Carbohydrates and dental health\textsuperscript{1,2}

Juan M Navia

ABSTRACT Carbohydrates, together with fats and proteins, provide the necessary energy needs for humans, and constitute, therefore, an integral part of a normal diet. An etiological model for caries that considers local and host factors is presented, highlighting the interaction of bacteria, saliva, minerals, and trace elements with food residues and with other intrinsic environmental and behavioral characteristics of the host. Consideration of this complex multifactorial etiology precludes the identification of any one factor as the only one to be singled out in prevention and management of oral disease. Sugar is, unquestionably, a risk factor for caries, and, therefore, its total substitution with energetic or nonenergetic sweeteners has been found useful. However, partial substitutions of fermentable sugars may have limited practical value in controlling caries prevalence. Caries preventive programs that take into consideration not one, but several etiologic factors, will be effective and beneficial to the general population. \textit{Am J Clin Nutr} 1994;59(suppl): 719S–27S.

KEY WORDS Caries, host factors, sugar, sweeteners, bacteria

Introduction

During the past decade, the science of nutrition has been concerned with maintenance of health and prevention of diseases, particularly chronic diseases (including dental caries), which are responsible for the present high rate of morbidity and mortality seen in developed countries. Equally disturbing is the emergence of many of these chronic diseases in developing countries. This has stimulated research efforts to understand the role of nutrition and diet in reduction of risk for chronic diseases. It has also contributed to the excitement we see today in discussions involving nutritionists, public health professionals, clinicians, food manufacturers, consumer representatives, and policymakers in attempts to determine amount, combination, functional properties (1), and sequence of foods in the diet, as well as the interactions of nutrients that could reduce the risk factors associated with specific disease conditions (2).

Dental caries is a disease caused by the presence and pathogenicity of bacterial plaque. It is characterized by a progressive, intermittent demineralization of enamel, dentin, and/or cementum with a characteristic pattern of decay that may lead to total destruction of coronal dental tissues and the formation of pulpal abscesses. A number of studies in humans (3–6) and animals (7, 8) have identified carbohydrates (including starches), and sugars in particular, as important caries-promoting components of foods. Consequently, during the past 40 y, research and health education programs have focused on decreased sugar in the diet and fluoride delivery (an agent that prevents caries) as the only interventions needed to control and prevent caries. Extensive research has reaffirmed the importance of these two actions on the management of the disease, but it has also contributed additional information and understanding about the role of diet, foods, nutrients, and nutrition in general that needs to be considered when the relation between sugars, carbohydrates, and dental caries is being evaluated.

The relationship of sugar consumption to other chronic diseases has been examined by a number of national committees in the United States (9) and in the United Kingdom (10) and the final conclusion has always been that consumption of sugars is a risk factor for caries.

Sugar consumption and availability and dental caries

Determining the sugar consumption of a population living in a country or region is difficult because of the many factors influencing such data: 1) different methods are used to record total sugar consumption, 2) some data are given as raw sugar and some as refined sugar, and 3) most of the data represent sugar disappearance and not actual consumption. In many regions of the developing world, for example, there is widespread consumption of sugar cane and other raw sugar products that are not included in consumption or disappearing sugar data and yet definitely contribute to dental caries (11). Attempts to evaluate sugar consumption is further complicated by the fact that although some sugar is used directly from the sugar bowl, many foods are processed with varying amounts and types of sugar that are frequently difficult to quantitate and include in food composition tables. Per capita disappearance estimates of sweeteners represent the amount of sweeteners delivered by refiners or importers to food industry, wholesalers, and retailers of such commodities. Such data for sugar consumed in various countries around the world is shown in Table 1. The values for different countries differ in a major way. Countries in Europe (13), North, Central, and South America, and Oceania have the highest mean consumption, and those in Africa and Asia show half as much sugar consumption as the other countries. Data for the United States

\begin{table}[h]
\centering
\begin{tabular}{|c|c|c|c|c|c|}
\hline
Country & Sugar Consumption (g/day) & Source \hline
Europe & High & Ref. \hline
North America & High & Ref. \hline
Central America & High & Ref. \hline
South America & High & Ref. \hline
Oceania & High & Ref. \hline
Africa & Low & Ref. \hline
Asia & Low & Ref. \hline
\hline
\end{tabular}
\caption{Sugar Consumption in Various Countries}
\end{table}

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TABLE 1
Mean sugar consumption per capita (kg, raw value)*

