renewed interest in industry and government in attempts to measure the cariogenicity of specific foods. Such information may lead to the development of less cariogenic foods.

The purpose of this article is to review studies in humans on the decay-promoting role of particular foods and beverages and to point out the limitations of many of the clinical trials and epidemiological surveys.

Historical Observations

Aristotle, in *Problems*, observed that soft, sweet figs adhered to the teeth, putrified, and produced damage. Johnannes Arculanus, in his 15th-century *Practica Particularium Morborum Omnium*, wrote a regimen for saving the teeth that included a rule to "avoid rich and sticky foods, especially those which are sweet and sticky as are confections of honey and dried figs." W. D. Miller, writing in 1890, demonstrated that different kinds of foods (bread, sugar, but not meat) mixed with saliva and incubated at 37°C could demineralize teeth (*8*). He considered "saccharides and amylaceous [starchy] foods" as particularly cariogenic.

There are numerous anecdotal reports associating certain foods with caries. A rampant form of caries in Sweden was linked to the frequent consumption of pastilles (*9*). Severe caries has also been described in children from French Polynesia who consume large amounts of a sugar-laden confection that is molded into place between the teeth and the lips or cheeks and left there until it dissolves (*10*).

Special Population Groups

A study of a limited number of children of dental school faculty in the United Kingdom (*11*) showed that those whose sweet and biscuit eating was restricted between meals and at bedtime had fewer caries than either those for whom these products were restricted only at bedtime, or those whose sugar consumption was not restricted.

There have been several reports (*12*) that children of Seventh Day Adventists, who are counseled to restrict their intake of highly refined starches, sugar, and sticky foods and are urged to refrain from eating between meals, have less decay than other children; the data, however, are limited.

Restriction of sugar is an important aspect of the dietary management of diabetics. However, data on caries prevalence in diabetic children have not shown consistent differences from those of normal children. Some studies found fewer caries (*13*), whereas others found no difference (*14*). These inconsistent findings may reflect differences in how rigidly individual diabetics restrict their dietary sugar.

Children with intestinal sucrase deficiency, who must avoid foods containing sucrose, had less decay and fewer numbers of *S. mutans* in their plaque than normal children (*15*). In contrast, children who took sugar-based syrup medicines daily for at least 6 months for chronic medical disorders had signifi-

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cantly more caries than a control group of similarly aged children who did not receive syrup medicines (*16*).

Hereditary fructose intolerance (HFI) is an uncommon metabolic disorder caused by drastically reduced activity of fructose-1-phosphate aldolase in the liver, lower renal cortex, and small bowel. The most common dietary sources of fructose are foods containing sucrose, which has one molecule of glucose and one of fructose. People with HFI have a strong aversion to all sweets, cakes, candies, and fruit; they eat foods containing glucose, galactose, lactose, and starch, such as dairy products, bread, pasta, rice, and potatoes. Their intake of sucrose averaged only about 2.5 grams per day, whereas a similarly aged control population consumed about 48.2 grams per day (*4*). This restricted sucrose intake is reflected in the fact that *S. mutans* is isolated less often and in greatly reduced numbers from the plaque of people with HFI (*17*); moreover, persons with HFI are either caries-free or have little decay (*4, 5, 18*). The low prevalence of caries in these patients indicates that starchy foods do not produce decay, whereas sugary foods do.

Epidemiological Observations

Circumstantial evidence linking consumption of sucrose and prevalence of caries can be found in epidemiological surveys among such populations as Australian aborigines, New Zealand Maoris, Eskimos, Ghanaians, and Tristan da Cunhans. Before exposure to European-type diets, these people ate little sucrose. As their diets changed to include more products containing sugar, caries prevalence increased (*5*).

In England, which imports most of its sucrose, records of the last 100 years show a steady increase in per capita consumption of sucrose, from about 20 pounds per year in 1820 to over 100 pounds per year today. Present consumption of sucrose in the United States is about the same. This represents 15 to 20 percent of an individual's caloric requirements. Concomitant with this increased consumption of sucrose has been an almost parallel rise in the prevalence of caries (*19*). Conversely, surveys in Europe and Japan demonstrated that caries was dramatically reduced during periods of wartime restrictions of sugar, syrup, and all sugar products (*20*).

