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Nutritional role of sugars in oral health^{1,2}

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ABSTRACT The dental risk of dietary sugars is dependent mainly on the frequency of intake, but the prevalence of caries in a population is strongly modified by other dietary, social, and behavioral factors independent from intake of sugars. Regarding dietary factors, it must be remembered that hidden sugars in fruit as well as polysaccharides are cariogenic. The most important of the other factors is regular tooth brushing, which results in the removal of the bacterial plaque that causes caries and periodontal diseases and makes fluoride (which is contained in every advanced toothpaste) available for maintenance of the hard dental tissues and for remineralization wherever demineralization has occurred. This explains why in most highly developed countries caries prevalence has decreased markedly during the past 20 y although consumption of sugars remained high. *Am J Clin Nutr* 1995;62(suppl):275S-83S.

KEY WORDS Sugars, fruit, starches, dental caries, periodontal disease, bacterial plaque

INTRODUCTION

A discussion of the nutritional role of sugars in oral health requires consideration of three types of oral tissue with different structure, morphology, metabolism, and pathologic response: the hard tissues of the teeth (dental health), the supporting structures of teeth (periodontal health), and the oral mucosa (mucosal health). We will discuss dental health in more detail than periodontal health, and mucosal health will not be discussed separately. The reason for this selectivity is the high prevalence of caries in hard tooth structures and the etiologic role of dietary carbohydrates in general and sugars in particular as caries-promoting factors that put oral health at risk. Risk begins immediately after tooth eruption and lasts as long as teeth are present in the oral cavity; however, the enamel covering the anatomical crowns of teeth is not subject to systemic nutritional influences after tooth development and eruption.

The role of nutrition in general on the growth, development, and maintenance of oral tissues was recently discussed (1), and two major effects are clear: one systemic and the other local and dietary. The main result of nutrition is the systemic effect of absorbed nutrients on the growth, development, and maintenance of the tissues and organs and their functions. Local dietary side effects are of great practical importance, especially in the oral cavity. Dental enamel after eruption is particularly subject to local side effects from whatever enters the mouth. Diet and dietary components not only provide essential nutri-

ents for the host tissues but also for bacteria in the oral cavity and the digestive tract, which use them as readily available substrates. Direct as well as indirect side effects of nutrients result from their ion content, acidity, and physical properties. Erosion is a dental problem that often occurs as the result of frequent consumption of acidic food such as lemons but less so from consumption of manufactured foods that are properly formulated to prevent erosive effects of food acids.

It is therefore important in a discussion of oral health and its relation to nutritional factors that we differentiate between formative nutritional influences and post-eruptive local influences. The effect of nutrition on dental development is usually constructive. However, the influence of diet and its local side effects may be either damaging or stabilizing. Dental integrity is maintained as the result of a continuous interaction of protective and destructive influences. The difficulty is that nutritive and dietary protective and destructive factors act on the same tissues and their effects cannot be differentiated readily and assessed separately. Although formative influences are effective in an early distinct period of development and destructive influences usually do not start before exposure and functioning, the difficulty of distinguishing effects still exists. A well-known example of this is the question of the mechanism of action of water-borne fluoride. When fluoride in drinking water in the United States was first discovered to be the cause of mottling of teeth and was later also identified as the protective factor against caries, the conclusion was rapidly drawn: fluoride has an effect on teeth during formation that shows as mottling after eruption; therefore, the effect we see later on, fewer and smaller carious lesions, will likewise be due to the pre-eruptive deposition of fluoride. It took decades before the great importance of life-long topical fluoride availability was clearly recognized and dental scientists agreed that pre-eruptive administration of fluoride was less important.

With respect to the osseous, periodontal, mucosal, salivary gland, dentinal, and pulpal tissues, development and life-long integrity and functioning are associated with systemic molecular and cellular reactions to variables associated with nutrition, some of them interacting with local oral factors and bacterial antigens. Tooth enamel, in contrast, is subject to systemic influences on its development before eruption only; after eruption it interacts with local (topical) environmental factors exclusively.

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Because of the importance of local effects in controlling demineralization of teeth and inflammation of periodontal structures, which are the major risks to oral health, this review deals with the dietary side effects of sugars rather than with metabolic nutritional aspects. For the maintenance of periodontal and mucosal health, however, systemic nutritional factors are more relevant than they are in the maintenance of tooth structures. However, even in this case, the absence of local bacterial and cytotoxic irritants deserves more attention than do systemic nutritional factors.

DENTAL HEALTH

Etiology of caries

Dental caries is a bacterial plaque-dependent disease of the dentition that is characterized by a progressive, intermittent demineralization of enamel, dentin, and cementum, with a characteristic pattern of decay that may lead to total destruction of coronal dental tissues and the formation of pulpal abscesses. Oral microorganisms, when organized in voluminous masses as dental plaque on tooth surfaces, hydrolyze starches and metabolize sugars to form acids (mainly lactic acid) that demineralize the hard tissue underneath. Although the basic mechanism is simple, it is actually influenced by many factors that interact and are difficult to quantify.

Enamel consists of very small crystals, between which there is mainly water and some remnants of organic matrix. The volume not occupied by crystals and organic matrix is called the pore volume. The water can move freely and is the vehicle for ions diffusing in and out, exchanging with oral fluids. The water can also dry out in certain areas when air fills the pores otherwise filled with water. The normal total pore volume of enamel is 10%, but it is higher after demineralization and in fluorotic enamel. In the early phases of acid attack, demineralization is a diffusion-controlled process resulting in an increase in pore volume only, without disintegration of the mineralized tissues. Supply and access of saliva, which contains buffering systems and transports mineral and fluoride ions, contribute to remineralization between acid attacks. Both processes, demineralization and remineralization, are modified by numerous variables.

Figure 1 shows the major interrelated factors responsible for the development of dental caries (2). In the center are local oral factors headed by the bacteria in plaque. These bacteria include not only those that have been shown to be highly cariogenic, such as *Streptococcus mutans* and the lactobacilli, but others that are less cariogenic, such as species of *Actinomyces*, and still others, such as *Veillonella* that use lactic acid as a source of energy and thus interfere with acid accumulation and lowering of the pH in plaque, a phenomenon that slows down the caries process. There are four aspects that are important relative to the cariogenic action of bacteria in plaque and that should be considered as separate effects: the implantation mechanism involving the phenomenon of adhesion, colonization through competition with other bacterial components, the metabolic activity of established bacteria under the influence of food residues and oral environment, and increased thickness of plaque and its control by oral hygiene procedures.