<table>
<thead>
<tr>
<th>Countries (region)</th>
<th>1990</th>
<th>1988</th>
<th>1986</th>
</tr>
</thead>
<tbody>
<tr>
<td>Europe†</td>
<td>41.8</td>
<td>42.7</td>
<td>41.3</td>
</tr>
<tr>
<td>Albania</td>
<td>21.3</td>
<td>22.8</td>
<td>23.0</td>
</tr>
<tr>
<td>Netherlands</td>
<td>58.0</td>
<td>54.0</td>
<td>53.9</td>
</tr>
<tr>
<td>Poland</td>
<td>43.1</td>
<td>48.2</td>
<td>49.0</td>
</tr>
<tr>
<td>Sweden</td>
<td>44.2</td>
<td>45.7</td>
<td>45.8</td>
</tr>
<tr>
<td>Africa†</td>
<td>14.4</td>
<td>14.9</td>
<td>15.4</td>
</tr>
<tr>
<td>Egypt</td>
<td>32.0</td>
<td>32.3</td>
<td>34.5</td>
</tr>
<tr>
<td>Mozambique</td>
<td>2.8</td>
<td>2.8</td>
<td>4.9</td>
</tr>
<tr>
<td>South Africa</td>
<td>42.9</td>
<td>41.6</td>
<td>44.7</td>
</tr>
<tr>
<td>Swaziland</td>
<td>57.5</td>
<td>53.7</td>
<td>45.2</td>
</tr>
<tr>
<td>Oceania†</td>
<td>41.5</td>
<td>41.2</td>
<td>41.9</td>
</tr>
<tr>
<td>Australia</td>
<td>50.0</td>
<td>52.5</td>
<td>50.5</td>
</tr>
<tr>
<td>Papua New Guinea</td>
<td>8.0</td>
<td>8.3</td>
<td>8.0</td>
</tr>
<tr>
<td>North and Central America†</td>
<td>37.7</td>
<td>35.6</td>
<td>35.1</td>
</tr>
<tr>
<td>Canada</td>
<td>38.0</td>
<td>34.9</td>
<td>44.5</td>
</tr>
<tr>
<td>Costa Rica</td>
<td>60.6</td>
<td>58.7</td>
<td>60.5</td>
</tr>
<tr>
<td>Cuba</td>
<td>95.1</td>
<td>83.8</td>
<td>78.3</td>
</tr>
<tr>
<td>Haiti</td>
<td>10.0</td>
<td>11.6</td>
<td>10.3</td>
</tr>
<tr>
<td>United States</td>
<td>31.9</td>
<td>30.3</td>
<td>30.1</td>
</tr>
<tr>
<td>South America†</td>
<td>38.9</td>
<td>40.8</td>
<td>42.2</td>
</tr>
<tr>
<td>Argentina</td>
<td>28.7</td>
<td>29.9</td>
<td>38.1</td>
</tr>
<tr>
<td>Colombia</td>
<td>38.4</td>
<td>37.4</td>
<td>39.0</td>
</tr>
<tr>
<td>Paraguay</td>
<td>24.1</td>
<td>23.8</td>
<td>22.8</td>
</tr>
<tr>
<td>Venezuela</td>
<td>38.0</td>
<td>41.7</td>
<td>43.8</td>
</tr>
<tr>
<td>Asia†</td>
<td>12.2</td>
<td>11.9</td>
<td>11.3</td>
</tr>
<tr>
<td>China</td>
<td>6.5</td>
<td>6.1</td>
<td>7.0</td>
</tr>
<tr>
<td>India</td>
<td>13.4</td>
<td>13.2</td>
<td>11.7</td>
</tr>
<tr>
<td>Japan</td>
<td>22.8</td>
<td>23.1</td>
<td>22.4</td>
</tr>
<tr>
<td>Malaysia</td>
<td>41.1</td>
<td>39.3</td>
<td>37.7</td>
</tr>
</tbody>
</table>

* From reference 12.
† Mean sugar consumption per capita for the region.

can be obtained from sources describing World Consumption Data (Table 2) or from USDA National Consumption Data (15).

During the past 15 y, the availability of total sweeteners (considering the data for per capita disappearance) has been stable at ≈57 kg·person⁻¹·y⁻¹, or per capita ≈156 g/d. The amount increased slightly in 1991 and is now close to 63 kg·capita⁻¹·y⁻¹. One important change is the increased consumption of corn sweeteners during the past two decades (13% of total sweeteners in 1965, and in 1985 it had increased to 47% of total sweeteners consumed). This change was mainly due to the increased use of high-fructose corn syrup (HFCS) in the manufacture of carbonated beverages and other foods that previously were sweetened with sucrose. Data obtained from international agencies such as the Food and Agricultural Organization of the United Nations (FAO) and national food balance sheets do not always provide the same exact picture as national consumption surveys. The USDA nationwide food consumption survey (Table 3) was carried out in 1977–1978 and then again in 1987–1988. The mean daily intake of total carbohydrates in 1977–1978 for all individuals surveyed (36 254) was 193 g, whereas the amount consumed in 1987–1988 increased to 201 g (n = 238 510). Consumption of carbohydrate was highest among children 6–11 y of age (238 g) and seemed to be lower for females than for males (172 vs 238 g in the 1987–1988 survey).

Despite the high variability in the consumption of various carbohydrates, specifically sugars and the different food vehicles in which they are consumed, several attempts have been made to study the relationship between sugar consumption and dental caries (17). The study of this relationship is complicated by the following limitations among others:

1) Sugars are consumed in different physical forms, with different foods and different dietary patterns that affect oral clearance rate of these dietary sugars.

2) The data, whether obtained from food balance sheets or from dietary surveys, represents an approximation of the actual sugar consumption.

3) The data represents information relating to consumption during the year, whereas the carious lesions take several years to develop in either the primary or secondary dentition.

4) Caries is influenced by many other etiologic factors including fluorides, health services, education, oral hygiene practices, and other habits and circumstances.

Regardless of these limitations, Sreebny (17) was able to illustrate the fact that in nations with high availability of sugar,

TABLE 2
US per capita consumption of sweeteners (kg, dry basis)*

<table>
<thead>
<tr>
<th>Selected years</th>
<th>Refined sugar†</th>
<th>HFCS‡</th>
<th>Total sweeteners</th>
<th>US population§</th>
</tr>
</thead>
<tbody>
<tr>
<td>1980</td>
<td>38.0</td>
<td>8.4</td>
<td>17.8</td>
<td>56.3</td>
</tr>
<tr>
<td>1985</td>
<td>28.5</td>
<td>20.4</td>
<td>30.6</td>
<td>59.7</td>
</tr>
<tr>
<td>1987</td>
<td>28.4</td>
<td>21.4</td>
<td>31.8</td>
<td>60.8</td>
</tr>
<tr>
<td>1989</td>
<td>28.6</td>
<td>22.1</td>
<td>32.8</td>
<td>62.0</td>
</tr>
<tr>
<td>1991</td>
<td>29.3</td>
<td>22.6</td>
<td>33.6</td>
<td>63.6</td>
</tr>
</tbody>
</table>

* From reference 14.
† Sugar consumption is the total US sugar deliveries for food and beverage use.
‡ HFCS, high fructose corn syrup.
§ Total population, including Armed Forces, overseas, July 1 of year (in millions).