Data from 18 countries on the relation of sugar consumption and average numbers of decayed, missing, and filled (DMF) teeth of children 10 to 12 years old (Fig. 1) (*21*) reveal a high positive correlation ($r = .95$, linear regression analysis). Analysis of data from the World Health Organization's Global Oral Epidemiology Bank also revealed a statistically significant positive correlation ($r = .72$, $P < .005$) between sugar supplies and dental caries for 12-year-old children in 47 populations (*22, 23*). A high prevalence of caries, or low percentage of persons free from caries, was found among teen-age South Africans with a high sugar intake. Conversely, rural blacks of similar age who ate less sugar had fewer caries (*24*).

A methodological problem in all these studies is that per capita consumption of sucrose is estimated by dividing sugar supplies by population, which yields a value that exceeds the amount reported on dietary recall questionnaires (*25*). The data on consumption thus actually represent the disappearance of sugar—the average of the sum of actual consumption, industrial use for purposes such as fermentation of alcohol, and waste, both industrial and household, such as from spillage and discarded leftovers (*26*). The data on disappearance may be valid only in relative ranking of some populations and countries.

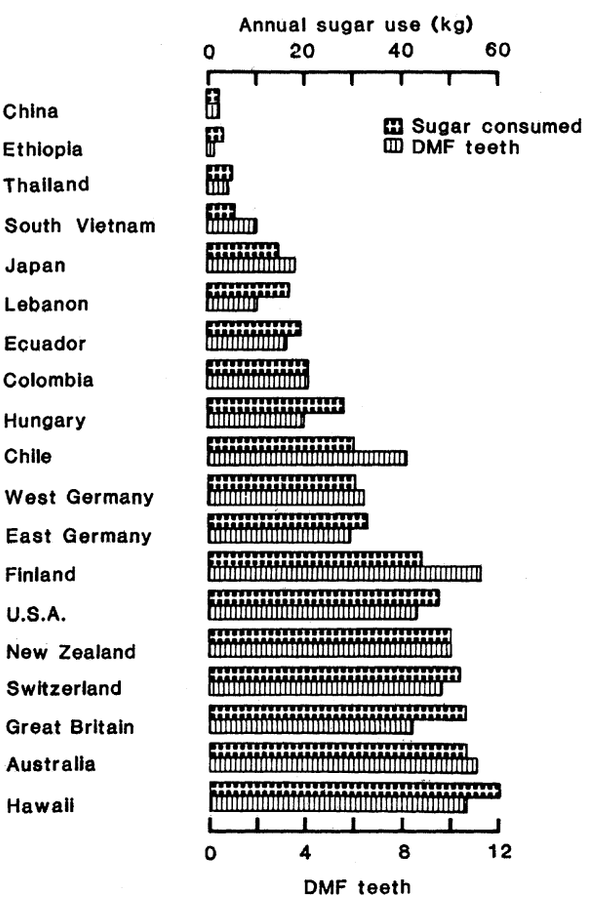

Fig. 1. Cumulative dental decay prevalence, expressed as decayed, missing, and filled (*DMF*) permanent teeth, in children ages 11 to 12; and corresponding annual 1959 per capita sucrose utilization data for 18 countries and the state of Hawaii, from the Food and Agriculture Organization of the United Nations. [Adapted from (*21*)]

Controlled Longitudinal Human Studies

In a study at Vipeholm, a mental institution in southern Sweden, 436 adult patients on a nutritionally adequate diet were observed for several years. They were found to develop caries at a slow rate. Subsequently, the patients were divided into nine groups to compare the effect of various changes in their carbohydrate intake. Sucrose was included in the diet as toffee, chocolate, or caramel, in bread, or in liquid form. Caries increased significantly when foods containing sucrose were ingested between meals. Not only the frequency but the form in which sucrose was ingested was important: sticky or adhesive forms were more cariogenic than forms which were rapidly cleared from the mouth (*27*). After 2 years on the test diets, the patients were again placed on the control diet, and the caries activity reverted to the pretest pattern.

This study demonstrated that it is possible to increase the average consumption of sugar (from about 30 to 330 grams per day) with little increase in caries (0.27 to 0.43 new carious surfaces per year), provided that the additional sugar is consumed at meals in solutions. These observations in an institutional popula-

Table 1. Effect of variation in a single food category on caries experience. Comparison of annual caries increments of control subjects eating a "normal" diet and subjects on a sugar-supplemented diet. Abbreviations: DMFS, decayed, missing, and filled surfaces of teeth; DFS, decayed and filled surfaces of teeth.

