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Sugars – The Arch Criminal?

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Abstract
Numerous lines of evidence have conclusively established the role of sugars in caries etiology and the importance of sugars as the principal dietary substrate that drives the caries process has not been scientifically challenged. While sugars appear to differ little in acidogenic potential, sucrose has been given special importance, as the sole substrate for synthesis of extracellular glucans. Water-insoluble glucans might enhance accumulation of mutans streptococci on smooth tooth surfaces and appear to enhance virulence by increasing plaque porosity, resulting in greater acid production immediately adjacent to the tooth surface. Data indicating that the sugar consumption/caries relationship is now weaker have led to suggestions that recommendations to restrict sugar consumption are no longer necessary. Clearly, fluoride has raised the threshold of sugar intake at which caries will progress to cavitation, but fluoride has its limits, and caries remains a serious problem for disadvantaged individuals in many industrialized countries and is a rising problem in many developing countries. A weakening of the sugar/caries relationship may also be explained by many technical, biological, behavioral and genetic factors. Future research should aim to determine the biologic and behavioral factors that influence caries risk. Measures to educate the public on the dangers of frequent sugar consumption, combined with recommendations for proper oral hygiene and fluoride use, are still warranted. Individual dietary counseling is highly recommended for patients at high caries risk. As dental caries is a preventable disease, each country must decide: what level of disease is society willing and able to tolerate?

Existing Evidence

The role of the consumption of sugars in dental caries is a very broad topic, which is somewhat controversial and eminently important. The title of this article has been carefully crafted by the Symposium Planning Committee and refers to 'sugars' and not sugar, which is often used synonymously for sucrose. The term sugars includes all the monosaccharides and disaccharides, the most common of which are glucose, fructose, sucrose, maltose and lactose (table 1) [Moynihan, 1998]. Sucrose, which has been considered to play a special role in dental caries [Newbrun, 1969], will be discussed separately later in the paper.

The understanding that sugars are an important etiologic factor in dental caries has been with us since the dawn of civilized man, but the controversy surrounding this subject is a more recent phenomenon. Consistent with comments made by Prof. Fejerskov [2004, this issue], we must keep in mind that science is not conducted

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Table 1. Carbohydrate nomenclature

Monosaccharides
Glucose (dextrose)
Fructose (fruit sugar)
Galactose
Invert sugar (1:1 glucose and fructose)
Disaccharides
Sucrose (table sugar)
Maltose
Lactose (milk sugar)
Trehalose (mushroom sugar)
Natural and manufactured oligosaccharides (3–10 units)
Polysaccharides (>10 units)
Starch

Adapted from Moynihan [1998].

in a vacuum, and many social, political and economic forces come into play, both in the execution of research as well as in its interpretation. Table 2 provides a partial list of review articles that have looked at the role of sugars in dental caries, with most authors supporting the relationship up until fairly recently. This subject has been the focus of many recent review articles and considerable debate, with some authors reaching different conclusions from basically the same studies.

There is an extensive literature on this topic. A PubMed search conducted in April 2003, covering papers back to 1965, identified 2,784 articles using the terms 'dental caries' AND 'sugar'. If only articles written in English are included, the number is lowered to 2,027. This short review cannot be all-inclusive and will mainly focus on the clinical data, while recognizing that other model systems, especially animal models, have added considerably to our understanding on this subject.

Classic Evidence

The classic evidence supporting the role of sugar in dental caries in man is summarized in table 3, and includes studies that are readily recognizable by name – The Vipeholm Study, Turku Sugar Study, World War II Food Rationing, Hopewood House Study, Tristan da Cunha, Hereditary Fructose Intolerance, Experimental Caries in Man, and Stephan Plaque pH Response. No doubt many other studies could be included on this list. This literature collectively still forms in many respects the basis of our understanding of the etiology of dental caries. In particular, the Vipeholm Study has been praised, criticized and even condemned. In the author's opinion, the Vipeholm Study remains one of the most important contributions in the entire dental literature and definitively established that the more frequently sugar is consumed, the greater the risk, and that sugar consumed between meals has a much greater caries potential than when consumed during a meal.