TABLE 3
Carbohydrate intake over 3 d*†

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>n‡</th>
<th>Intake§</th>
<th>n‡</th>
<th>Intake§</th>
</tr>
</thead>
<tbody>
<tr>
<td>3–5</td>
<td>M/F</td>
<td>1719</td>
<td>174 ± 4.4</td>
<td>10 972</td>
<td>170 ± 2.7</td>
</tr>
<tr>
<td>6–11</td>
<td>M</td>
<td>1851</td>
<td>225 ± 5.4</td>
<td>10 515</td>
<td>238 ± 4.0</td>
</tr>
<tr>
<td>6–11</td>
<td>F</td>
<td>1939</td>
<td>209 ± 4.9</td>
<td>10 677</td>
<td></td>
</tr>
<tr>
<td>30–39</td>
<td>M</td>
<td>1906</td>
<td>229 ± 7.0</td>
<td>19 128</td>
<td>238 ± 3.6</td>
</tr>
<tr>
<td>30–39</td>
<td>F</td>
<td>2849</td>
<td>157 ± 4.1</td>
<td>20 570</td>
<td>172 ± 2.7</td>
</tr>
<tr>
<td>All</td>
<td>M/F</td>
<td>36 254</td>
<td>193 ± 1.3</td>
<td>238 510</td>
<td>201 ± 0.9</td>
</tr>
</tbody>
</table>

* From reference 16.
† USDA Nationwide Food Consumption Surveys.
‡ n, number of US individuals surveyed.
§ f ± SE.
ranging from 57 to 115 g·person⁻¹·d⁻¹, 12-y-old children had decayed, missing, and filled teeth (DMFT) values ranging from 3.2 to 4.8, compared with DMFT values of 0.1 to 2.7 in DMFT age-matched children in nations with people having average sugar intakes < 50 g·person⁻¹·d⁻¹. There were, however, some apparent discrepancies. In nations such as Samoa and Swaziland, where people have sugar availabilities of 78 and 99 g·person⁻¹·d⁻¹, respectively, 12-y-old children had a DMFT value of 1.1. When Sreebny calculated the correlation coefficient for data from 47 nations, he found an \( r = 0.72 \), which suggested that \( \approx 50\% \) of the variability in caries could be explained by sugar availability, but there were still many other factors that accounted for the remaining sources of variability. The differences between disappearance data and actual consumption data may explain some of these departures from the trend.

In a very comprehensive review (18), Sreebny evaluated the voluminous literature addressing the relationship between sugar consumption and dental caries. From this careful review emerges the conclusion that communities or groups of people who have very low intakes of sugar show very low DMFT scores, and those who have very high availability of sugar in their diets seem to have severe caries. He also suggested that the sugar exposure-response curve to dental caries could have a sigmoid relationship. However, the major problems yet to be defined are: the lowest sugar intake that induces minimum caries; the infection point where caries response begins to increase; and the dose-exposure level at which no further increases in caries are obtained with increased sugar intake. Sreebny’s data suggest that an average “availability” of 50 g sugar·d⁻¹·person⁻¹ in a community (corresponding to < 10% of energy provided by sugar) may represent an outer limit of safe or acceptable sugar consumption. This could correspond approximately to an intake of 18 g·capita⁻¹·y⁻¹ and is considerably lower than the intake of all countries in Europe (13) and most countries in America and Oceania, and would only be met by a few countries in Africa and Asia.

Missing from evaluation of the relationship between sugars available to individuals in different international communities and their experience of caries are considerations relating to the types of sugar-containing foods eaten, whether these are consumed with or between meals, as well as other factors affecting the oral biology of the consumers. A study of 275 Icelandic children aged 12 or 13 y, who were followed for 1 y, indicated that sweet buns were consumed by 32% of children with meals and by 58% of them between meals, whereas soft drinks were consumed by 42% and 46%, respectively. Eighty-three percent of children reported eating confectionery products daily. Interestingly, this study (19) reported a higher caries incidence for those children with frequent intake of buns and cakes with meals, and also with high confectionery intake.

The relationship between sugar intake and caries was initially established with the Vipiholm study (3) and reaffirmed with the extensive review of Sreebny (17, 18) describing the many investigations that support this relationship. There have been some studies done in South Africa (20, 21), Canada (22) and the United States (23) in which this relationship has not been clearly established. There is a need therefore, to examine in greater detail the overall conditions determining caries, to ascertain the main factors and interactions that need to be considered in designing and implementing health policy, or in developing health behavior, educational, or preventive programs where recommendations relative to sugar utilization are made.

### Etiology of dental caries

Figure 1 presents a model of our current understanding of the multifactorial etiology of dental caries. In the center is the local, oral factors headed by the bacteria in plaque. These bacteria include not only those shown to be highly cariogenic, such as the mutans streptococci and the Lactobacilli, but also others that are less cariogenic such as the Actinomycetes, and still others like Veillonella, which utilizes lactic acid as a source of energy and thus interferes with acid accumulation and pH decreases in plaque, a phenomenon that affects the caries process. There are three aspects important relative to the cariogenic action of bacteria in plaque: 1) the implantation mechanism involving adhesion, 2) the colonization through competition with other plaque bacteria components, and 3) the metabolic activity of established bacteria under the influence of food residues and oral environment.