Length of study (years)	Age of subjects (years)	Control			Sugar-supplemented			Statistical analysis	Comment	Reference
		Diet	Number of subjects	Caries increment*	Diet	Number of subjects	Caries increment*			
					Soft drink					
3	8 to 21	Water, twice a day	132	3.42 DMFS	Sugary soft drink, twice a day	119	4.05 DMFS	No significant difference for all surfaces; test and statistic not provided	Significantly more buccolingual caries in soft drink group; poor oral hygiene; high caries activity in both groups	(*35*)
					Presweetened breakfast cereal					
3	13	Cereal non-eaters	73	2.14 DMFS	Cereal eaters	302	1.73 DMFS	No significant difference, $t = -1.7$; test partialing out effects of use, sex, and initial DMFS also showed no difference	Cereals mostly eaten only at breakfast, not every day; noneaters defined as < 7 ounces in 4 days	(*34*)
2	7 to 11	Regular cereal	979	1.86 DFS‡	Presweetened cereal	979	1.93 DFS	No significant differences across consumption levels, analysis of variance; no significant difference between high consumers of regular and presweetened cereals, $t = 1.38$	Mean data for low, medium, and high consumers; subjects categorized by type of cereal consumed and encouraged to eat cereal once a day	(*33*)
3	3 to 18	Cereal non-eaters	46	1.63 DMFS	Cereal eaters (67 percent presweetened)	80	1.75 DMFS	No significant difference, $t = 0.43$	Cereal eaters averaged 8 ounces a month; noneaters defined as < 8 ounces a month	(*36*)
					Sugary chewing gum					
2.5	12.5	Sugarless gum, five times a day	149	1.97DMFS	Sugary gum, five times a day	129	2.37 DMFS	Significant difference, $t = 1.7$, $P \leq .05$	Gum chewed five times a day for 20 minutes; institutional subjects all served same foods; sugar gum group had 80 percent higher caries in posterior proximal surfaces	(*37*)
2	6 to 12	No gum	157	1.92 DFS	Sugary gum, twice a day	164	2.61 DFS	Significant difference, analysis of variance; $F = 3.71$, $P < .05$; pairwise, $t = 2.39$, $P < .01$	Gum chewed two times a day, supervised at school	(*38*)

*Caries increments are determined by scoring DMFS or DFS at the beginning and end of the dietary study period, usually 2 to 3 years, and dividing the difference by the duration of the study to obtain annual increment.

tion, however, do not mean that the total amount of sugar consumed does not affect the level of dental caries. In the Vipeholm study the conditions were artificial because some groups were allowed to consume sugary foods only at meals. When there is free access to sugar-containing foods and beverages, however, they may commonly be eaten between meals (*28*).

Sugar and other refined carbohydrates were excluded from the diet of children at Hopewood House, an orphanage in Bowral, New South Wales, Australia, which at one time accommodated 82 children. Dental surveys of these children from the ages of 5 to 13 revealed an average score for decayed, extracted, and filled deciduous teeth (def) and for decayed, missing, and filled permanent teeth (DMFT) of 1.1, about 10 percent of that of the public school population of the same age. These children were examined for a number of years, and their caries increments were consistently less than those in the general population. When the children were relocated and no longer adhered to the original diet, there was a steep increase in DMFT (*29*). This finding indicates that their teeth had not acquired permanent resistance to caries but had not decayed in the earlier period because few cariogenic foods were ingested.

Longitudinal Studies on Sugar Substitutes

In Roslagen, Sweden, the cariogenicity of candies made with sucrose was compared with that of candies with Lycasin, a hydrogenated potato starch hydrolyzate containing a mixture of sorbitol, maltitol, maltotriitol, and higher saccharide alcohols (*30*). Children 3 to 6 years old were observed for 1½ to 2½ years. The study began with 225 subjects; after 2 years there were 113. In addition, replacement of sucrose was far from complete—almost all the children in the Lycasin group ate some candies containing sucrose, and some drank lemonade with sucrose. The difference in caries increments between the two groups was not statistically significant (*t*-test). This study illustrates the difficulty in attempting to replace products containing sugar in an unsupervised setting where compliance is lacking.