The second component is saliva, an important protective biological fluid in the oral cavity (3). Foods stimulate saliva

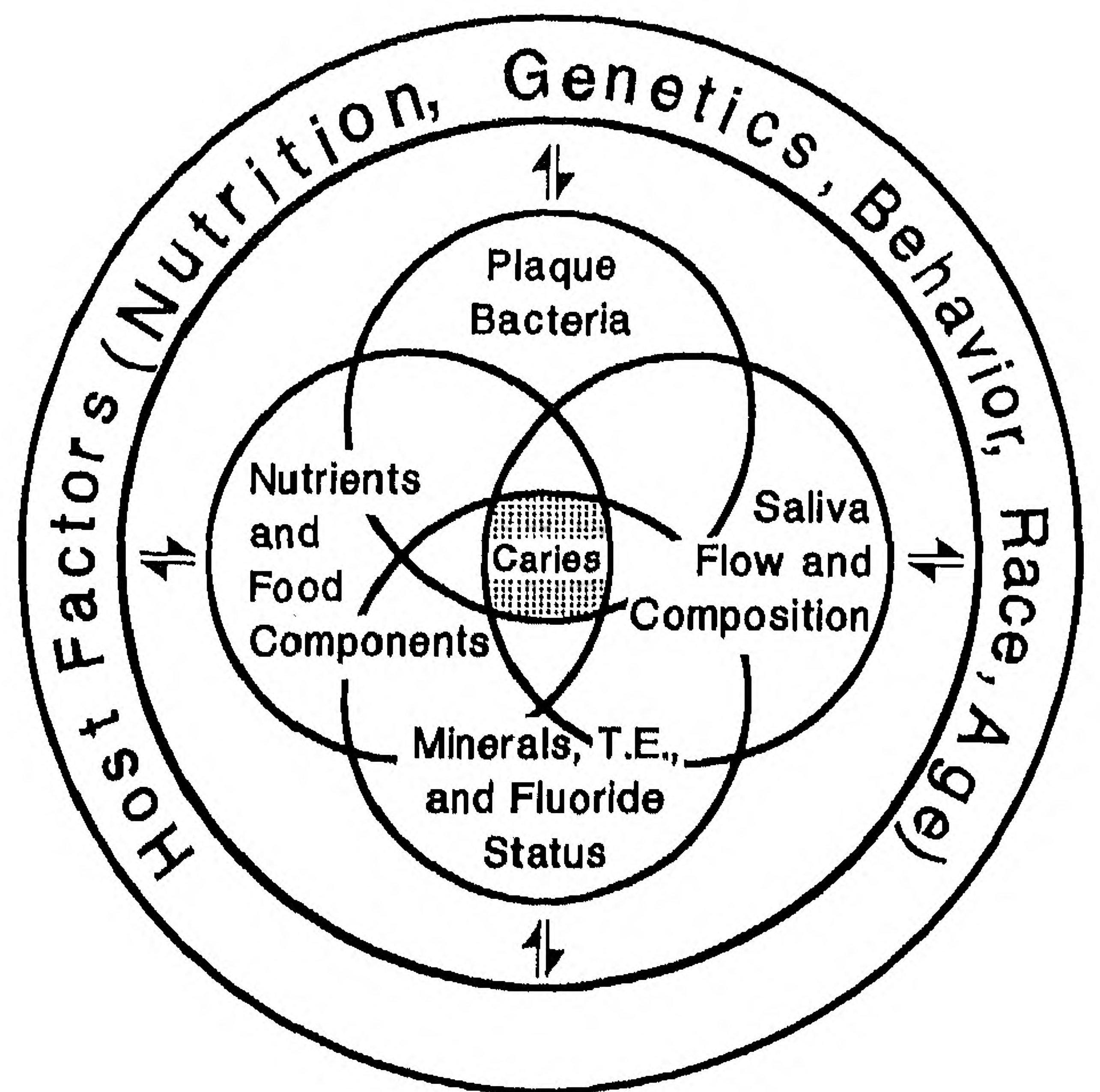


FIGURE 1. The multifactorial etiology of dental caries. T.E., trace elements. From Navia (2).

production through olfactory, masticatory, and gustatory actions; the amount, flow, and composition of saliva can be greatly influenced by these physiologic stimuli. Salivary gland secretory function can be influenced early in development by nutritional insults and later through malnutrition or the chronic effects of age-related conditions (4, 5). Although aging per se does not seem to have an overt effect on salivary gland function (6, 7), malnutrition, diseases, and chronic medication, which are frequently associated with aging, may compromise the ability of the gland to protect oral tissues. For example, anti-hypertensive drugs, those used in Parkinsonism therapy, antidepressants, and many others commonly used by older people can induce mild or even severe dry mouth syndrome (xerostomia) and other pathologic side effects (3). Thus a dry mouth caused by impaired salivary function may result in an increased prevalence of malnutrition in elderly people (8).

Another important group of factors includes minerals and trace elements, particularly fluoride. In the oral cavity, calcium and phosphorus in plaque and saliva are major contributors to the remineralization of enamel surfaces affected by acid. Calcium and phosphorus are also beneficial to the maintenance of skeletal tissues, such as the alveolar bone supporting the dentition. The concentration of these ions can be further increased by drinking water or food residues rich in calcium and phosphorus. The beneficial effect of certain cheeses can be traced largely to their high mineral content, which, together with the protein in the cheeses, neutralizes the acidogenic effects of sugars (9, 10). The presence of low concentrations of fluoride ions in plaque, saliva, and enamel originally provided by the water, food, or other sources, contribute substantially to remineralization after each demineralizing acid attack (11). This natural repair process is essential for maintaining the integrity of enamel, exposed dentin, and cementum surfaces.

