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THE ROLE OF DIETARY CARBOHYDRATES IN DENTAL CARIES

by

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B.A.E., Wayne State College-Wayne, NE, 1973

A MASTER'S REPORT

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Foods and Nutrition

KANSAS STATE UNIVERSITY
Manhattan, Kansas

1977

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INTRODUCTION

Dental caries is one of the most prevalent diseases affecting mankind today. Heredity and nutrition influence the resistance of the teeth to dental decay throughout life.

During the past century extensive research has been conducted for the prevention of dental caries. Studies have shown that carbohydrates, principally sucrose, must be present in the crevices of the teeth for caries-producing organisms to grow (1).

Practical dietary advice on caries prevention is complicated by the introduction of a great variety of new foods in recent decades. Eating patterns have changed during the past fifty years to include frequent eating of snack foods, usually containing sugar, between meals. There is high consumption of refined carbohydrates, particularly refined wheat flours and refined sugars.

Research has indicated that it is not the increased consumption of carbohydrates in the diet alone causing the larger number of dental caries. The physical form in which they are eaten, other ingredients of the food with which they are compounded, the amount eaten, and the frequency with which they are eaten affect the susceptibility of the teeth to dental caries.

The purpose of this paper is to review the incidence of dental caries, to discuss its causes and to review the methods of prevention. The research concerning the relationship between dietary carbohydrates and dental decay is investigated thoroughly.

EPIDEMIOLOGY OF DENTAL CARIES

Dental caries is found rarely in ancient skulls. It is considered a modern disease that increased with the development and adoption of civilized and sophisticated life styles that was caused, primarily, by increased consumption of refined foods and changed dietary habits to meet the demands of increased numbers of people in today's affluent society (3).

In some areas of the world such as India, Africa and Indo-China, dental caries still is considered a rare disease. However, in areas such as North and South America and Europe, 98% of the population have experienced some dental decay during their lifetime. Data from a dental caries survey (4) of groups of 20- to 24-year-old civilians from different countries throughout the world show that the greater prevalence of dental caries is in the Americas (figure 1).

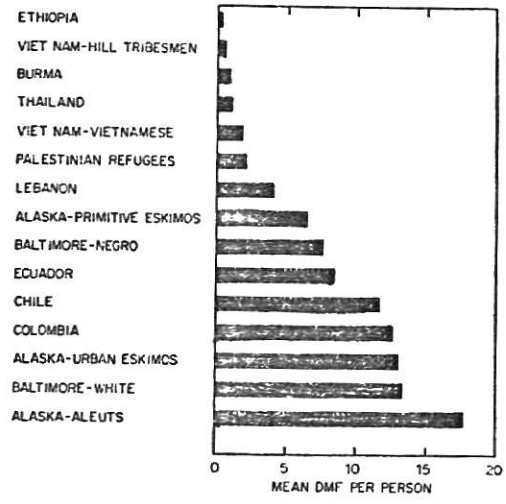

Fig. 1. Mean decayed, missing and filled teeth (DMF) per person for civilian groups aged 20-24 years.

Refined carbohydrate intake is correlated directly with increased prevalence of dental caries, as shown by Russell (5) in a dental survey (table 1).

TABLE 1

Dental caries prevalance correlated with sugar consumption in 1963

	Dental caries prevalence	Sugar consumption
Far East and Ethiopia	lowest	6-16 kg/person/year
Near East (Lebanon)	intermediate	13-19 kg/person/year
United States, Central, and South America	highest	23-44 kg/person/year

Apparently, it is not increased ingestion of complex carbohydrates, such as starches, that increase dental caries incidence, but rather an increase in fermentable sugars. For example, the diets of the people in Ethiopia, Far East and Near East include all starch foods of the cereal type (wheat, corn, maize, rice, teff) and of the root type (cassava, yam), but the consumption of sugar and sugar products is low.

In the United States, dental caries is considered a native disease (6). Several independent dental caries surveys over a 100-year period have shown repeatedly that natives of the New England and northwestern areas of the country experience about twice as many dental caries as the natives of the south central states (6). There are several possible explanations for this observation. The presence of natural fluorides in the water supplies used by the natives of the south central states, no

doubt, accounts for a great part of this difference. However, there must be additional factors. Other trace elements, that exert cariogenic activity, may be present in the soils and foods of the New England and northwestern areas, or environmental factors (7), including sunshine, humidity and temperature, may influence regional food habits such as the amount of sugar-sweetened soft drinks that are consumed.