In Turku, Finland, the caries increment, amount of plaque formation, and composition of plaque flora were compared in adults, average age 27.5 years, on unsupervised diets with either sucrose (N = 35), fructose (N = 35), or xylitol (N = 47) as the sweetening agent (*31*). The three sugars were used in about 100 products, such as tea, soft drinks, juices, porridges, jams, marmalade, pastries, sweets, chocolates, bonbons, chewing gum, marinated herring, pickles, mustard, and cough mixture. After 1 year, a dramatic reduction in caries increments was found in the group consuming products containing xylitol—more than 85 percent fewer carious lesions than in the sucrose group, on the basis of all types of caries, ranging from incipient (only visible changes) to actual cavitation. If these incipient lesions are disregarded and the usual diagnostic criteria for dental caries are employed, the reduction in DMFS was 56 percent after 1 year and 60 percent after 2 years (*32*). Patients in the xylitol group had less plaque and lower colony counts of *S. mutans* in their plaque. Xylitol-containing soft drinks could produce transitory diarrhea, but only one person dropped out of the study because of this problem.

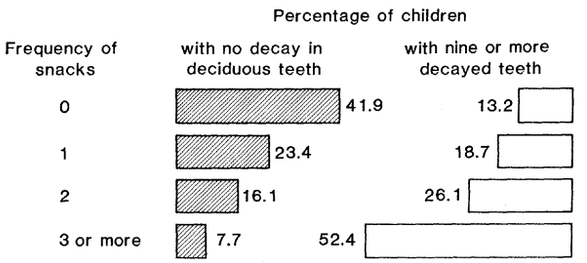

Fig. 2. Percentage of children 5 and 6 years of age with no decay in deciduous teeth and with nine or more decayed deciduous teeth, according to their mothers' recollection of the frequency of between-meal snacks in the previous 24 hours. [Adapted from the data of Weiss and Trithart (*39*)]

Longitudinal Clinical Trials on Specific Food Items

A number of studies (*33–38*) have compared caries increments in children who consumed certain foods or beverages containing sucrose with the increments in groups of similarly aged childen who did not consume the items. Soft drinks, presweetened breakfast cereal, and chewing gum (Table 1) were tested. In these studies, only the one item was varied and the rest of the diet was kept the same as in the control population; in other words, the intake of sugary foods was not totally controlled. Consequently, control groups as well as test groups often had a high caries activity—about two new decayed surfaces annually—which might explain the absence of significant differences in some studies (*33–36*).

In the two studies on the effect of chewing gum (*37, 38*), however, the children using the gum containing sucrose had more caries. In one study, the children chewed five sticks of gum daily and had a 33 percent higher caries increment on all surfaces and an 80 percent higher caries increment on posterior proximal surfaces (between the teeth) than those who used sugarless gum under the same conditions (*37*). In the other study, the children who chewed a gum with 60 percent sucrose and 20 percent glucose twice daily between meals had a 36 percent greater increase in new decayed and filled surfaces than children who were not receiving chewing gum (*38*). The slow release of sugar from the chewing gum base and the use of chewing gum between meals may make it especially cariogenic. Presweetened cereals eaten only once a day may not be as cariogenic as sugary gum, but their cariogenic potential would probably increase when they are eaten more frequently.

In the study on soft drinks (*35*), both the control and the test populations were inmates of a mental institution and had poor oral hygiene and high caries activity (Table 1). A comparison of caries increments on all DMF surfaces showed no statistically significant difference between the two groups; but when buccolingual surfaces (the cheek and tongue sides of the teeth) only were compared, the test group (consuming soft drinks twice a day) had statistically significant higher increments.

Cross-Sectional Caries Surveys and Dietary Recall

A survey of the dietary habits of preschool children (average age 5 years, 9 months) indicated that the typical between-meal items they consume, such as gum, candy, pastries, soft drinks, and ice cream, are high in sugar content. A direct and consistent relation was found between the frequency of between-meal eating and caries prevalence in these children (Fig. 2) (*39*). The frequency of eating various foods and the prevalence of caries in 910 eighth grade Hawaiian schoolchildren was measured (*40*). A 24-hour dietary recall questionnaire with 39 food and beverage items, arranged in eight logical groups, was completed by each child on three successive mornings, one of which was a weekend day. Multi-

ple regression analysis was used to test the effect of dietary factors on caries, and only two food groups—candy and gum, which are eaten predominantly between meals—demonstrated a statistically significant association with DMF teeth. The frequency of eating breads, buns, rolls, and breakfast cereals, all of which are usually consumed at meals, was negatively associated with caries. The candy and gum group included chocolate candy, caramels, toffees, hard candy, soft jelly candy, marshmallows, and gum.