The second factor is saliva, the most important protective biological fluid in the oral cavity (24). Foods stimulate production of saliva through olfactory, masticatory, and gustatory actions and the amount, flow, and composition of saliva can be influenced by these physiologic stimuli. Salivary gland secretory function can be influenced also by nutritional insults early in development (25), and also later through chronic effects of malnutrition (26). Although aging per se does not seem to have an overt effect on salivary gland function (27, 28), diseases and malnutrition frequently associated with aging, compromise the ability of the gland to protect oral tissues. In addition, drugs and medications commonly used by older individuals can also induce mild or even severe xerostomia and other pathologic side effects (29). There is a potential contribution of drug-induced xerostomia to the increased prevalence of malnutrition in elderly individuals (30).

The next group of factors includes minerals and trace elements, particularly fluoride in the oral environment. Calcium and phosphorus in plaque and saliva are major contributors to the remineralization of enamel surfaces affected by acid. The concentration of those mineral ions can be further increased by drinking water or eating food rich in calcium and phosphorus. The beneficial
effect of certain cheeses (31, 32) can be traced largely to their high mineral content which, together with protein, will neutralize the acidogenic effects of sugars. The presence of low concentrations of fluoride ions in plaque, saliva, and enamel originally provided by the water, food, or other sources, contributes also to the natural repair process that maintains the integrity of enamel or exposed cementum surfaces.

The final factor in this group of local, oral etiologic components are nutrients and food residues left in the oral cavity after masticating and swallowing the food bolus or drinking beverages. These residues are retained on the dorsum of the tongue and in other retentive sites in the oral cavity, and from these locations continue to yield soluble and nutritious substances that can be utilized by plaque bacteria as specific substrates to enhance any of the three activities previously mentioned: implantation, colonization, and metabolic activity of the plaque bacteria. Sugars and starches (8, 33) are part of these residues and exert their effects on plaque depending on the type of carbohydrate (sucrose, glucose, sorbitol, etc); the concentration; the retention time in the oral cavity or the frequency with which the fermentable carbohydrates are replenished by repeated intake; the action of saliva in diluting this substrate or converting starch into fermentable glucose and maltose through α-amylase or providing the necessary buffering capacity to neutralize acid end products that result from such fermentations. These food residues therefore, have caries-promoting properties (rather than cariogenicity, because foods do not directly determine caries) that influence the caries process.

All of these local factors can exert their particular positive or negative influence on caries development and they do so in an episodic and intermittent manner. This periodicity of activity is related to food intake and the circadian rhythm that has such an important effect on salivary gland function. Local effects are important, but the etiology of caries is also influenced by a number of host factors (depicted in the external circle of Fig 1) that are characteristic for each individual. The nutritional status, both during development (34) and later in life (26), the age of the individual, which determines whether the primary or secondary dentition or newly exposed cementum or dentin is at caries risk, the hereditary and racial characteristics of the person, and finally, the individual’s lifestyle, education, and socioeconomic status will also contribute to or interfere with caries development.

It is not difficult to understand, considering the complexity of the etiologic model of dental caries, why one single factor, such as the amount of sugar available for consumption in a country cannot be readily extrapolated with a very high degree of confidence to explain the disease severity at all levels of sugar intake. The complete absence of one of these etiologic factors will elicit a major change in the caries process. For example, lack of dental plaque or absence of fermentable carbohydrates will be associated with low caries whereas compromised salivary function or absence of fluoride will enhance the carious process. Extreme situations will produce clear results, but the majority of people in communities around the world find themselves in intermediate ranges for these different etiologic factors, and in such circumstances it is the interaction between two or more factors rather than a single main factor that determines the disease outcome.

Recently, interesting research efforts have dealt with the etiologic relation between sugar and caries, particularly the study of the effects of different amounts of sugar intake on the oral implantation and distribution of mutants streptococci. The theory behind those studies is that sugars, particularly sucrose, not only stimulate the metabolic activity of these bacteria, but also their ability to implant and colonize on tooth surfaces. Sepa et al (35), using the dip-slide method, studied the relationships among concentrations of Streptococcus mutans, sucrose intake, and dental caries in 841 13-y-old children. High S mutans counts were associated with a significant increase in caries scores and a highly significant (P < 0.001) decreased salivary flow rate. In general, there was no significant correlation between the reported intake of sucrose-containing foods and S mutans; however, children with the very highest S mutans counts were also the ones that had the highest sucrose intake. The relationship between sugar consumption and distribution of mutants streptococci in different population groups around the world has also been studied (36). Table 4 presents data for a group of 168 Polish children who were studied to determine their frequency of consumption of sugar-containing foods. In this population with high sugar intake, a considerable number showed high counts of bacteria, but a moderate percentage had low counts. The overall caries experience for this population was a mean DMFT of 4.7 at 10–12 y of age. In this same study, two developing countries, Mozambique and Sudan, with low average sugar consumptions of 11 and 18 kg per capita, respectively, found 10–12-y-old children with high numbers of mutans streptococci and an average DMFT that ranged between 0.5 and 2.0 with the majority of younger children with DMFT values well under 0.5. In Mozambique (Table 4), children with caries also had high counts of mutans streptococci, however 26% of children that were caries free also had high salivary counts of cariogenic bacteria. Carlsson (36) concluded that, “The differences in diet between the populations or groups were not sufficient to affect the counts of lactobacilli or mutans streptococci, but have been sufficient to create the differences in caries prevalence.” Interpretation of these data is made difficult by the fact that mutants streptococci includes a number of strains with different serotypes, biochemical properties, and degree of cariogenicity in animal models. Loesche (38) pointed out the differences between S mutans, S sobrinus, S rattus, and S rattus, and indicated that some, like S rattus, found frequently in Tanzanian children, are specific in their ability to hydrolyze arginine (an amino acid frequently found in substantial concentrations in malnourished children), and is able to colonize teeth when sucrose and/or glucosyltransferase are present in the oral cavity. S mutans, however, is not so dependent on sucrose, because attachment to the acquired enamel pellicle is mediated by an adhesin that interacts with salivary components. Differences in strains, ability to produce acids or bases, and attachment mech-