MULTIFACTORAL AND COMPLEX NATURE OF DENTAL CARIES ETIOLOGY

Dental caries is prevalent in the young and is the principal cause of tooth mortality up to the age of 35 years. Dental decay is a progressive, destructive process involving the loss of tooth enamel, dentin and cementum. In the process bacterial enzymes, within the dental plaque, ferment carbohydrates, primarily sucrose, which results in the production of organic acids. Those acids, chiefly lactic acid, are produced by certain microorganisms that live on the teeth. Lactic acid is produced from sugar and starches ingested as food and drink. Organic acids diffuse out of the plaque, attack the enamel tooth surface and initiate decalcification of the enamel and dentin. The cariogenic bacteria then have access to the dentin and cause protein degradation of the cementum resulting in cavitation (3, 8, 9, 10, 11).

Dental decay is fundamentally a dieto-bacterial disease that is influenced by host, agent and environmental mechanisms that characterize all human diseases. In this disease, the tooth is the host factor and its resistance to decay depends on the stability of its apatite lattice.

Adhering to the surface of the tooth is dental plaque, the agent factor. Dental plaque can be removed only by mechanical cleansing. The bacteria in the dental plaque grow by rapid production and become virulent only if there is a diet substrate rich in sucrose and low in protein. A low salivary flow also can contribute significantly to increasing the rapidity and extent of the caries process. Thus, it is the combination of (a) vulnerability of the tooth, (b) virulence of the bacteria, (c) a nutrient substrate suitable for bacterial proliferation, and (d) the chemical and physical properties of the saliva that influence the initiation, development and extent of the dental decay process. Considerations that dental clinicians must deal with if they are to prevent or control dental decay are listed in table 2. It is apparent that dental caries is not a simple disease (3, 12).

TABLE 2

Procedures necessary to prevent and control dental caries

Tooth	1. Improve quality and structure
	2. Increase resistance of enamel surface
Bacteria	3. Decrease dental plaque formation
	4. Interfere with bacterial enzyme activity
	5. Remove dental plaque mechanically
Saliva	6. Stimulate flow rate
	7. Increase ability to neutralize acid
	8. Increase remineralization capacity
Food	9. Decrease sucrose intake
	10. Decrease frequency of eating
	11. Increase oral clearance
	A. Less sticky
	B. More firm and detersive
	12. Improve food quality and food practices

Nature of the Tooth Surface

The properties of the tooth, including its composition, structure, and morphology, as well as its position in relation to tongue, cheeks, and other teeth contribute to its susceptibility or resistance to caries (13). The factors that influence the resistance of the host to dental caries operate largely in two ways: first, through their influence on the structure of the dental tissues and secondly, through increasing or decreasing the stagnation areas around the teeth (14).

Finn et al. (12) pointed out that heredity plays a part in caries resistance. The morphology or shape of the tooth is governed by genetic factors. Of major importance are the pits and fissures, defects of the enamel, which most frequently occur within the grooves on the biting surfaces of the teeth. Because of the narrow dimension of those faults, food debris can become impacted and stagnate within those confined areas, producing carious lesions. Those teeth are highly susceptible to decay in contrast to those that do not contain pits and fissures and that have flattened cusps (15). If teeth are crooked and out of alignment or lack tight contact areas with adjacent teeth, they may have large areas of food stagnation.

Before a tooth erupts and after eruption, each tooth continues to increase in hardness through a process of maturation or increased mineralization (16). Prenatal and pre-eruptive maturation takes place through the blood stream that nourishes the developing tooth. Posteruptive maturation occurs locally within the oral cavity and is derived from the foods that we eat and from the saliva. The longer the teeth remain in

the mouth, the harder and better calcified the surface of the enamel becomes. This is one reason why decay activity decreases as one advances in age, other conditions being equal.

In man, the 20 deciduous teeth begin to mineralize between four and six months in utero. Mineralization of the crowns of the incisors is completed by the first three months of life and of the canines and molars before the first year.

Of the permanent teeth, the first molars begin to mineralize at birth and mineralization of the crown is completed during the third year; in the incisors and canines, mineralization begins during the first year and is completed by the age of seven years. Mineralization of the premolars and second molars begins during the second year and is completed by the age of eight years (17).