The final factor in this group of local, oral etiologic components is nutrients and food residues left in the oral cavity after food has been masticated and the food bolus swallowed or after

beverages have been drunk. These residues are retained on the dorsum of the tongue and in other retentive sites in the oral cavity. From these locations they continue to provide soluble and nutritious substances that can be used by plaque bacteria as specific substrates to enhance any of the four activities previously mentioned: implantation, colonization, metabolic activity of the plaque bacteria, and thickening of the plaque. Sugars and starches are a part of these residues and exert their effects on plaque depending on the type of carbohydrate (sucrose, glucose, polysaccharide, sorbitol, etc), the concentration and retention time in the oral cavity, or the frequency with which the fermentable carbohydrates are replenished by repeated intake, and the action of saliva in diluting this substrate, in converting starch into fermentable glucose and maltose via amylases, or in providing the necessary buffering capacity to neutralize acid end products that result from such fermentations (12, 13). All food carbohydrate residues have caries-promoting properties rather than cariogenicity (sugars do not directly damage the teeth) and, therefore, determine caries indirectly under conditions that allow local fermentation and acid accumulation. If mature, the plaque has increased pathogenic potential and retention of carbohydrates occurs, conditions that considerably stimulate the carious process. The fermentability and caries-promoting differences among different sugars are not large. Sucrose has always been, and will remain, the primary sugar in human nutrition; most foods, if they contain sugars, contain a mixture of sugars. A diet without sugar would be very exceptional.

Taking together the various plaque-born microbial components listed above and retained food residues acting as substrates for acid formation, it is obvious that a person's oral hygiene practice can greatly influence the caries-forming process (provided oral hygiene techniques result in regular removal of both plaque and food debris). If a dentifrice containing fluoride is used, this important ion that favors remineralization will be present frequently in effective cariostatic concentrations. Oral hygiene, therefore, must be considered an important modifying factor; whether it really becomes effective depends on whether it is a habit. This in turn is a behavioral issue dependent on cultural, economic, and sociologic patterns and conditions.

As teeth age, the clinical crowns become longer because of gingival recession. The roots are exposed and become prone to carious destruction, starting with the (thin) cementum and progressing into the root dentin. The etiologic mechanism of cementum and dentinal caries of roots does not, in principle, differ from coronal caries, which starts in enamel and progresses into dentin. Occasionally, the composition of the bacterial plaque flora on root surfaces differs from that on enamel surfaces, but acidogenic microorganisms always play key roles in the development of all types and sites of caries attack. Root lesions usually progress more quickly than do enamel lesions. This is due to the low degree of mineralization of root cementum and dentin, to difficulties in keeping root surfaces clean with routine brushing of teeth, and to a decrease of salivary secretions, which is rather common in elderly people. As with coronal (enamel) caries, high frequency of sugar intake increases the risk of root caries whereas good oral hygiene and regular external fluoride administration minimize the risk of root caries.

Sugar consumption, its availability or disappearance, and dental caries

Sugar consumption is a risk factor for caries, and it is therefore important to consider intake of sugar as well as changes of other major variables over time when interpreting epidemiologic data from a population. In view of the complexity of the etiology of dental caries, it is not difficult to understand why one factor, such as the amount of sugar available for consumption in a country, cannot confidently be extrapolated to explain the disease severity at all sugar intakes. An absence of one of the etiologic factors will produce a major change in the caries process. For example, lack of dental plaque or absence of fermentable carbohydrates will be associated with low caries risk, whereas compromised salivary function or absence of fluoride will enhance the carious process. Extreme situations produce clear results, but most people are in intermediate ranges for these factors. In such circumstances it is the interaction between two or more factors, rather than a single main factor, that determines disease outcome (2).

Determination of sugar consumption in a country, let alone in a region, is difficult because of many factors influencing such data: different methods used to record total sugar consumption, some data given as raw sugar and some as refined sugar, most of the data representing sugar disappearance and not actual consumption, and data not corrected for export and import in industrialized countries. England and the Netherlands, for example, produce a lot of sweets, export substantial amounts, and import some from Switzerland. Switzerland exports much more chocolate than it imports, and in all countries there is an imbalance between import and export of sugar in the form of sweets. The European Community authorities in Brussels have adopted a system of correction for import and export so that the real amounts sold and disappearing in a country can be determined.

In many parts of the developing world there is widespread consumption of sugar cane and other raw sugar-containing products that are not included in sugar-consumption or sugar-disappearance data even though they contribute to the caries process (14). Attempts to evaluate sugar consumption is further complicated because 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. Recently an assessment was made despite these difficulties (2) and some points are dealt with later.

Early epidemiology of caries

Several studies in humans (15-18) and experimental animals (12, 19) 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 delivery of fluoride as the main interventions recommended to control and prevent caries. Extensive research has reaffirmed the importance of these two measures in caries. This research has also contributed to an understanding of the role of diet, foods, nutrients, and nutrition in general in caries formation; a review of these facts is not within the scope of this review but they have been discussed extensively elsewhere (1, 2).

Despite the high degree of variability that exists in the consumption of various carbohydrates, specifically sugars, as well as in the different food vehicles in which they are consumed, several attempts have been made to study the relation between sugars consumption and dental caries (20). The study of this relation is complicated by the following limitations, among others (2): 1) Sugars are consumed in different physical forms with different foods and different dietary patterns that affect the clearance rate of dietary sugars; 2) the data, whether obtained from food balance sheets or from dietary surveys, represent an approximation to the actual sugar consumption; 3) the data give information relating to consumption during the year, but carious lesions sometimes take several years to develop in either the primary or secondary dentition; and 4) caries prevalence is influenced by many other etiologic factors including fluoride availability, health services, education, oral hygiene practices, and other habits and circumstances.

Regardless of these confounders, useful and meaningful data have been collected. Summarizing early studies, Sreebny (20) reported that in populations with high availability of sucrose ($57\text{--}115\text{ g} \cdot \text{person}^{-1} \cdot \text{d}^{-1}$), 12-y-old children had a number of decayed, missing, and filled teeth per individual (DMFT values, or number of unsound teeth) ranging from 3.2 to 4.8 compared with 0.1 to 2.7 in paired children in populations having average sucrose intakes $< 50\text{ g} \cdot \text{person}^{-1} \cdot \text{d}^{-1}$. There were, however, some apparent discrepancies. In Samoa and Swaziland, where people had sucrose availabilities of 78 and 99 $\text{g} \cdot \text{person}^{-1} \cdot \text{d}^{-1}$, respectively, 12-y-old children had a DMFT of 1.1. Sreebny calculated the correlation coefficient for data from 47 nations to be 0.72, which suggested that $\approx 50\%$ of the variability in caries could be explained by the sucrose available, whereas there were obviously many other factors that accounted for the remaining sources of variability (*see* confounders listed in the above paragraph).