TABLE 4
The level of mutants streptococci in some selected populations (percent)*

<table>
<thead>
<tr>
<th>Population</th>
<th>Colony-forming units</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 1–10 11–50 51–100 ≥ 100</td>
</tr>
<tr>
<td>Mozambique, 8 villages</td>
<td>2 13 27 18 40</td>
</tr>
<tr>
<td>Sudan, Khartoum, 3 schools</td>
<td>3 13 33 18 53</td>
</tr>
<tr>
<td>Thailand, Bangkok</td>
<td>2 18 40 19 21</td>
</tr>
<tr>
<td>Thailand, Petchaboon</td>
<td>18 37 21 10 15</td>
</tr>
<tr>
<td>Poland, Warsaw</td>
<td>6 25 37 17 15</td>
</tr>
</tbody>
</table>

* From reference 37.
anisms complicate interpretation of relationships among sugar intake, mutans streptococci counts, and caries severity. It can be concluded from these results and the previous discussion on the etiology of caries that: 1) specific bacteria are the principal etiologic agents for dental caries, 2) a very low concentration of sucrose is sufficient to allow implantation and colonization of mutans streptococci in the oral cavity (7), 3) implantation of the various strains within mutans streptococci and degree of virulence is dependent on the interaction between specific biochemical properties of the strain and substrates present in the oral environment, 4) increases beyond this low intake of sugar-containing foods will stimulate the metabolic activity of the established cariogenic plaque flora, and therefore, determine proportionate increases in caries experience, and 3) these increases will be modulated by many other etiologic factors such as type of food providing the sugar, frequency of consumption, oral hygiene status, availability of fluoride, salivary function, and other host factors. Considering the ubiquity of the cariogenic bacteria, consumption of sugar-containing foods constitutes an increased risk to oral health. However, the magnitude and severity of this challenge vary with individuals and population groups. Prevention programs to control and eradicate dental caries should not be limited to consideration of sugar intake alone, but must also be extended to include other etiologic factors that also influence and moderate development of the disease.

Energetic and nonenergetic sweeteners

Sucrose has a special caries-promoting role because it is a specific substrate for glucosyl transferase, the enzyme in mutans streptococci responsible for the synthesis of the extracellular polysaccharide glucan. However, the capacity of sucrose to stimulate metabolic activity of bacteria in plaque is no different from that of glucose and fructose (33). Other sugar alcohols used as bulk sweeteners (39) such as sorbitol, mannitol, maltitol, lactitol, Palatinit (an equimolar mixture of two disaccharide polyols; Zuzducker Ab, Germany), Lycasin (a starch hydrolysate, 53.8% maltitol; Roquette Frères, Lille, France), Malbit (79.9–88.4% maltitol; Nikken Chemical Company, Limited, Japan), and xylitol (a five-carbon sugar alcohol) have energy value, but have also been shown to have either low or no caries-promoting properties. Xylitol may even have anticariogenic properties (40).

Other synthetic sweeteners such as saccharin, cyclamates, aspartame (a methylated dipeptide), as well as others being developed now such as sucralose (a chloro-substituted sucrose) (41), have high sweetness properties that vary in intensity and quality, but yield no energy value. The very small concentrations of these synthetic sweeteners in manufactured foods and beverages, and their chemical inability to produce acids through the glycolytic pathway make them unable to stimulate caries. Although the caries impact of the synthetic sweeteners is difficult to prove epidemiologically, the high consumption of these sweeteners in a multitude of food products and prescribed drugs may have contributed to a reduced caries risk in populations with access to these foods.

During the past 30 y, nearly one dozen human clinical trials and numerous animal studies have been done to evaluate the caries-promoting properties of sugar alcohols (6, 40). Many of these studies have been designed or endorsed by the World Health Organization (WHO), such as the ones conducted in Thailand, two in French Polynesia, and one in Hungary (6). These studies usually included experimental designs characterized by low intake of the polyols being tested and use of mixtures of sorbitol and xylitol, varying baseline caries prevalence between groups, and a duration of 2–3 y. Caries increments, expressed as DMF surface counts in relation to the number of teeth at risk, are listed for four studies in Table 5. Inspection of these results indicates that regardless of the initial baseline scores in the different groups, problems with controls and other methodological limitations, the substitution of sucrose by sorbitol and/or xylitol led to a decreased caries challenge. Mäkkinen and Iso-kangas (40), in a comprehensive review of the literature on the relationship between carbohydrate sweeteners and plaque-dependent diseases, recognized that because caries has a multifactorial etiology, a single intervention would not lead to its eradication. However, total substitution of the sucrose content of a food by sugar alcohols would decrease the food's caries-inducing potential, and therefore, is a viable preventive measure. Total substitution of sucrose in a human diet by selected sugar alcohols, such as xylitol, to evaluate its effect on caries has been done in Turku, Finland. In this study, manufactured foods containing either sucrose, fructose, or xylitol were given to individuals assigned to three groups. It was found that the sucrose group had the highest caries activity, the fructose group had slightly less, and the xylitol group had considerably less caries than the other two sugar groups (43). In another study, subjects consumed their usual diet, however, one group chewed gum containing sucrose and the other group was given gum containing xylitol (44). Results also indicated that subjects using the xylitol-containing gum had fewer caries. During these studies, it was observed that not only did the xylitol group have less caries, but there was a qualitative impression that the presence of xylitol could contribute to the mineralization of incipient caries. Rekola (45), using a pla-