National Surveys

Additional evidence is provided from the analysis of national surveys comparing data on caries experience and sugar supply data (table 4). These reports have established a relationship between sugar consumption and dental caries at the population level. However, it is also evident from the later surveys that the nature of this relationship has changed in most industrialized countries where fluoride use in all forms has dramatically decreased the prevalence of caries at the dentinal caries level in young children [Marthaler et al., 1996]. The weakening of this relationship in industrialized countries may also be explained by the high level of sugar consumption by a majority of the population and the well-known problem of obtaining accurate data on sugar intake [Sreebny, 1982b; Honkala and Tala, 1987; Marthaler, 1990]. The low caries prevalence and high sugar consumption in industrialized countries leaves little room to establish a clear relationship. This relationship is further complicated by the wide variation in sugar consumption patterns among individuals as well as many other factors that will be discussed in the next section of the article.

Comparison of the relationship between sugar consumption and caries among different countries is also limited by the reliability of sugar consumption data as well as the reliability of the caries data. There is a lack of consistency in how sugar consumption is reported among countries and in many cases estimates are based on 'disappearance' data (and not actual consumption) that are provided by industry or government sources. A wide range of terms are used, including sugar intake, sucrose intake, added sugar, and nonmilk extrinsic sugar, calculated as grams/person/day, kilograms/person/year, or sugar intake as percentage of total energy intake. Caries data are limited by the many well-known problems, including examiner calibration across different countries and the impact of treatment effects on the F component of DMF scores among countries. It is remarkable that, given these limitations and the complex nature of dental caries, a relationship between sugar intake and caries has been consistently demonstrated.

Table 2. Review articles on the relationship between sugar (diet) and dental caries

Author(s)	Main conclusions
Marthaler [1967]	foodstuffs containing simple sugars are far more cariogenic than common starchy foods
Newbrun [1969]	called for the specific elimination of sucrose or sucrose-containing foods rather than restricting total carbohydrate consumption
Bibby [1975]	snack foods share importance with sucrose in caries causation
Sreebny [1982a]	total consumption and frequency of intake contribute to dental caries; lacking evidence about the precise definition of the relationship
Newbrun [1982a]	compelling evidence that the proportion of sucrose in a food is one important determinant of its cariogenicity
Sheiham [1983]	sugar is the principal cause of caries in industrialized countries; recommended that sugar consumption be reduced to 15 kg/person/year or below
Shaw [1983]	studies in animals consistent with the clinical evidence on the relationship between sugar and caries
Rugg-Gunn [1986]	cariogenicity of staple starchy foods is low; the addition of sucrose to cooked starch is comparable to similar quantities of sucrose; fresh fruits appear to have low cariogenicity
Bowen and Birkhed [1986]	frequency of eating sugars is of greater importance than total sugar consumption
Walker and Cleaton-Jones [1989]	degree of incrimination of sugar as a cause of caries is grossly exaggerated; questioned predictions of reductions in caries from decreases in sugar and snack intakes
Marthaler [1990]	in spite of dramatic reductions in caries due primarily to widespread use of fluoride, sugars continue to be the main threat to dental health
Rugg-Gunn [1990]	dietary modification involving restriction on the frequency and amount of extrinsic sugars can be more effective than other control measures
König and Navia [1995]	acknowledged the relationship between frequency and sugar intake and caries; recommended removing the focus away from elimination of sugar and towards improved oral hygiene and use of fluoride toothpaste
Ruxton et al. [1999]	evidence strongly supports formulation of advice on frequency of consumption, not amount
König [2000]	dental health problems do not require any dietary recommendations other than those required for maintenance of general health
van Loveren [2000]	if good oral hygiene is maintained and fluoride is supplied frequently, teeth will remain intact even if carbohydrate-containing food is frequently eaten
Sheiham [2001]	sugars, particularly sucrose, are the most important dietary cause of caries; the intake of extrinsic sugars greater than 4 times a day increases caries risk; sugar consumption should not exceed 60 g/day for teenagers and adults and proportionally less for younger children