In another comprehensive review, Sreebny (21) evaluated the voluminous literature dealing with the relation between sucrose consumption and dental caries. The conclusion emerged that communities or groups of people who had very low intakes of sugar showed very low caries scores, and those that had very high availability of sugars in their diets at that time (up to the 1970s) developed high numbers of carious lesions. A recent report (22) found no sugar-caries relation in developed countries whereas the association continued to be noted in developing countries. This means that because of oral hygiene and regular use of remineralizing fluoride, sugar exposure no longer has the strong causative effect on caries activity that it had in the pre-oral-hygiene era, which still prevails in developing countries.

Recent epidemiology of caries

The relation between sucrose intake and caries was initially established by the Vipeholm study (15) and reaffirmed by the extensive reviews of Sreebny (20, 21), which described the many investigations supporting this relation. The main aim of the Vipeholm study was to determine the effects of frequency and quantity of sugar intake on the formation of carious lesions. After exposure of dental plaque to sugar, bacteria can produce acid only for a limited period, on average, 0.5 h. The Vipeholm experiment showed that restriction of sugar intake to the four main meals daily did not significantly increase the (low) baseline caries activity, even if large amounts of sugar

were given. On the other hand, when 8 or 24 between-meal snacks were given daily, caries incidence rose dramatically because of the total demineralization time per day increasing to 4 and 12 h, respectively. However, there is a certain resemblance between these early epidemiologic data and the conditions under which the Vipeholm study was conducted: the Vipeholm population consisted of mentally retarded, severely handicapped subjects who were unable to perform any oral hygiene technique and there were no dental hygienists available at that time (early 1950s) to do regular professional cleaning. A similar condition prevailed in normal populations even in developed countries until the 1970s. Regular oral hygiene was only performed by a minority and tooth brushing was particularly unpopular among children of school age, in whom nearly all the caries epidemiologic studies were performed.

This situation has changed conspicuously in the past 20 y, at least in the hygiene-conscious populations of the United States, Scandinavia, Switzerland, and the Netherlands, where tooth brushing among children has become a habit that has kept plaque accumulation low. It is well known that sugars do not give rise to the production of dangerous amounts of acid in the oral cavity when plaque is absent or only present in thin layers. Therefore, it is feasible to separate modern epidemiologic research from early findings accumulated in the prehygiene era. This is important because during the time when oral hygiene practice developed, the sale of toothpastes containing fluoride rose to $> 95\%$ of total toothpaste sales in the countries mentioned. Regarding the populations in developing countries and certain subpopulations not practicing oral hygiene in developed countries, frequent consumption of sugar-containing foods and drinks still results in high caries risk.

Although the direct relation between sugars and dental caries is accepted by all dental clinicians and researchers, 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 focus solely on the elimination of sugar from the diet, the currently emerging view emphasizes the need to adopt a more comprehensive preventive approach that recognizes the importance of improved dietary habits, better oral hygiene, appropriate use of fluoride and sealants (plastic coating of fissures for caries prevention), enhancement of salivary function, and other considerations in an integrated and complete oral health program throughout the life of the individual.

There have been many attempts to study the specific relation between sugar intake, dietary and nutritional variables, and dental caries in humans. Walker and Cleaton-Jones (23), without rejecting the well-known effect of sugar on caries, have identified several situations in caries epidemiology reported in the literature for which sugar intake alone did not explain the caries found in the consumers. Additionally, a study of diet, oral hygiene, and dental caries in 457 Canadian children did not reveal meaningful correlations between independent and dependent variables (24). Three studies conducted on English (25), American (26), and Canadian (27) children deserve special consideration (2). The English study involved 405 children, with a mean age of 11.6 y, who were followed for 2 y. Statistical evaluation of the data indicated that a daily consumption of 118 g sugars (43 kg/y), which also accounted for

21% of total energy intake, had the highest significant correlation (0.143) with caries increments. The gingival index (severity of periodontal inflammation) increased the multiple correlation to 0.193, but other variables (sex, social class, and tooth brushing frequency) did not. Thus, total sugar intake explained only 4% of the variance in caries increment; 94% of the variance remained unexplained by factors considered in the study. The authors believed that much of the variance was due to methodologic errors, such as those related to dietary assessment, and a large intersubject variability that could mask a true relation. Although this may be true, other etiologic factors such as saliva, plaque, fluoride, and oral hygiene practices should not be dismissed lightly. In this study, frequency of sugar consumption, although significant, had a much lower correlation value (0.099) with caries than did the weight of sugary foods consumed (0.143). The study also provided some interesting conclusions: it was important to avoid sugary foods shortly before bedtime ($R = -0.101$ for time between last food containing $> 10\%$ sugars and bedtime), milk intake was positively correlated with caries increment ($R = 0.102$), and caries and vitamin D intake were negatively correlated in boys only ($R = -0.117$, $P = 0.0004$).

The US study (26) was a 3-y investigation in a nonfluoridated community in Michigan of 499 children aged 11–15 y. These children consumed on average 142 g sugar/d (51 kg/y) and sugars accounted for 26.5% of their total daily energy intake. Pit and fissure caries were not correlated with any aspect of sugar consumption but increments in carious lesions on adjacent (approximal) surfaces of teeth were related to dietary sugar intake variables. Although children who consumed a high proportion of their energy intake as sugar had the higher increases in approximal caries, there was no relation between caries scores and average frequency of eating during the day or average number of sugary snacks consumed between meals. Results of this study did not differ much from those obtained in the English study and suggest 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 places was not sufficiently caries promoting to make a major difference in their caries experience.