### Table 5

Field studies of sugar substitutes*

<table>
<thead>
<tr>
<th>Study</th>
<th>Age, begin-end</th>
<th>Regimen</th>
<th>Absolute rate</th>
<th>Relative rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thailand</td>
<td>7–10</td>
<td>Xylitol/sorbitol/fluoride</td>
<td>1.73</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fluoride rinsing</td>
<td>1.70</td>
<td>98</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sugar/fluoride</td>
<td>4.72</td>
<td>273</td>
</tr>
<tr>
<td>Thailand</td>
<td>10–13</td>
<td>Xylitol/sorbitol/fluoride</td>
<td>2.84</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fluoride rinsing</td>
<td>2.25</td>
<td>79</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sugar/fluoride</td>
<td>3.70</td>
<td>130</td>
</tr>
<tr>
<td>Polynesia</td>
<td>10–13</td>
<td>Xylitol/sorbitol/fluoride</td>
<td>6.18</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fluoride rinsing</td>
<td>13.44</td>
<td>218</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sugar/fluoride</td>
<td>9.39</td>
<td>152</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Xylitol/fluoride</td>
<td>9.77</td>
<td>158</td>
</tr>
<tr>
<td>Hungary</td>
<td>10–13</td>
<td>Xylitol/fluoride</td>
<td>6.60</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fluoride in milk or water</td>
<td>14.73</td>
<td>223</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Control</td>
<td>13.93</td>
<td>211</td>
</tr>
</tbody>
</table>

* Modified from reference 42. Comparison of four field trials. Caries increments are expressed as decayed, missing, and filled (DMF) surface counts in relation to the numbers of teeth at risk. Rates are given as annual absolute values and as relative values in percentage terms after assigning the value 100 to groups with combined xylitol and fluoride regimens.
nimetric evaluation procedure, examined projections of approx-
imal lesions on bite-wing radiographs of the subjects in the 2-y
Turku study. Results indicated a highly significant increase of
approximal lesion size in the sucrose group, whereas in the xy-
litol group lesion sizes remained unchanged. The conclusion of
this study is that total substitution of sucrose in foods with xylitol
would reduce the progress of carious lesions and favor the arrest
and decrease of the lesions. However, because total substitution
of sucrose by xylitol is not feasible in nonexperimental subjects,
the value of xylitol as an anticariogenic sugar remains question-
able. In reviewing the very extensive literature (46) dealing with
fermentable sweeteners, the best candidates as substitutes for su-
crose for the purpose of caries control are sorbitol, xylitol, malt-
titol, and starch hydrolizates containing different proportions of
polysols.

The conclusion of a number of reviews and evaluations (47–
50) is that consumption of sorbitol as a food ingredient, aside
from its possible laxative effect due to slow intestinal absorption,
poses no special health risk for the consumer. Individuals who
consume large doses (> 50 g at one dose) of sorbitol may de-
velop these intestinal symptoms, but no other negative symptoms
have been detected. Similar precautions should be considered in
the use of xylitol as a sweetener in foods (40). However, care
should be exercised in promoting the use of these sugar alcohols
for individuals in less developed communities where diarrhea is
already a chronic problem and ingestion of these sugars may
further complicate this debilitating condition.

The effectiveness of unfermentable carbohydrates such as sor-
bitol and xylitol in reducing the caries risk of foods is predicated
on the following properties of these sugar alcohols:

1) Fermentation of sorbitol by plaque bacteria is slow and
therefore, reductions in plaque pH are minimal (51). In the case
of xylitol, mutans streptococci are unable to metabolize it, and
probably no other bacterial component of human dental plaque
can ferment it either.

2) The metabolism of sorbitol by bacterial cells requires the
presence of sorbitol dehydrogenases which are not constitutive
enzymes, and therefore, the induction of these enzymes requires
the presence of sorbitol in order to carry out the necessary en-
zyme synthesis, a process that is inhibited by glucose.

3) None of the sugar alcohols are specific substrates of glu-
cosyl transferase, so extracellular polysaccharides, which are es-
cential in the attachment process of several streptococci, cannot
be synthesized. Only sucrose can be utilized by the glucosyl and
fructosyl transferases to synthesize either glucans or fructans,
and therefore sucrose is specially effective in promoting the im-
plantation and colonization of those mutans streptococci that depend
on this mechanism to adhere to enamel or acquired pellicle.

4) The fermentation of sorbitol differs from that of glucose in
that instead of yielding large quantities of lactate as the major
end product plus small quantities of formate and acetate under
anaerobic conditions, it produces major amounts of formate and
ethanol and very small quantities of lactate. This could affect the
capacity to demineralize enamel depending on the amount and
type of acids produced (52).