Unlike bone, the hard substance of tooth does not undergo metabolic changes once it is formed. Bone is continually being resorbed and rebuilt throughout life, and early deficiencies caused by shortage of calcium or other substances may be corrected if the diet is improved. However, teeth will retain formative defects throughout life and it is essential, therefore, that a completely adequate diet should be available during the fetal period and the first eight years of life if the crowns of the teeth are to be well-formed and well-calcified. The only teeth that are likely to be influenced by systemic physiological factors beyond the age of eight are the third molars (wisdom teeth), whose mineralization may not be completed until the age of 20-25 years. Calcium, phosphorus, vitamin D, and many other minerals are essential to tooth formation (12, 17). Fluorides hasten tooth maturation and incorporate within the enamel elements that

would decrease its solubility in acid. Therefore, a more caries-resistant tooth is formed. For this reason, there is a great need for continued effort to fluoridate communal water supplies. In areas where communal water supplies do not exist, school water systems can be fluoridated. Another method suggested for adding fluorides is to fortify foods such as salt, milk, and sugar (12). In addition, fluoride solutions or gels can be applied locally to the teeth (18). Human studies have shown the benefits of direct topical fluoride applications by mouthrinses, dentifrices, and tablets. The effectiveness of these applications is not nearly as great as that obtained from water fluoridation.

The Composition and Metabolism of the Plaque

Dental plaque collects principally on those parts of the teeth closest to the gum margin and on those sites of the tooth surface that are the least self-cleaning; for example, in the pits and fissures of the teeth. These areas of the tooth that are least disturbed will acquire the most plaque.

Plaque consists mainly of bacteria of several different species embodied in a sticky gelatinous polysaccharide matrix dependent on the diet of the host. The carbohydrates are broken down by *Streptococci* and *Lactobacilli* in the plaque to produce acids that attack the underlying tooth substance. The dental enamel covering the crown of the tooth consists almost entirely of calcium salts, and the dissolution of these salts by acids from the plaque is the first stage of dental caries. If the amount of acid formed in the plaque is small and produced over a short span of time, there will be no damage to the tooth, probably, since

the buffering power of the saliva may be able to neutralize the acid. Continual renewal of the carbohydrates in the plaque by eating sweets and carbohydrate snacks between meals results in a persistently acid reaction, and the tooth surface is attacked rapidly, especially when the carbohydrate is sucrose (19).

Although various microorganisms are present in the dental plaque, it has been observed that certain specific strains are more decay-producing than others. Irrefutable proof of this came with the advent of germfree experimental animals. These animals were born and allowed to live in a completely germfree environment. Germfree rats were fed caries-producing diets containing large amounts of sugar with no resulting decay. When various specific microorganisms were introduced, decay developed. Observations indicated that certain Streptococci (S. mutans) are of prime importance in the development of caries, although the Lactobacilli (L. casei and L. acidophilus) or other microorganisms may be involved (20, 21).

The processes that cause the deposition of plaque on the tooth surface are known only partially. Plaque appears to begin as a deposition of salivary mucoid on the enamel surface. Bacteria and food particles adhere to this layer of mucoid. The production of acid in situ will cause further deposition of mucoid from the saliva, more bacteria and food debris accumulate and so the thickness of the plaque increases (22).

The density of bacterial population is much greater in plaque than in saliva. Stephan (11) demonstrated that plaque from caries-resistant subjects, in general, accumulate less acid than that from caries-active

subjects. The formation of organic acids by the microorganisms in the plaque is an intracellular process. Therefore, suitable substrates must diffuse into the plaque, and be absorbed by the microorganisms in a form in which they can be metabolized to acidic end-products. Then, acid formed must pass out of the bacterial cell, diffuse through the plaque and reach the tooth surface in sufficient quantity to raise the hydrogen ion concentration at the enamel surface to the point where demineralization can occur. It is not only the formation of acid which is important, but the extent to which it accumulates at the tooth surface. It is the relation of dietary factors to this complex situation which is so important in caries resistance (22).

Metabolic Factors in Saliva

One of the characteristics of the oral cavity is the presence of saliva. It is within this environment that the forces that produce dental caries must function. The physical and chemical properties of the saliva affect caries susceptibility. It was observed (23, 24) that, in instances where salivary flow has been decreased greatly, as among individuals receiving radiation to the salivary glands for the treatment of tumors, widespread tooth decay is a common result.