A third study was performed in Canada (27) with 232 11-y-old schoolchildren to evaluate the association between dietary patterns and dental caries. Nutritional data were 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 best compliance with nutritional recommendations). The frequency of sugary food consumption at mealtime and between meals was also considered. Results indicated that the nutritional status of these children was compromised. Almost 50% of the children had a nutritional quality level of 3 and only 8.2% of the subjects had a quality level of 5, which is the minimum recommendation of the Canadian guidelines for the use of the four food groups, a system devised to facilitate the selection of foods to ensure a nutritionally adequate diet. Although there was a trend toward a lower caries increase with improved nutritional quality of the diet, analysis of variance did not show statistical significance. Furthermore, no significant association was observed between frequency of consumption of sugary foods and increments in caries.

Although dietary sugars are unquestionably an important determinant in the development of caries, they are not the sole etiologic factor. This is highlighted in studies in special populations such as mentally retarded children (28), in whom the frequent consumption of candy did not seem to be a significant determinant of caries; rather, poor oral hygiene status appeared to be a more important caries risk factor. Similar conclusions were reached in a study evaluating the oral health of Latin American preschool children living in Malmö, whose general and dental health status was inferior to that of native Swedish children (29). Children with gums practically free from clinical signs of inflammation had only 1.1 tooth surfaces that were not sound, whereas those with gingivitis with and without bleeding on probing had 6.2. Another study, which examined the oral health status of Greek immigrant children compared with Swedish and rural Greek children, illustrated the complexity inherent in the evaluation of determinants of oral health (30). The carbohydrate content of the diet consumed by these three groups was approximately the same, but rural Greek children had only 15% caries-free primary teeth and a prevalence of decayed and filled tooth surfaces in primary and permanent teeth that was higher than in the other two groups. There was also a similar distribution of mutans streptococci and lactobacilli in the three groups, which 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 was made evident by the increased use of toothbrushes and improved oral hygiene of these groups compared with the group of rural Greek children and this could have been an important determinant of their oral health.

The studies described above consisted of cross-sectional observations. However, there are also several interesting studies over time that cover secular changes in caries prevalence over some decades. For example, it was found that reduction in availability and intake of sugar in Europe during World War II was accompanied by a decrease of caries prevalence in the populations involved, but these effects were not permanent and disappeared when the dietary restrictions ended (31).

Both severe sugar restriction and optimal fluoride availability decrease caries prevalence. The relative effect of the two inhibitory factors can be judged from the percentages of caries-free children in Basel, Switzerland, subjected to prewar, wartime, and postwar sucrose supplies (32, 33) (Figure 2). Before the war, when an unlimited sugar supply was available and children had poor oral hygiene and no fluoride was available, the percentage of caries-free 7-y-old children did not exceed 2–3%; wartime restrictions reduced the sugar supply from ≈ 40 to $16 \text{ kg} \cdot \text{person}^{-1} \cdot \text{y}^{-1}$, with a resulting increase in the percentage of caries-free children to $\approx 15\%$. This improvement of oral health tended to disappear when sugar became freely available after the war. At that time the improvement seemed impressive, but it ceased being impressive when compared with the improvement after fluoride supplies, fluoride dentifrices, oral hygiene instructions at school, and water fluoridation became available in 1962. Although sugar consumption rose rapidly after World War II and has been $\approx 45 \text{ kg}$ per capita during the past 40 y, the number of caries-free schoolchildren aged 7–15 y had risen to 65% by 1989 and the DMFT index for 12-y-old children had decreased to 1.0 (32).

The Netherlands is one of the industrialized countries where caries prevalence within the past 25 y has decreased rapidly