Systematic Review

There have been many additional epidemiologic (cohort, case-control and cross-sectional) studies evaluating the relationship between sugar consumption and caries risk. The topic has recently been the subject of a systematic review by Burt and Pai [2001], which was conducted as part of the NIH/NIDCR Consensus Development Conference on Diagnosis and Management of Dental Caries throughout Life. This review specifically addressed the question: 'in the modern age of extensive fluoride exposure, do individuals with a high level of sugar intake experience greater caries severity relative to those with a lower

Table 3. Classic evidence from humans supporting the role of sugar in dental caries

Study	Reference(s)	Main conclusions
Vipeholm Study	Gustafsson et al. [1954]	the more frequently sugar is consumed the greater the risk; sugar consumed between meals has much greater caries potential than when consumed during a meal
Turku Sugar Study	Scheinin et al. [1976]	when sugars are almost completely replaced by non-fermentable sugar substitutes (xylitol), caries increment is dramatically reduced; fructose is less cariogenic than sucrose
World War II	Toverud [1957a, b] Takeuchi [1961]	caries decreased and increased with sugar consumption during and after the war, respectively
Hopewood House	Harris [1963]	modern diet more cariogenic than vegetarian low sugar diet
Tristan da Cunha	Holloway et al. [1963] Fisher [1968]	introduction of a modern diet including sugar and refined carbohydrates to this remote island greatly increased caries prevalence
Hereditary Fructose Intolerance	Marthaler [1967] Newbrun et al. [1980]	less caries in individuals that must avoid sucrose and fructose, but not other sugars and complex carbohydrate
Experimental Caries in Man	von der Fehr et al. [1970] Geddes et al. [1978]	incipient caries can be rapidly induced by frequent rinsing with high-concentration sucrose solutions in the absence of oral hygiene
Stephan Plaque pH Response	Stephan [1940, 1944]	demonstrated the relationship between sugar exposure resulting in the acidification of dental plaque and caries experience

Table 4. Data from national surveys

Reference	Parameters	Main findings/conclusions
Sreebny [1982b]	dmft in 6-year-olds from 23 nations and DMFT in 12-year-olds from 47 nations; sugar supply (g/person/day)	significant positive correlation ($r = 0.72$; $p < 0.005$) between caries prevalence and national sugar supplies for 12-year-olds only; ingestion of 50 g of sugar/day may be the upper limit of 'safe' or 'acceptable' sugar consumption
Woodward and Walker [1994]	DMFT in 12-year-olds from 90 nations; sugar supply (kg/person/year)	linear relationship between DMFT and sugar consumption when all 90 nations were included; no evidence of a relationship with a separate analysis of 29 industrialized nations
Miyazaki and Morimoto [1996]	DMFT in 12-year-olds (kg sugar/year); 1957–1987; low fluoride exposure	excellent correlation ($r = 0.91$; $p < 0.01$) between DMFT and per capita sugar consumption in Japan
van Palenstein Helderman et al. [1996]	caries experience in Africa, Europe and North America; salivary mutans streptococci	caries experience on three continents is attributable to dietary differences and not prevailing mutans streptococci species
Downer [1999]	dmft in 5-year-olds and DMFT in 12-year-olds; available sugar (kg/person/year)	strong positive correlation over time (50 years) between caries experience and national sucrose availability in the UK

level of intake?' A total of 809 papers were identified in their initial MEDLINE and EMBASE search. Of these, 69 papers met their inclusion-exclusion criteria and were scored and recorded in evidence tables. Thirty-six papers with a quality score of 55 or higher were rated for the strength of the relationship between sugar and caries and were used as the basis for their conclusions. They reported that only 2 papers found a strong relationship, 16 found a moderate relationship and 18 found the relationship to be weak-to-none (table 5). Based on this systematic review,