Finn et al. (25) illustrated the importance of saliva in caries development in experimental studies with hamsters. The salivary glands were removed in one group and left functioning in another. The desalivated animals, when fed a cariogenic diet, showed an extreme breakdown of the teeth as compared to those with intact glands.

Two functions of salivary flow are to bathe the teeth and wash away food particles and soluble substances and to act as a lubricant. Salivary flow is greatest at mealtimes and only negligible during sleep. If diminished, salivary flow favors dental caries, then decay should be accelerated during sleep (12).

Hartles et al. (26) examined the properties of various salivas during those periods between meals when there was a minimal supply of substrate from external sources. The object of the investigation was to observe the magnitude and variation of oxygen consumption, by use of Warburg manometric technique, and the lactic acid production in the salivas of subjects with varying degrees of dental caries to see whether those factors bore any relation to the caries experience of the subjects. The subjects included one completely free of caries, one virtually free, two with slight caries activity and five with a considerable number of carious lesions. Oxygen consumption and lactic acid were measured using the "resting" saliva collected from the subjects in the early morning. The oxygen consumption and lactic acid production by the saliva incubated at 38^{o} in the presence and absence of glucose was determined. In addition, the lactic acid and total acid produced under anaerobic conditions were determined with and without the addition of glucose. Oxygen consumption of "resting" saliva was about one-third to one-half of that in the presence of glucose. In the absence of added glucose, lactic acid could not be detected. The amount of anaerobic metabolism of "resting" saliva was about one-quarter to one-half of that in the presence of glucose. No lactic acid could be demonstrated in the absence of

glucose, although total "acid-production" measured as carbon dioxide released from a bicarbonate buffer could be determined. Thus, in samples of "resting" saliva there was a measurable metabolic activity; no lactic acid production was detected under any conditions, but there was considerable anaerobic activity leading to the formation of some acidic end-products. There was no obvious relation between acid production and dental caries.

In the absence of an external supply of substrate, salivary flora depend on the salivary components for their supply of nutrients. Plaque organisms have the ability to accumulate food debris and also to synthesize polysaccharides for subsequent use when the external supply of substrate is minimal. Thus, there is intracellular storage of polysaccharide by organisms from the dental plaque. The proportion of microorganisms capable of synthesizing and storing polymers of the glycogenamylopectin type is higher in plaque from caries-active than in that from caries-inactive subjects (22). Organisms that have such stores will metabolize them to acids when the environment is depleted of carbohydrate, thereby maintaining a low plaque pH over a long period of time. However, the restriction of dietary carbohydrate produces a reduction in the number of polysaccharide-storing bacteria in dental plaque. Restoration of carbohydrate to the diet reverses the situation.

Certain chemical properties of saliva are important. Since acid production is associated with caries formation, the buffering capacity of the saliva can neutralize considerable acid before it can damage the

teeth. The buffering capacity of the saliva is attributed primarily to the presence of bicarbonate, phosphate, protein and mucin (12).

Jenkins (27) found bicarbonate to be the most effective buffer followed by phosphate. His results indicated that saliva from caries-resistant individuals has a higher buffering capacity than saliva from caries-active persons. That may be one explanation for the reduction in tooth decay in caries-resistant individuals.

Calcium and phosphorus are constituents of saliva, and it is believed, generally, that there is a continual exchange of those inorganic ions between the saliva and tooth surface. Therefore, both buffering capacity and inter-surface ion exchange should influence the resistance to decay (28).

It is evident that decreased salivation is conducive to increased caries and there is evidence to suggest that increased flow of saliva may be beneficial. The physical properties of saliva, its chemical composition, buffering capacity, rate of flow and immunological properties all contribute to the integrity of the tooth surface.

Composition of the Diet

Pre-eruptive dietary effects. Numerous laboratory animal studies have indicated impaired organic matrix formation and alterations in the structure and eruption timing of teeth as a consequence of malnutrition during the pre-eruptive phase of tooth development and maturation (29).

Teeth and oral structures develop in three stages: (1) a hyperplastic phase during which the organ grows by increasing the number of cells; (2) an intermediate period in which cell proliferation slowly diminishes

and cell growth begins; and (3) a hypertrophic phase in which tooth growth is almost entirely the result of increase in cell size (30). During those three stages of development, the cells are involved in intense metabolic activity. Therefore, the demand for nutrients during pregnancy is great.