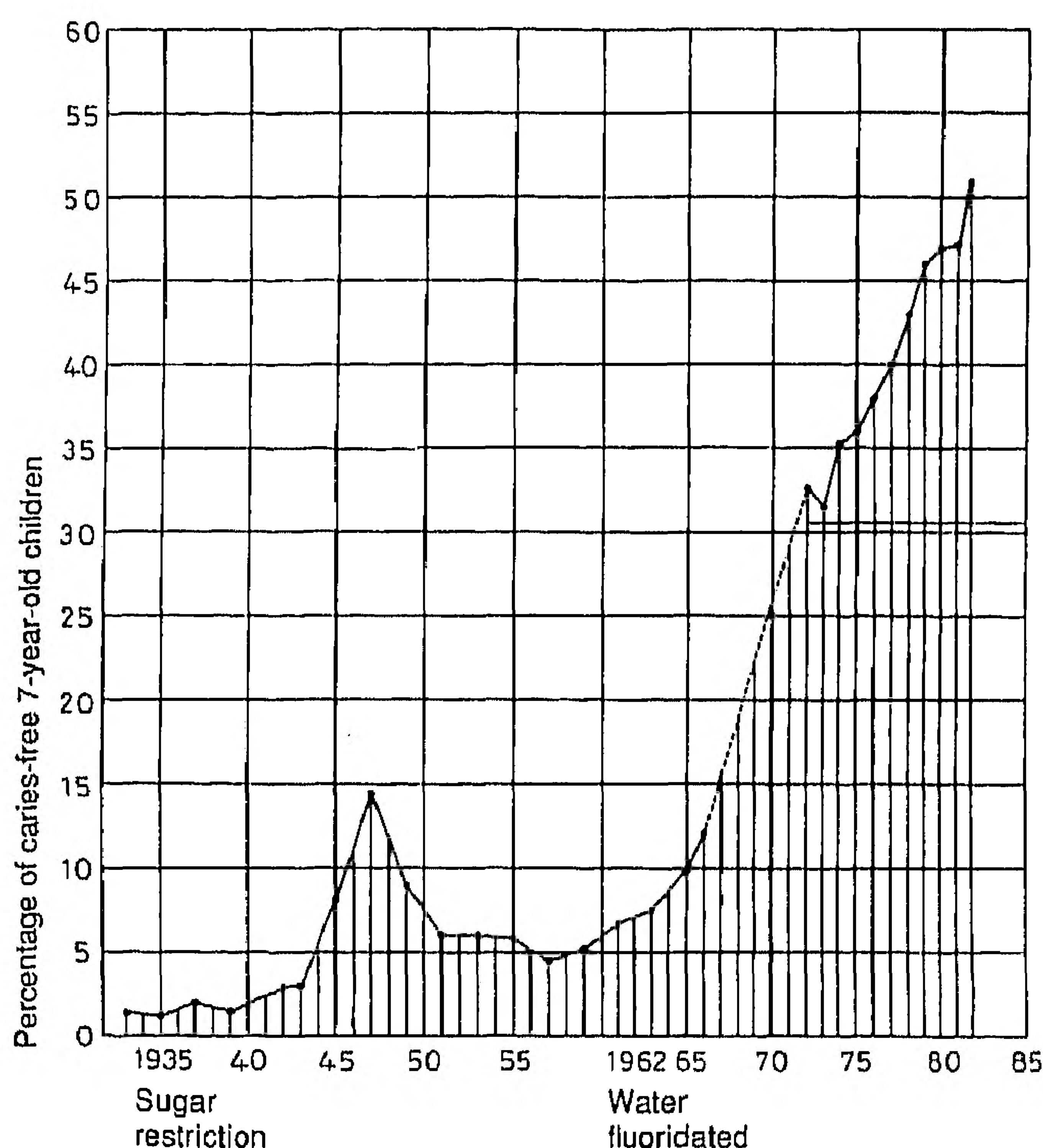


FIGURE 2. Percentages of caries-free first grade (7-y-old) schoolchildren in Basel, Switzerland, between 1933 and 1982. From König (33).

(33). By 1993 the average DMFT of 0.8 in 12-y-old children was substantially lower than the goal that the World Health Organization had set for the year 2000, although sucrose consumption was still $>90\%$ of what it was in 1965 [$38.5 \text{ kg} \cdot \text{person}^{-1} \cdot \text{y}^{-1}$ in 1985 and in 1992; see Figure 3, which is based on König (33) and Truin et al (34)]. In Sweden (35), Norway (36), and New Zealand (33) sugar consumption increased between 1982 and 1985, but regular epidemiologic monitoring of caries data showed that the caries rate in children continued to decrease (Figure 4).

In his analysis of these secular trends, Marthaler (37) concluded that in many highly developed industrialized countries there was no longer a correlation between caries prevalence

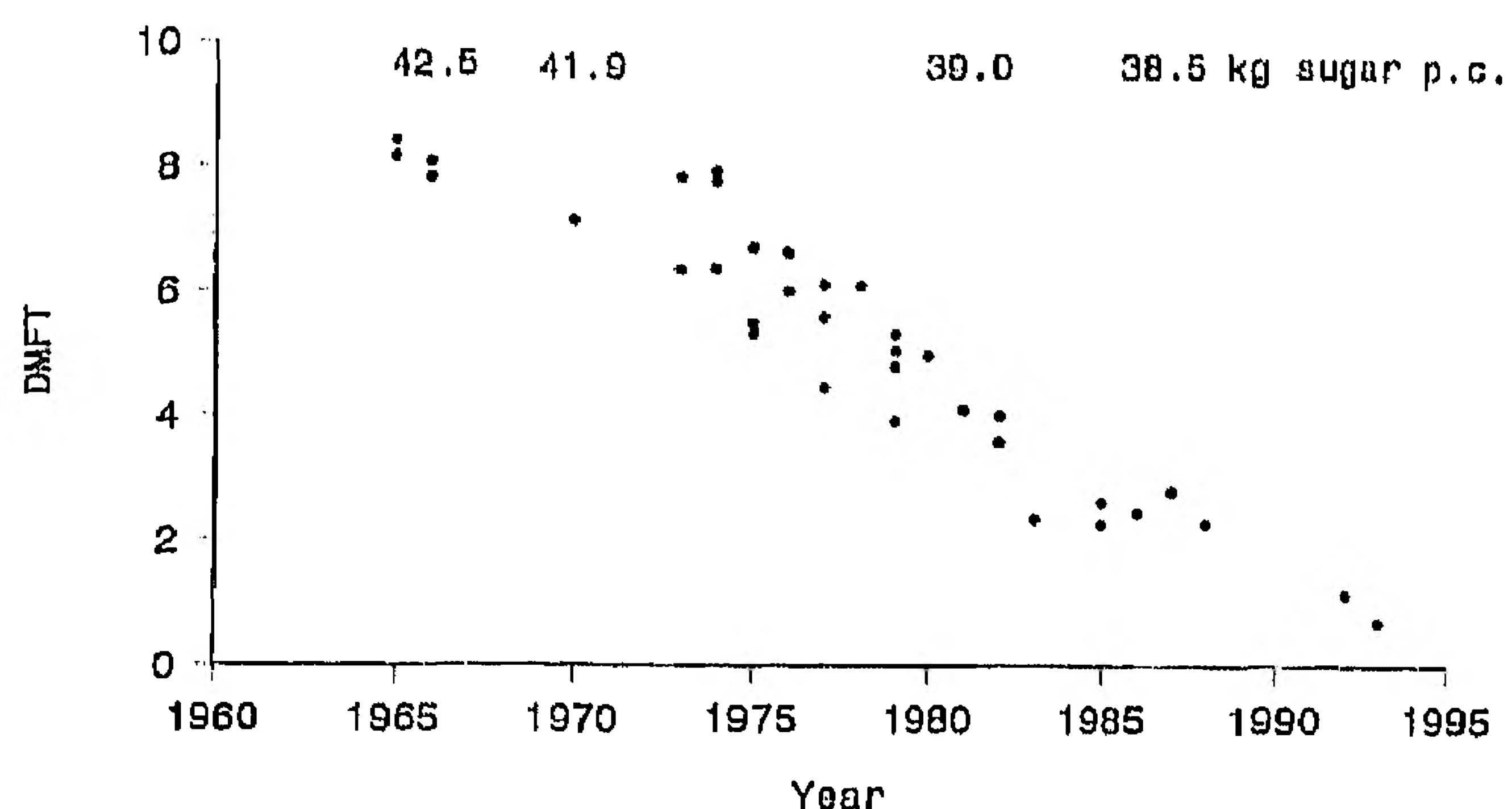


FIGURE 3. Average number of decayed, missing, and filled teeth (DMFT) in 12-y-old children in the Netherlands between 1965 and 1993, and per capita (p.c.) sugar disappearance per year in the Netherlands, corrected for import and export of sugar products. Each dot represents the average DMFT of a group of children ($n = \approx 100$) in a field study. From König (33).