Table 5. Systematic review evaluating the relationship between sugar consumption and caries risk

Study design	Relation between sugar intake and caries			
	strong	moderate	weak	totals
Cohort	1	6	5	12
Case-control	0	1	0	1
Cross-sectional	1	9	13	23
Totals	2	16	18	36

Strong = Risk ratio (odds ratio or relative risk) ≥ 2.5; moderate = risk ratio between 1.5 and 2.4; weak = risk ratio ≤ 1.4 (adapted from Burt and Pai [2001]).

the authors concluded that while the relationship between sugar consumption and caries is not as strong as it was in the prefluoride era, restriction of sugar consumption still has an important role in caries prevention.

There are limitations to these types of epidemiological studies as well. Most studies use different kinds of dietary surveys including 24-hour recall interviews, 2-, 3- and 7-day diet diaries, 7-day weighed inventories, and food frequency questionnaires. However, these tools have only rarely been validated. Furthermore, the role of sugar in caries etiology is quite complex because sugar is rarely eaten in a pure form. The cariogenicity of sugar-containing foods can be modified by many factors including the amount and type of carbohydrates (sucrose vs. other sugars, sugar/starch combinations), protective components (proteins, fats, calcium, phosphate, fluoride), and physical and chemical properties (liquid vs. solid, retentiveness, solubility, pH, buffering capacity, sialagogue properties). While some studies have measured frequency of ingestion, most studies do not account for other behaviors associated with food consumption, such as eating sequence in relationship to other foods, eating before bedtime, late night snacks, and behaviors after food consumption, such as oral hygiene and fluoride use, and gum chewing. In addition, environmental, genetic, social, economic, political and educational factors may confound the relationship between sugar consumption and caries, if not controlled for.

Other Evidence

Other indirect evidence in support of the role of sugars in caries comes from animal studies, in situ studies, plaque pH studies, and laboratory studies. In particular, animal studies have been highly supportive of the clinical data [Shaw, 1983], and have added key elements to our current understanding. The main use of these model systems has been to evaluate the cariogenic potential of individual food items with the aim of ranking them, which is something that cannot be done in human clinical trials due to the impact of a highly variable background diet. Based on two consensus conferences, one in San Antonio, Tex., USA, sponsored by the American Dental Association in 1985, and a more recent one in Hertfordshire, UK, sponsored by the British Dental Association in 1999, the animal caries and human plaque pH models were considered acceptable methods [Stamm et al., 1986; Curzon and Hefferren, 2001]. The more recent UK conference also supported the use of in situ models for this purpose.

A working group consensus report from the San Antonio conference stated that 'the true cariogenicity of a food can only be established by experimentally determining in humans the extent of tooth decay associated with a given food', while cariogenic potential was defined as 'a food's ability to foster caries in humans under conditions conducive to caries formation' [Stamm et al., 1986]. The cariogenic potential of a particular food or beverage is influenced by its properties, most importantly the sugar content and the presence of protective factors, and the consumption pattern, most importantly the frequency of consumption [Bowen et al., 1980]. Edgar [1985] further divided the possible factors that can influence the cariogenicity of foods into *food factors* (amount and type of carbohydrate; food pH and buffering power; food consistency and retention in the mouth; eating pattern; factors influencing the oral flora; factors modifying enamel solubility; sialogogue properties, and other substrates for bacterial metabolism) and *cultural and economic factors* (availability and distribution; selection, and marketing).