The previously mentioned characteristics of sorbitol and xy-
litol make sorbitol a very low caries-promoting sugar alcohol,
and xylitol either a non- or even anticiogenic ingredient in foods
(40). However, frequent consumption of sorbitol is not without
a potential caries risk because of the possibility of adaptive changes
that would enable a more effective utilization of this
sugar by plaque bacteria. Several human and some animal studies
have presented evidence indicating that such adaptive transforma-
dation does not take place, but others have reported ecologic
changes that seem to suggest increased caries potential when an
abnormally high amount of sorbitol is consumed (46). Studies
using laboratory rats with wild cariogenic oral flora or inoculated
with S mutans seem to have provided evidence suggesting variant
caries behavior with sorbitol added to the diet. Firestone and
Navia (53), using a rat plaque model, found a significant drop in
plaque pH after the application to rat dental plaque of glucose
and sucrose in comparison with sorbitol and xylitol. Plaque pH
decrease was maximal after a 10% sucrose solution was applied,
but did not decrease further with application of a 20% sucrose
solution. In a follow-up study, these investigators (54) tested
whether adaptation to sorbitol fermentation could be observed in
rat plaque. The study involved making pH measurements of sul-
cal plaque after topical applications of 10% sorbitol to rats that
were fed for 16 d a caries-promoting diet containing 62% starch
and 5% sucrose and inoculated with S mutans 6715. One group
of rats was then fed for a 13-d period a diet containing 20% sucrose
and a second group a diet with 20% sorbitol. A significa-
cantly greater (P < 0.01) pH drop was observed after oral ap-
lication of a 10% sorbitol solution to the rats in the group being
fed a sorbitol-containing diet than in those rats in the sucrose
group. There was no difference in response between the two diet
groups when a solution of 10% sucrose was applied to sulcal
surfaces and the pH was measured. Clearly, in rats, adaptation to
sorbitol intake can take place and can be measured by an in-
duced drop in sulcal pH due to either selection of sorbitol fer-
menting bacteria or induction of sorbitol-metabolizing enzymes.

Maltitol is a disaccharide polyol made of one molecule of glu-
cose and one of sorbitol, and found in large quantities in hydro-
genated starch products such as Malbit (80–90% maltitol) and
Lycasin (∼54% maltitol), which also contain sorbitol and other
polysols. Recently, it has been crystalized and its caries-promot-
ing properties have been evaluated using animal models (55).
Acid production from maltitol and also sucrose was tested by
using 14 different varieties of oral streptococci, and none of them
promoted caries under these conditions or were found capable of
fermenting maltitol to produce acid. The authors suggested that
maltitol, which has a sweetness equivalency of 90% compared
with sucrose, is an appropriate sucrose substitute because it is
not fermentable by cariogenic oral bacteria, and may even inhibit
the synthesis of insoluble glucan from sucrose by glucosyl trans-
ferases of the mutants streptococci. Further research is needed to
validate these promising results in human populations.

Review of the extensive literature dealing with sucrose sub-
stitutes such as the polyols indicates that use of these bulk sweet-
eners under controlled intake that does not exceed 40–50 g per
dose poses no threat to general health and could help maintain
oral health with its low caries-promoting properties (56). Like
any other food ingredient, excessive use and abuse of these prod-
ucts may lead to undesirable side effects, and could even negate
their beneficial properties. Saccharin and aspartame are the only
non-bulk sweeteners approved in the United States at this time,
and neither has caries-promoting properties; when consumed in
small amounts they do not pose serious risk to human health.
Several reports have been published indicating an inhibitory ef-
fect of saccharin on growth and acid production of oral strepto-
cocci (57–60) and on animal caries. These reports on saccharin
and its effects on bacteria still need to be reevaluated in human
subjects, but if shown to have the same effects in humans as in laboratory animals, the increased consumption of synthetic sweeteners seen in the past 20 y would need to be considered as one reason for the decrease in caries seen in many developed countries.

**Dietary sugars and caries: field studies**

Although the direct relationship between sugars and dental caries is accepted by all clinicians and researchers working in the dental field, the degree of emphasis on the importance of this factor in prevention and control of the disease varies. The information we have available today should allow for a more scientific and rational approach to the role of fermentable carbohydrates in dental caries. Rather than focusing solely on the elimination of sugar from the diet, the currently emerging view underscores the need to adopt a more comprehensive preventive approach that recognizes the importance of improved dietary habits, better oral health, appropriate use of fluoride and sealants, enhancement of salivary function, and other considerations in an integrated and complete oral health program throughout the life of the individual.

Walker and Cleaton-Jones (21), without rejecting the well known effect of sugar on caries, identified a number of situations in caries epidemiology reported in the literature where sugar intake alone does not explain the caries status of the consumers. Rugg-Gunn and Murray (4) also reviewed the published evidence and pointed out the special importance of sugar in caries etiology. They pointed out that the amount, frequency, and type of sugar had to be considered, although it was expected that the frequency of sugar intake and the total quantity consumed were probably closely correlated. They also noted the marked variability in caries susceptibility of individuals consuming sugar and sugar-containing foods, an observation consistent with earlier investigations such as the Vipeholm study (3).

Complete replacement of sucrose with non-caries-promoting sugars like xylitol (44), or reductions in availability and intake of sugar as was seen in Europe during World War II (61), are paralleled by proportional caries decreases in individuals involved in such dietary changes, but these effects are not permanent and disappear if the strict dietary changes are interrupted or discontinued. Caries experience of individuals who experienced a sugar intake reduction during the war years showed that there was no long-lasting beneficial effects when they were exposed to a caries-challenging situation during the post-war period (61). Emphasis on dietary habits and appropriate consumption patterns of caries-promoting foods could be important in implementing long-lasting, optimal dietary habits, which, when coupled with other measures, assure good oral health throughout life. It is important to begin to separate the role of fermentable sugars in terms of the implantation and colonization of an abundant cariogenic flora vs the stimulation of the metabolic activity of the established flora. It may be that these two different ecological effects of dietary sugars and sugar alcohols on oral bacteria and dental caries are suggestive of the need for different recommendations and interventions. Further research based on these concepts using human populations may provide valuable insights for new dietary recommendations to prevent and control caries.