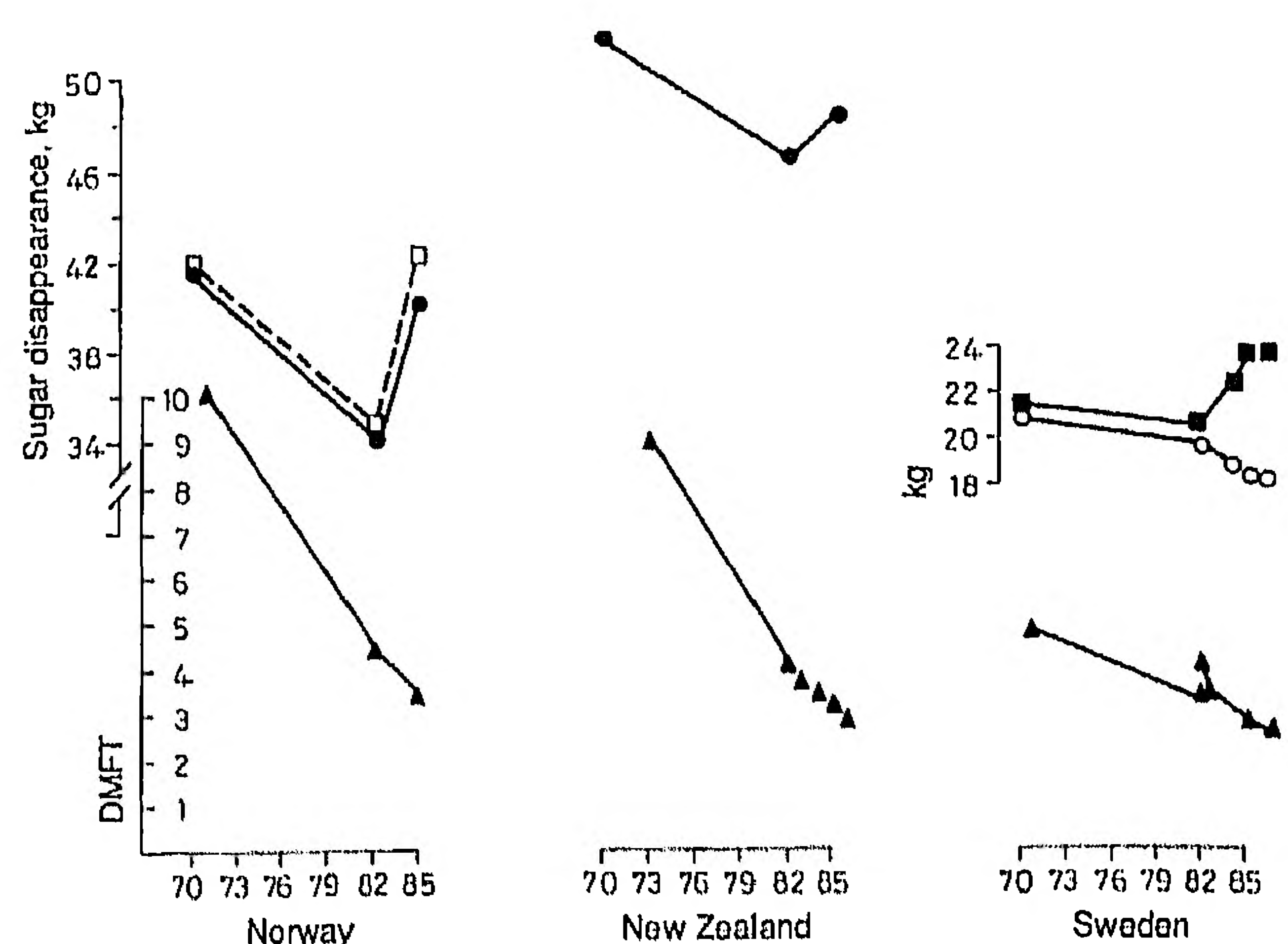


FIGURE 4. Average decayed, missing, and filled teeth (DMFT) in 12-y-old children (\blacktriangle) and per capita sugar disappearance in Norway, New Zealand, and Sweden between 1970 and 1985. Norway: \square , sugar data, uncorrected; \bullet , data corrected for import and export. New Zealand: \bullet , sugar data, corrected. Sweden: \blacksquare , sugar incorporated in industrial products; \circ , sugar for household and other use. From König (33).

and average sugar consumption. Nevertheless, we must remember to consider high-risk populations in developing countries, subpopulations (mostly ethnic minority groups) even in these low-caries countries, and individuals exposed to very high frequency of sugar contacts, eg, babies with continuous availability of carbohydrate-containing solutions in feeding bottles, which leads to dental decay.

Conclusion regarding sugar and caries risk

Oral health preventive programs have to go beyond focusing solely on the relation between sugar intake and dental caries and begin to pay increased attention to other factors. Among these are oral hygiene procedures, including fluoride delivery (contained in toothpaste, but also in foods such as tea or water); bacterial components of plaque (particularly 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; type of preventive and restorative care; immunologic response of the individual; and nutritional and dietary practices. There are many ways 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 other preventive measures, especially improvement of oral hygiene and use of fluoride dentifrices, have been found to be more acceptable and more effective than dietary restrictions in decreasing caries prevalence. A rational and judicious use of available information will provide an effective and acceptable approach to the eradication of dental caries in populations from both developed and developing countries around the world.

PERIODONTAL HEALTH

Maintenance of periodontal health is achieved by preventing chronic inflammation of the gingiva, the periodontal connective tissues, and the supporting alveolar bone.

Nutritional factors

One of the oldest observations on nutrition and periodontal health is James Lind's account of scurvy in the first controlled therapeutic trial conducted in 1747; he experimented on sailors suffering from general weakness and putrid periodontal inflammation on board the *Salisbury*. He subjected the sailors to different treatments but the only effective treatment was the consumption of oranges and lemons. This observation strongly biased investigators for > 200 y because of the striking evidence that periodontal disease was a systemic disease caused by nutritional deficiencies. Before Loe et al (38) published their work on the bacterial causation of gingivitis in 1965, nutritional and other systemic factors were assumed to be virtually exclusively responsible for periodontal diseases, even though it had become clear that vitamin C deficiency could only explain a small segment of the problem.