Several approaches have been recommended for ranking foods. Bowen et al. [1980] developed the cariogenic potential index that uses the rat caries model. The cariogenic potential index is calculated by dividing the rat caries score for the test food by the rat caries score for pure sucrose. Several authors [Krasse, 1985; Burt and Ismail, 1986] have supported the contention that a combination of the tests would be a more valid way of ranking the relative cariogenic potential of foods as has been proposed by Matsukubo et al. [1985].

Using the various models, a wide array of foods with varying types and concentrations of sugar have been shown to have cariogenic potential. Some methods in particular, such as the indwelling plaque pH method, seem to be very sensitive to even low sugar foods that are not normally implicated in dental caries, and may accentuate

food retention, creating a worst case scenario [Edgar and Geddes, 1986]. Starches have also been shown to have cariogenic potential in many model systems, including in vitro models [Renz and Bibby, 1989], plaque acidity models [Lingström et al., 1993], animal caries models [Firestone et al., 1982; Mundroff et al., 1990] and in situ caries models [Brudevold et al., 1988; Kashket et al., 1994; Lingström et al., 1994; Pollard, 1995]. However, definitive data in humans are lacking [Lingström et al., 2000], suggesting that some degree of caution should be used when interpreting results from these models.

While the ability to rank foods based on their relative cariogenic potential seems desirable, there are several problems with this approach. Both the human plaque acidity models and animal caries models do not account for how foods are actually consumed, in regard to the frequency of ingestion, patterns of ingestion, or relationship of the dietary intake of other foods, which can greatly modify the actual cariogenicity of a given food. Furthermore, the actual susceptibility of a given individual to caries will vary mainly based on the composition of their oral microflora, their salivary flow rate and composition, and fluoride exposure. In situ demin/remin models have several advantages in this regard. The question has also been raised as to how can the relative ranking of foods with different cariogenic potential be employed for dietary counseling and caries control [Burt and Ismail, 1986]. Once a food is determined to have cariogenic potential, is it ethical to recommend that food item to a patient over another food that is ranked slightly more cariogenic based on cariogenicity testing?

Special Role of Sucrose

While the original claim that 'Sucrose is the Arch Criminal of Dental Caries' [Newbrun, 1969] has been softened over the years, it continues to be the most common form of added sugar in the diet, even with continuation of the trend towards increased used of high fructose corn syrup in many industrialized countries. There does not appear to be any difference in the acidogenic potential [Imfeld, 1977] or the ability to directly induce in situ enamel demineralization [Koulourides et al., 1976] among the common sugars, sucrose, maltose, glucose and fructose. Lactose has less acidogenic potential than the other sugars and, as a constituent of milk is not considered to be cariogenic mainly due to the protective factors in milk.

Sucrose has been given special importance due to its involvement as the sole substrate in the synthesis of extracellular (water-soluble and water-insoluble) glucans meditated by microbial glycosyltransferases, which have been the subject of intense study for many years. Glucans can form a major component of the structural intermicrobial matrix of dental plaque [Guggenheim, 1970]. It has been proposed that water-insoluble glucans enhance the ability of mutans streptococci to accumulate on the smooth surfaces of teeth [Gibbons, 1984]. When compared to other dietary sugars (glucose, fructose, and lactose) in the rat caries model, sucrose has been shown to be more cariogenic in some studies. However, the effect appears to be strain specific and is also influenced by the type of animal model and the effect is generally associated with smooth surfaces [Frostell et al., 1967; Tanzer, 1979; Van Houte and Russo, 1986]. However, the specific cariogenicity of sucrose compared to equimolar mixtures of glucose + fructose was not supported by a study conducted in monkeys [Colman et al., 1977].