As part of a ten-state survey of nutritional status (*41*), dietary data were collected by the 24-hour recall method for black, white, and Spanish-American children from 10 to 16 years of age. In states with relatively high average family incomes, all three ethnic groups showed a positive association between DMF teeth and between-meal consumption of carbohydrates. In states with low family incomes, only black children had a positive association. Not all carbohydrates are cariogenic; therefore, the specific type of between-meal snack (*42*) should be considered in attempts to establish such an association.

In a study of high school students 14 to 17 years old, no relation of between-meal eating habits and dental caries was detected (*43*). The students filled out a questionnaire indicating whether food items such as candy, cake, ice cream, bread, crackers, and so forth, were "never eaten," "sometimes eaten," or "usually eaten" between meals. The caries prevalence was compared with differences in patterns of between-meal eating, particularly the frequency and type of foods containing sucrose. Snacks were classified as "sticky sugar," "sugar," or "low sugar."

Other investigators obtained 5-day dietary records from the parents of 234 first grade and 223 seventh grade children (*44*) to compare "refined" and total carbohydrate consumption with caries prevalence. They found no statistically significant correlations, but the parents were given scant instructions—only a sample diet record—and the method for defining "refined" carbohydrate is not clear. Also, the food values did not include data on the sugar content of foods.

Another study also reported a lack of correlation between dietary carbohydrate intake and dental decay (*45*), and the investigators further concluded that dietary sucrose intake is not important in the etiology of caries. These conclusions were based on dental examinations of 131 patients by dental students at Emory University. Because no details were provided about this population nor about the method of obtaining the dietary history, little weight can be given to the conclusions.

Parents of caries-free 3- to 5-year-old children were surveyed as to their child's preference for and frequency of consumption of breakfast cereals, baked foods, candy, and sweet drinks; only cereal consumption correlated directly with caries experience (*46*). The investigators concluded that a one-time survey of consumption habits is not an adequate basis for evaluating factors contributing to caries.

Multivariate analysis was used to investigate the relation between dental caries and various aspects of sugar consumption, with data from the First National Health and Nutritional Examination Survey (NHANES) (*47*). Results showed that (i) total calories ingested from the three principal sugar-containing food categories had little relation to DMF values, (ii) total calories from sugary foods ingested between meals had a stronger relation, (iii) the relation between DMF and frequency of consumption of between-meal sweet snacks was strongest, and (iv) consumption of nonsugary snacks between meals was related to DMF in some age groups.

This survey has several serious limitations. First, in conducting the dental examinations, the investigators did not dry or probe the teeth unless they showed overt signs of decay. The sensitivity of detecting caries is considerably increased when they are dried. Second, neither the total sugar content nor the sucrose content of foods was included in the computer program. The three food groups considered "sugar-rich" were cereals and grain products, sugar and primarily sugar products, and desserts and sweets. A closer look at these three food groups suggests that they are too broad and include many items that may not be cariogenic or are of low cariogenicity, such as crackers and pretzels. Furthermore, another food group, milk and milk products, was not considered sugar-rich in the analysis although it included items that may have a high sucrose content, such as ice cream (*48*) and milk puddings, flavored milk drinks, and fruit-flavored yogurt. These inconsistencies may explain why there was an apparent, although small, association between frequency of ingestion of so-called nonsugary snacks and caries experience in some groups.