There have been many attempts to study in humans the specific relationship between sugar intake, dietary and nutritional variables, and dental caries, but three studies conducted in English (62), American (63), and Canadian (64) children deserve special consideration. The English study involved 405 children who had an initial average age of 11.6 y and they were followed for 2 y. Statistical evaluation of the data indicated that daily consumption of 118 g sugar/d (43 kg/y), which also accounted for 21% of the total energy intake had the highest significant correlation (+0.143) with caries increments. The gingival index increased the multiple R to +0.193, but other variables (sex, social class, and toothbrushing frequency) did not. Therefore, total sugar intake explained only 4% of the variance in caries increment and 94% of the variance remained unexplained by the factors under consideration in the study. The authors believed that a large proportion of the variance was due to methodological errors, such as those related to dietary assessment, and a large intrasubject variability that could mask the true relationship. Although this may be true, other etiologic factors such as saliva, plaque, fluoride, and oral hygiene practices should not be dismissed lightly. In that study, frequency of consumption of sugar, although significant, had a much lower correlation value (+0.099) with caries than the weight of sugary foods consumed (+0.143). The study also provided some interesting conclusions: 1) the importance of avoiding sugary foods shortly before bedtime (−0.101 correlation for time between last food > 10% sugar and bedtime), 2) milk intake was positively correlated with caries increment (+0.10), and 3) a negative correlation was seen between caries increment and vitamin D intake in boys only (−0.238, P = 0.004).

The study done in the United States (63) was a longer, 3-y investigation of 499 children aged 11–15 y in a nonfluoridated community in Michigan. These American children consumed, on the average, 142 g sugar/d (51 kg/y) and sugars accounted for 26.5% of their total daily energy intake. In this study, pit and fissure caries were not found to be correlated with any aspect of sugar consumption, but increments in approximal caries were observed to be related to dietary sugar intake variables. Although children who consumed a high proportion of their energy intake as sugar had the higher increments of approximal caries, there was no relationship between the caries scores and the average frequency of eating during the day or the average number of sugary snacks consumed between meals. Results of this study did not differ much from those obtained in the study done in England and suggested that although sugar continues to be a clear etiologic component of the carious process and a high frequency of sugar consumption increases the risk of the disease, the dietary behavior of the populations in these two sites was not sufficiently caries-promoting to make a major difference in their caries experience.

A third study was done in Canada (64) with 232 11-y-old students to evaluate the association between dietary patterns and dental caries. Nutritional data was collected by using a quality index based on the eating frequency recommended in food guides and divided into eight levels (1 being the worst nutrition and 8 being the rank given to the group that best complied with nutritional recommendations), as well as the frequency of sugary food consumption at meal time and between meals. Results of the study indicated that the nutritional status of these children was compromised; almost 50% of the children had a nutritional quality value of 3 and only 8.2% of the subjects had a quality value of 5, which is the minimal recommendation of Canada’s Food Guide for the use of the four food groups (65). Although there
was a trend toward a decreased caries increment with improved nutritional quality of the diet, analysis of variance did not show statistical significance for this trend. Furthermore, no significant association was observed between frequency of consumption of sugary foods and increments in caries.

It is obvious that dietary sugars are unquestionably an important determinant in the development of caries, but they are not the sole etiologic factor responsible for the disease. This understanding is highlighted in studies done with special populations such as mentally retarded children (66) in which again, the frequent consumption of candy did not seem to be a significant determinant of caries, but rather oral hygiene status appeared to be a more important caries risk factor in this group. Similar conclusions were reached in a study (67) evaluating oral health of Latin American preschool children living in Malmo; children with a gingival score of 01 had only 1.1 decayed, missing, and filled surfaces (DMFS), whereas those with gingivitis with and without bleeding upon probing had 6.2 DMFS. Another study (68) examining the oral health status of Greek immigrant (GI) children in comparison with Swedish (S) and rural Greek (G) children, illustrated the complexity inherent in the evaluation of determinants of oral health. The carbohydrate content of the diet consumed by these three groups was approximately the same, however, only 15% of children in the G group had caries-free primary dentition. They also presented a higher incidence of decayed and filled tooth surfaces in primary and permanent teeth than the other two groups. There was also a similar distribution of mutans streptococci and lactobacilli in the three groups, and this indicates a potential risk for any of the children if other caries conducive conditions are increased. The availability of health services for children living in Sweden (S and GI) was made evident by the increased use of toothbrushes and improved oral hygiene of these groups compared with children in the G group and this could have been an important determinant of their oral health status.

Conclusion

It is increasingly evident that oral health preventive programs have to transcend a sole focus on the relationship between sugar intake and dental caries and begin to pay increased attention to bacterial components of plaque (particularly the cariogenic bacteria), quality and quantity of food residues that will either decrease or increase the numbers or the metabolic activity of oral bacteria, the amount and composition of saliva in the oral cavity, oral hygiene procedures including fluoride status, type of preventive and restorative care, immunologic response of the individual, type of dentition (primary or permanent) to be considered, as well as nutritional and dietary practices of the subjects to be protected against caries through preventive procedures. There are many approaches to implement adequate maintenance of oral health that have been validated by sound research in the past 20 y. Proper selection of a nutritionally balanced diet that does not provide an overwhelming challenge to oral health is one of them, but a more rational and judicious use of all the knowledge and information available now will provide an effective and acceptable approach to the eradication of dental caries in populations from both developed and developing countries around the world.

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References

CARBOHYDRATES AND DENTAL HEALTH