Since teeth begin calcifying before birth and calcification is not completed until about 16 years of age, the effect of diet on tooth development must be considered both pre- and postnatally. In the first stage of tooth development a matrix of protein must be laid down. As the matrix becomes progressively calcified, vitamin D, calcium, phosphorus, and fluoride must be present to assure optimal calcification. In the prenatal period, the embryo must have sufficient nutrients available to synthesize its own protein, carbohydrate and fat. Thus, the availability of nutrients to the mother and their ability to cross the placenta into fetal circulation is important (30).

Navia (31) reported that changes in the organic matrix, form, morphology, and eruption patterns of teeth of adult female rats can be attributed directly to deficiencies of protein during critical periods of development. He found that when female rats were fed a low protein diet during pregnancy, their offspring grew slowly and were prone to develop decayed teeth later in life. Their molars were abnormally small and erupted later than usual. Also, submandibular salivary gland function was drastically impaired.

Post-eruptive dietary effects. During the post-eruptive life of the tooth, certain components of the diet have a marked effect on the tooth and play a significant role in the production of dental caries (32).

Dental caries in animals can be reduced virtually to nil when the animals are fed by stomach tube. Kite et al. (33) established the essentiality of an oral substrate by feeding two groups of rats a decay-producing diet; one group received the diet directly into the stomach by a stomach-tube, and the other by way of the mouth. The stomach tube fed group developed no caries, whereas the eating group showed considerable caries.

The substances that are most readily metabolized by the plaque organisms are those that will diffuse easily and can be absorbed by the plaque bacteria. The commonest of these are the simple soluble sugars: glucose, fructose, maltose, and sucrose, with smaller amounts of lactose. Starch, which is a large molecule, is unlikely to penetrate rapidly into the plaque and is not a suitable substrate for microorganisms. However, saliva contains a powerful amylase (ptyalin) which converts starch to maltose. Hence, starches can be broken down into soluble, diffusible sugars capable of being metabolized. That breakdown takes time; therefore, it is unlikely that the concentration of diffusible sugar achieved by amylolysis will be as great as that obtained by the presence of sugary foods (22).

The substrate on which the bacteria feed and multiply can come from sources other than sugar or starch; e.g., the amino acids, B-vitamins, and other nutrients that are essential for their survival. The microorganisms, therefore, do not necessarily need carbohydrates, although their major source of energy comes from sugars and starches taken into the oral cavity in the form of food and drink. The carbohydrate most commonly indicated is sucrose (12).

Nutrition can affect the buffering capacity of saliva, which in turn influences its cariogenic properties. A diet relatively low in protein and rich in sucrose promotes proliferation of cariogenic bacteria. Protein is important not only in the formation of the tooth structure and its ability to resist decay, but also in its regulation of bacterial growth (3, 9). Casein, the phosphoprotein in milk, when used as a source of protein in animal experiments, exerted an anti-caries effect (34). When a protein rich diet was fed to humans there was increased salivary flow that neutralized the acids produced in the dental plaque, and thus, reduced caries development.

Sterky et al. (35) observed a group of adolescent diabetics in which the diet contained a high level of protein and a low amount of refined carbohydrates such as sucrose. A low caries frequency was noted.

Fats, because of their ability to produce a protective oily film on the surface of the teeth, serve as a barrier to acid penetration into the enamel (34). Hartles (22) showed that a significant fall in dental caries in rats resulted when 5% of groundnut oil was incorporated in a high-sucrose diet.

Calcium, phosphorus, fluoride, vitamin A, vitamin D, and ascorbic acid are all important nutrients for the post-eruptive development of teeth and gums (32). Lack of vitamin A during tooth development results in an uneven protective layer of enamel. Then, fissures will be present and the teeth will tend to decay. Deficiency of ascorbic acid results in abnormalities in the supporting tissues, dentin and the nonepithelial cement substances of the teeth.

The ill-effects of modern diet are seen constantly with almost 80% of all 5-year-olds suffering from dental caries and as many as 10% suffering from "rampant caries" in which all the teeth are destroyed. The modern diet of most children contains a high level of carbohydrate, which may lodge in the teeth and undergo rapid acid fermentation. There is a need for a diet that requires chewing such as coarsely ground wholemeal bread, fresh vegetables and fruits (36).