Canadian investigators studied the diets of 360 children in three age groups—5 to 6 years, 11 to 12 years, and 16 to 17 years—from city, urban, and rural communities in Ontario, some of which had fluoridated and some nonfluoridated water supplies (*49*). A detailed nutrient computer program was used to analyze the data obtained from 7-day food diaries for each child, as compiled by dietitians who interviewed the families. The initial results show no correlation between total sugar intake (mean 120.4 pounds of sugar per child per year) and past caries experience, but a statistically significant correlation between the prevalence of dental caries and both the total sugar eaten between meals and sucrose eaten between meals. As in other studies, there was no significant relation between dental caries status and consumption of sugar and cereal at breakfast. The frequency of intake of selected food items also was compared with caries prevalence. Because only cookies showed a positive correlation ($P < .01$), the investigators concluded that items looked at in isolation from a total diet are not likely to show a statistical correlation with the prevalence of decay (*50*).

At Columbia University the dietary patterns of caries-resistant (DMFT = 0, $N = 47$) and caries-susceptible (DMFT > 15, $N = 33$) adults were compared; data from a 3-day diet history, 24-hour diet recall, and a questionnaire were used (*51*). No difference was found in the frequency of exposure to sucrose between the two groups; both had approximately seven exposures per day. To compute the total sucrose intake, the Case Western Reserve data bank on sucrose content of foods was used. Analyses indicated low consumption of sucrose for both groups (less than 20 grams per day), suggesting that this data bank may be inadequate.

The surveys reviewed have many limitations. In some, snacking or dietary habits were judged on the basis of a one-time survey of a 24-hour dietary recall of food consumption, and day-to-day variation in individual patterns was not recorded. In the NHANES survey a questionnaire on the frequency of eating certain foods for the preceding 3 months was administered, but the information was not used. Undoubtedly the major limitation of cross-sectional studies is that the caries data are cumulative. The DMF index measures lifetime caries experience, and therefore may not have a direct relation to a person's current dietary profile. Furthermore, there is also a treatment effect on DMF score, because the criteria used by a dentist in deciding whether to fill a tooth may not be the same as the criteria an epidemiologist uses in deciding whether a tooth is decayed. Clearly, longitudinal measure-

ments of caries increments and multiple dietary histories are essential in any study of the association between caries and eating patterns.

In most of the studies, sugar or sucrose content of the foods was not determined, or even known, yet some investigators attempted to categorize different food groups as sugar-rich or sugary and low-sugar or nonsugary. Any correlations based on such vague and nonquantifiable definitions of foods are inappropriate.

Conclusion

Studies of the dynamics of sucrose metabolism by cariogenic organisms (*52*), investigations of experimental caries in animals (*53*), and some clinical and epidemiological observations of the relation of sucrose intake and caries experience all provide compelling evidence that the proportion of sucrose in a food is one important determinant of its cariogenicity. An approximate ranking of the cariogenicity of specific foods could be made on the basis of a combination of in vitro tests (for example, plaque-forming ability, adherence, viscosity, acid-producing ability), human in vivo tests (for example, ingestion time, intraoral clearance time, plaque *p*H response), and experimental caries in animals. But because of the multifactorial nature of caries etiology, the fact that humans eat a mixed diet, and the evidence that the sequence of eating various foods may affect their cariogenic potential, the precise cariogenicity of any one food cannot be predicted with any degree of accuracy. Cumulative evidence nevertheless suggests that both the amount of sugar consumption and the frequency of ingesting between-meal sugary snacks are related to caries in humans as well as in animals.

Studies relating eating habits to caries in humans are invariably hampered by important methodological problems that can affect the results obtained. Accurate data on dietary intake are difficult to obtain. The diets of human subjects can rarely be controlled to the extent necessary, even for a short term. And, to properly assess the effect of dietary factors on caries—a chronic disease in which it usually takes 2 years for a clinically detectable lesion to develop—a strictly controlled diet maintained over a long period would be desirable but is an unlikely prospect. Because the food consumed by individuals normally varies considerably, gross distortions of the magnitudes of computed correlations and estimated regression coefficients are possible in epidemiological studies. The sugar content of the diet itself is difficult to assess accurately because different sources provide different figures for the same foodstuffs. It is often impossible even to compare the results of different studies, since frequently they are reporting on different sugars. Testing potentially cariogenic foods is further complicated by the fact that caries, once cavitation has occurred, is irreversible; ethical considerations therefore rule out human experiments to deliberately test caries-promoting foods and beverages. Finally, many studies have not contained enough subjects for the results of statistical analyses to be meaningful. For these reasons, positive proof of the diet's role in human caries will not easily be established, and we will mostly have to rely on animal studies and in vitro testing.

References and Notes

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