Based on differences in sucrose consumption between the USA and Britain, Burt [1993] suggested that replacement of sucrose by monosaccharides may reduce proximal- and smooth-surface caries. Several studies have indicated that the caries-associated virulence of glucan may have more to do with an alteration in plaque ecology than effects on the accumulation of specific bacteria in plaque, whereby sucrose-mediated synthesis of glucans increases the porosity of plaque, permitting deeper penetration of dietary sugar into the biofilm and greater acid production immediately adjacent to the tooth surface [Dibdin and Shellis, 1988; van Houte et al., 1989; Zero, 1993]. In studies using an intraoral caries model, *Streptococcus mutans* plaque prepared from sucrose-containing cultures was found to have markedly enhanced demineralization potential compared with glucose grown plaque [Zero et al. 1986; Cury et al. 2000]. The effect has been attributed to an alteration of the diffusion properties of plaque due to the presence of water-insoluble glucan synthesized from sucrose. Recent studies have found a relationship between water-insoluble glucan synthesis by mutans streptococcal strains and caries incidence in young children and suggested that the capacity of mutans streptococci to synthesize insoluble glucans may be more important than their levels in plaque [Mattos-Graner et al., 2000; Nobre dos Santos et al., 2002].

Where Are We Now?

While the classical literature continues to inform us, more recent data indicate that the relationship between sugar consumption and dental caries is not as strong as it

was in the prefluoride era. In industrialized countries where fluoride exposure has become the norm through the use of fluoride dentifrice and/or water/salt fluoridation and other vehicles of fluoride delivery, the relationship between sugar and caries has been more difficult to demonstrate. This has led some authors to conclude that recommendations to restrict sugar consumption may no longer be necessary [König, 2000; van Loveren, 2000]. Clearly, fluoride has raised the threshold at which the caries process will progress to a frank cavitation, in that a higher cariogenic diet can be tolerated before caries occurs in many individuals. However, fluoride has its limits, and caries remains a serious problem for economically disadvantaged individuals and new immigrants in many highly industrialized countries. It is a rising problem in many developing countries, where sugar consumption is increasing, fluoride use has not been widely adopted and the provision of dental care is not available. The expected results from these population-based human experiments have unfortunately been shown time and again.

There continues to be a discussion about the nature of the relationship between sugar intake and caries and whether there is a safe level of sugar intake. Newbrun [1982b] proposed that the relationship can be best described by an S-shaped curve based on animal studies, and speculated that the S-shaped curve may have moved to the right in the postfluoride era (fig. 1a and b, respectively). Woodward and Walker [1994] reported that the relationship is linear based on their analysis of sugar consumption in 90 countries (fig. 1c). In individuals with good oral hygiene and regular fluoride exposure, higher levels of sugar consumption may be tolerated before caries occurs (author's conjecture) (fig. 1d).

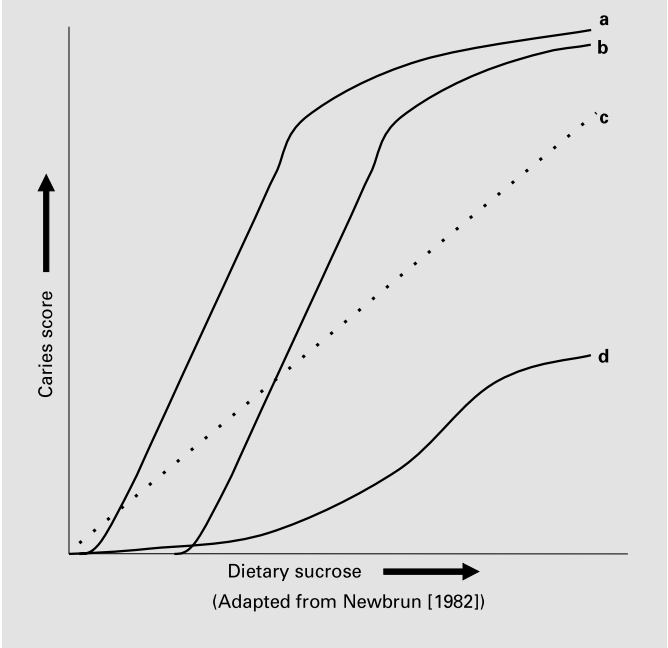

Fig. 1. Proposed relationships between sugar intake and caries. **a** S-shaped relationship in the prefluoride era [Newbrun, 1982b]. **b** S-shaped relationship shifted to the right in postfluoride era [Newbrun, 1982b]. **c** Linear relationship calculated from data in 90 countries [Woodward and Walker, 1994]. **d** Individuals with good oral hygiene and regular fluoride exposure (author's conjecture).