CARBOHYDRATES AND DENTAL CARIES

The two major sources of carbohydrate in contemporary Western diets are starches and sucrose in various forms. The information now available from human epidemiological studies, together with animal and laboratory experimentation of various dietary components, justifiably claims sucrose to be the major cariogenic food factor (37). A clear distinction in cariogenicity can be made between those two sources of carbohydrate. Starches of high molecular weight are not immediately available substrates for microorganisms; it is necessary that they be hydrolyzed to smaller units before they can enter plaque. That can be done, but it takes time. The small sucrose molecule provides an immediate source of substrate for the plaque bacteria. The general statement that polysaccharides are much less cariogenic than mono- and disaccharides can be said without dispute.

Although the proportion of total carbohydrate in the Western diet is lower than at the turn of the century, the percentage of total carbohydrate provided by sugars has increased continuously (38). With decreased consumption of grain products, there has been a corresponding decline in the proportion of starch in the diet. Data in figure 2 show that early in

the century, sugars accounted for about one-third of the total carbohydrate. By the late 1950s, sugars and starch contributed about equally to the total amount. In 1972 sugars made up a little more than half of the carbohydrate.

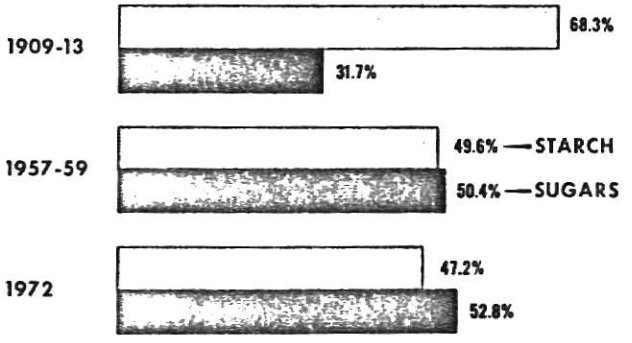

Fig. 2. Proportion of carbohydrate in the national food supply provided by starch and sugars, selected periods (38).

Comparison of the Cariogenicity of the Various Carbohydrates

Both in vitro and in vivo studies, in which the relative cariogenicity of different carbohydrates (sucrose, maltose, lactose, glucose, fructose, and starch) were compared, showed that sucrose was the most cariogenic and starch the least (39). Figure 3 illustrates the cariogenicity of different carbohydrates as determined in animal feeding experiments and in human studies.

Since hereditary fructose intolerance (HFI) was recognized as a new disease, in 1956, it has been possible to study dental decay in persons who live virtually without ever consuming fructose or sucrose. The disease, an inborn error of metabolism, is inherited as a recessive trait.

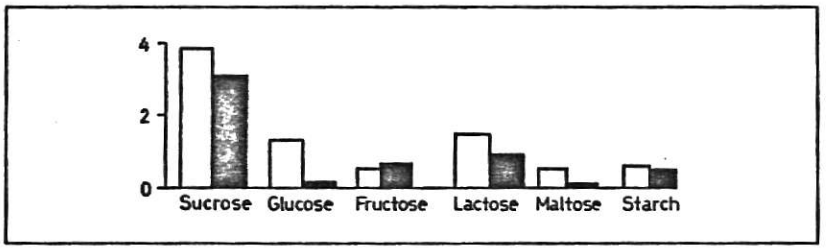

Fig. 3. Cariogenicity of different carbohydrates. □ = fissure lesions, ■ = buccal lesions. Guggenheim et al. (39).

Biochemically, a liver enzyme, fructose-1-phosphate aldolase, is missing in these patients. Any foodstuffs containing sucrose or fructose produce severe nausea, even when consumed in small amounts. Also, some vegetables, for example tomatoes and carrots, cause disturbances. Starchy foods such as bread, potatoes, corn, rice and pastry are well tolerated because they do not contain fructose (40).

Dental decay is uncommon in patients who suffer from HFI, and therefore avoid sugar, but freely eat starchy foods (37). Sullivan (41) reported complete freedom from caries in two sisters, who did not tolerate sugar, and their basal diet during early childhood consisted of skim milk and soya bean flour with added vitamins.

Carbohydrate in Vitro Tests

Although in vitro tests conducted by early investigators contributed to the establishment of the importance of carbohydrates in caries causation, researchers tended to discount the value of in vitro tests in favor of findings from rodent experiments. Presently, the findings from early

animal studies are considered to have little relevance to the cariogenicity of foods in man, and attention has been redirected to in vitro studies (2). Currently, in vitro methods include measurements of bacterial acid production, enamel demineralization, food retention in the mouth after eating, and acid formed in plaques on the tooth surface.