Nonnutritional factors

The view that systemic nutritional effects during development not only of teeth but also of the supporting periodontal structures have limited importance is rather recent. This explains why research in the 1950s was still directed toward the detection of nutritional and metabolic variables that might be associated with periodontal health and disease. Extensive epidemiologic studies on 21 559 persons in eight areas around the world were carried out by a multidisciplinary research team of nutritionists, biochemists, physicians, and dentists (39). The dentists fortunately included among the items to be studied local factors such as status of oral hygiene. The results reported in 1963 were unexpected. The group found periodontitis to be strongly dependent on age in all the populations studied, the severity increasing with age but much more closely associated with poor oral hygiene: 12% of the variation could be explained by age, 66% by poor oral hygiene. In contrast, the influence of the only nutritional influence detected, deficiency of vitamin A, was negligible: 1% of the total variation. Since then, the local bacterial causation of periodontal disease has been generally accepted. This led Rugg-Gunn (40), in his monograph *Diet and Dental Health*, to conclude that "by far the most important way of maintaining periodontal health in humans is regular, thorough, physical removal of dental plaque with a satisfactory toothbrush."

There is also new understanding of cellular and molecular effects of malnutrition on the immune response (41). Emphasis is not on the etiologic role, but rather on the role of specific nutrients, particularly those with antioxidant properties and those involved in tissue repair in the maintenance of periodontal tissues challenged by pathogenic bacteria.

There is some evidence from animal experiments that sugars as local dietary factors can negatively influence periodontal health, although indirectly: frequent eating of sugars stimulates the energy metabolism of plaque bacteria and results in copious formation of extracellular polysaccharides that increase the volume of plaque (42, 43). This can secondarily result in differentiation of the plaque organisms and development of an unfavorable subgingival flora. Obviously this applies only to animals. Regular removal of plaque by brushing the teeth, which is necessary to maintain periodontal health, eliminates this indirect risk. The same considerations are valid regarding the role of chewing soft, noncleansing food that has a long

retention time in the oral cavity compared with firm, fibrous, cleansing food. Chewing the toughest food is not sufficient to clean teeth completely, but complete lack of self-cleansing mastication can be compensated for by regular tooth brushing (44).

Conclusions regarding sugar and periodontal health

Periodontal diseases are caused by local inflammatory irritation caused by overgrowth and differentiation of subgingival dental plaque and not by systemic nutritional deficiencies or imbalances. Therefore, the principal rational method of prevention is regular tooth brushing and not dietary measures. There should be further research to explore the role of nutrients in the repair and healing of gingival and periodontal tissues as a way of reducing the prevalence of this disease. ■

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COMMENTARY

Kingman: Could you expand on the mechanism of action of fluoride in protecting teeth from decay?

Authors' reply: The oldest explanation is based on the observation that tooth apatite becomes more acid-resistant when optimum concentrations of fluoride prevail during tooth formation. During the past 10 y, evidence has been accumulating that frequent (preferably daily) local availability of fluoride after eruption of teeth is much more important than preeruptive incorporation. The mechanism of the local posteruptive effect is a significant enhancement of remineralization of tooth enamel, which is essential because of the frequent daily presence of demineralizing acids in the mouth (1). A third mechanism, inhibition of bacterial acid formation by fluoride, seems to play a minor role.

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Harland: Several observations have suggested that fruit consumption may have beneficial effects on periodontal health but that frequent consumption of fruit may result in an increased risk of caries development.

Authors' reply: Chewing of fruit only partially cleans the teeth and cannot replace brushing, so the periodontal benefit is limited. On the other hand, the same authors have shown that the (frequent) ad libitum consumption of fruit by fruit pickers resulted in the development of markedly higher numbers of carious lesions compared with comparable groups harvesting grains (1, 2).

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Saldanha: For children in low-income families in developing and developed countries who may not or do not have access to a dentist, fluoridated toothpaste, or fluoridated water, should sugary foods in the diet be restricted?

Authors' reply: Observations of many populations with widely varied cultures, eating habits, and socioeconomic status have shown that advice to reduce the intake of sugars is usually not accepted (1). In contrast, health education (especially by school health education programs starting as early as kindergarten age) results in, among other favorable effects, a high rate

of adoption of the habit of tooth brushing or the use of chewing sticks. Teaching of oral hygiene should be combined with making children aware of the great importance of hygiene in general as the only means of avoiding the hazards of infectious diseases.

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Sigman-Grant: Has there been any evidence of predisposing genetic factors?

Authors' reply: The only predisposing genetic factors that have a measurable influence on caries activity are the morphology of the teeth (retentive fissures) and crowding of big teeth in small jaws. Both conditions increase retention of bacterial plaque and food residues and render cleansing difficult.

Several participants pointed out that there were differences in fermentability of various carbohydrates in vitro.

Authors' reply: In dental plaque in the oral cavity, the multitude of bacterial species present provides a very large spectrum of hydrolyzing and glycolytic enzymes. Concomitantly with salivary amylases, they provoke acid formation on the tooth surface within minutes. Telemetry of pH changes after subjects have been rinsing with solutions of carbohydrate suspensions have shown the following (1): the acidogenicity of heated starches that are present in many kinds of food is nearly

the same as that of sugars, which explains their caries-promoting effects and, of the sugars, only the less frequently occurring galactose and lactose give rise to less acid than do other monosaccharides and sucrose.

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Edmondson: For individuals who practice reasonably good oral hygiene (brushing twice a day with a fluoride toothpaste), how much carbohydrate intake is safe for remaining free of caries?

Authors' reply: In populations with high percentages of caries-free adolescents [30% in Michigan (1) and 60% in The Hague (2)], parallel studies of eating habits show that up to six or seven reported intakes of food containing carbohydrates or sugars is compatible with a low caries risk, probably because of the availability of fluoride. Studies in vitro corroborate this (ten Cate and Duijsters, above). In any case, regular tooth brushing is a prerequisite.

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