Future Perspectives for Research

Given that not everyone on a high sugar diet will get caries, research should be directed at determining the biologic and behavioral factors that influence diet-related caries risk. The importance of sugar concentration in relationship to plaque accumulation at caries-susceptible sites requires further investigation, especially in regard to fluoride regimes necessary to counteract the caries challenge and the limitations thereof. There is a need for clinical studies that specifically address the role of sucrose compared to other sugars, including the clinical testing of existing intervention strategies and the development of new strategies directed at blocking the glucan-mediated cariogenic effects of sucrose. A related question for study is – how much dietary sucrose/sugar exposure is necessary to change the ecological conditions in plaque biofilm in favor of caries progression? Another conjecture worthy of investigation is that if glucan also enhances the diffusion of fluoride as well as sugar substrates through plaque, this may explain in part why fluoride is effective in counteracting the cariogenicity of sucrose. Research should be directed at the problem of increased soft drink consumption, especially high sugar/high caffeine products marketed at populations (adolescents). With an aging dentate population, the relationship between diet and root caries needs more attention. The advances in food science and the interest in 'functional foods' create many opportunities to decrease the cariogenic potential of high sugar foods by including protective additives.

Summary and Implications

While some questions have been raised by recent epidemiological data, the importance of sugars as the principal dietary substrate that drives the caries process has not

been scientifically challenged. The fact that sugars are readily metabolized by oral bacteria, leading to the production of organic acids in sufficient concentration to lower the pH of dental plaque was first shown in clinical studies by Stephan in the forties [1940, 1944]. The direct linkage of the frequent exposure to sugar with dental caries was firmly and irrefutably established by the Vipeholm Study. Experimental caries models in man, animals, and in vitro have all confirmed this linkage.

The overall weight of this evidence is exceptionally strong, and based on this review, as well as many others, a causal relationship between sugars and dental caries has been established. This does not mean that other carbohydrates such as starches or different combinations of sugars and starch are not cariogenic. Given the availability of food at every turn and our human propensity to graze, it is very likely that many starch-containing processed foods can contribute to caries formation. All research data are subject to criticism and it is relatively easy to create a climate of doubt in the minds of the public as is evidenced by the effectiveness of antifluoridationists in some parts of the world. To put it in another context, the clinical evidence implicating frequent consumption of sugar in the etiology of caries is much stronger than the evidence supporting the widely held belief that caries is an infectious disease caused by mutans streptococci. This contention is mainly based on studies in coprophagous rodents, and attempts to control caries using this approach have been unsuccessful thus far. However, there are not any multibillion-dollar industries with a vested interest in discounting the role of a specific microorganism.

Although caries has declined in many industrialized countries (even in the presence of increased sugar consumption), we should not be complacent. Dental caries still remains a very costly and widespread disease that in many industrialized countries affects mainly disadvantaged individuals and is of serious concern in many developing countries. At the population level, in industrialized countries, measures to educate the public on the dangers of frequent sugar consumption (especially foods with high sugar concentration) in conjunction with recommendations for proper oral hygiene and fluoride use are still warranted. In developing countries, public health strategies need to be developed to ensure that adequate educational resources and dental public health manpower are available before dental health problems manifest. On an individual basis, dietary counseling is highly recommended for patients that show signs of caries activity and/or are at high caries risk (hyposalivation, iatrogenic factors such as orthodontic brackets). Given that dental caries is a preventable disease, each country must decide: what level of disease is society willing and able to tolerate?

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