Although caries-like destruction of enamel has been produced in an apparatus designed to duplicate oral conditions, little information on the cariogenicity of carbohydrates has been obtained by this method. In reviewing in vitro studies, Bibby (42) concluded that no single test method can give a complete answer to the cariogenicity of foods. However, results obtained by using several test methods may be meaningful. Therefore, only the results from some of the recent in vitro tests will be reviewed.

Acid production. Miller (43) differentiated between the caries-producing capacity of foods on the basis of the amount of acid they formed when incubated in saliva. He concluded that bread and potatoes were more cariogenic than sugar because they formed more acid. Mundorff and Bibby (44) pointed out errors in experimental procedures used by Miller and disagreed with his conclusions regarding the cariogenicity of foods on the basis of acid production. Using a procedure that neutralized accumulating acid before it inhibited glycolytic enzymes, Mundorff and Bibby (44) showed that candies that yielded up to 20% solution of sugar in the mouth partially inhibited acid production. Similarly, the acidity of foods prevented fermentation, and thus no new acid was formed on incubation of most sour-fruit flavored candies and beverages in saliva. However, the inherent acid of such items produced enamel destruction in the mouth. Bibby and Mundorff (44) also

pointed out that flavor and other components of foods can modify, both quantitatively and qualitatively, the acids produced by fermenting foods.

Enamel demineralization. Although it appears that the amount of enamel that would be destroyed by a fermenting food would be proportional to the acid produced, that is not true. It has been shown experimentally (27, 45, 46) that there is no parallel between the sugar content of foods and the amount of enamel it will destroy in laboratory tests. Measurements of pH and titratable acidity indicated that fermenting foods produce different acidities that vary individually, in their ability to dissolve enamel (47). In addition, many foods, because of their mineral or protein content, buffer acids formed during fermentation, and thus reduce the effect of the acid on enamel. Furthermore, Weiss and Bibby (48) suggested that some foods contain enamel-protective factors, such as proteins, which are absorbed on the enamel, thereby making it more resistant to destruction by acid.

Food retention. The amount of food retained in the mouth after eating has not been used, widely, as an index of cariogenicity with in vitro tests. Although different methods of study have been used, it is agreed (49, 50) that starchy foods persist about the teeth for longer periods than high sugar foods, and thereby increase sugar retention. Rowley et al. (51) showed that foods with high sugar content were removed more rapidly than those with less of such a freely soluble component. Thus, with sugary foods, retention was high immediately after eating, but it did not continue at that level unless the other food ingredients were of a retentive nature. Liquid foods are removed more rapidly than dry ones, and carbonation appears

to speed removal. Probably, salivary stimulation by acid or sugar content play a part in food clearance, but this has not been clearly established.

Plaque pH. Measurement of changes in plaque pH give information on the extent and duration of acid production from foods on the tooth surface and should provide direct indication of their cariogenicity. Unfortunately, the interpretation of plaque pH is difficult because of a lack of agreement in the findings obtained by different techniques for plaque pH measurement. The procedure used most frequently is the measurement of acid production in a suspension of plaque and substrate in saliva in vitro. This procedure shows that glucose and sucrose have the same effect on plaque acidity (52). Small pH depressions were caused by maltose and lactose, and raw starch resulted in minimal changes. However, if starch was cooked, it depressed pH (53, 54), and if it was used in association with sugar, such as in bread and cookies, starch lowered the plaque pH to the same extent as sucrose and maintained the depressed pH for a longer period than with raw starch (53). Few investigators have tested more than one or two types of sugars or snack foods. The most extensive study (54) showed that pH changes produced in plaques by food were influenced by the pH and buffering capacity of the foods as well as by their carbohydrate content and retention on the teeth. A clear relationship between plaque pH and the sugar content of foods was not apparent.

Carbohydrate in Vivo Tests

Animal studies. Since it was first demonstrated that coarse rice and corn could produce caries in rats, a number of experimental animal diets has been used to produce carious lesions in rats, hamsters, and cotton

rats (2). With improvements in study methods, aimed at producing more consistent results in caries studies, the validity of previous findings has been questioned. It has been concluded that findings on rodent caries are of little value unless the animals are in good health and both the amount of food eaten and the frequency of eating are controlled (2).

Navia et al. (55) demonstrated that the addition of as little as 5% of sugar or sugar alcohols to a starch-based diet will produce active caries. Presently, researchers agree that most findings regarding caries in rodent studies should not be regarded as having significance for man. Therefore, only a brief summary of some findings of animal studies that might be paralleled in human beings will be presented.

Although many contradictory results are found in animal studies, most findings have shown that sugar is more conducive to caries than starch (31, 56, 57). There is uncertainty as to whether sucrose is more conducive to caries than glucose (55). Findings indicate that equal amounts of sugar in liquid or gel form produce only a fraction of the caries they produce in dry diets (58, 59), and that additions of fat can reduce caries production (60, 61).

More recent work has demonstrated that the same total amount of food, fed in many small portions, will produce a greater number of caries than that fed in fewer larger portions (62). Gelatinized starches are much more cariogenic than raw starch (63).

Early caries studies with monkeys appeared to show promise of findings which would be more applicable to human beings than findings obtained with other animals. However, beyond indicating that diets containing sucrose

are cariogenic, the work with monkeys cast no new light on the cariogenicity of human foods (64).

Human studies. Ethical and practical considerations have limited the number of attempts to use specific carbohydrates or foods to produce caries in the human mouth. Von der Fehr and co-workers (65) showed that small lesions developed on the teeth of patients who rinsed their mouths nine times a day for 23 days with a 50% sucrose solution and omitted oral hygiene. Using enamel blocks that were covered by gauze to hold "plaque" on them, Keller et al. (66) found that glucose produced as much enamel demineralization as sucrose.

Since dental plaque on the surface of the enamel is the initiator of surface dental caries, the effect of carbohydrates on the metabolism of dental plaque bacteria is important. Carlsson et al. (67, 68) compared the effect of a sucrose rinse with a glucose rinse on dental plaque formation on the surface of a human cuspid tooth. When the plaque was harvested, the sucrose-coated surface showed twice as many organisms as the glucose-coated one. The type of organisms usually associated with dental caries are extracellular and intracellular polysaccharide storing Streptococci and Lactobacilli. These bacteria are not only acidogenic but the Streptococci store polysaccharides in the form of dextran, levan and glycogen. Those storage depots of carbohydrate in the dental plaque can be degraded to sugar and organic acids by the cariogenic bacteria and used for their metabolism even when dietary sucrose is not available (69). However, when the dietary sucrose is restricted for a few weeks, the numbers of polysaccharide storing cariogenic Streptococci are reduced significantly. This may explain why the child who is fed sweets early in

life is more susceptible to caries than the older child who ingests sugar after his teeth are completely matured and mineralized (70).

Grenby et al. (71) compared the effects on plaque of boiled sweets made from sucrose and sweets made from wheat-starch hydrolysates, mainly glucose. Equal groups of 18 students ate 454 grams of the samples of sweets in addition to their normal diet during three days, without brushing their teeth. The extent of the plaque covering the teeth, plaque carbohydrate and the carbohydrate:protein ratio were significantly greater after the sucrose sweets.

Lambrou (72) fitted each of four young adults with an acrylic mouth-guard that covered the teeth of the lower jaw. Enamel slabs from newly extracted bovine teeth were prepared, their initial hardness was measured and they were attached to the mouthguards with wax. The appliances were worn for several days to accumulate plaque; they were not brushed and were not worn during meals. The slabs were then covered with a small amount of the food to be tested, which was held in place with Dacron gauze fixed in position with wax. One group of slabs was left uncovered with no food applied (control). To other slabs, coffee, cookie (flour from 50% extraction, sugar, flavoring), white bread (flour from 70% extraction) or whole-wheat bread (flour from 90% extraction) were applied. Then, the mouthguards were worn for one or more days, except during meals, after which the hardness of the enamel slabs was measured again. According to the test results, white bread had almost the same cariogenic action as the sweet foods, toffee and cookie. However, the whole-wheat bread was less cariogenic than white bread.

In all of the test procedures, sucrose-containing items produced results that confirmed their ability to produce caries, but none of the findings pointed to a direct relationship between the amount of sugar in a food and its level of cariogenicity (72). The acid production, demineralization, and in vivo caries tests that were conducted with nonsucrose materials indicated caries could be produced in the absence of sucrose. The results obtained with flour-containing foods in the demineralization, clearance and plaque pH in vitro and in vivo caries experiments suggested that mixtures of flour and sugar may be particularly destructive to the teeth.

In summary, many food properties, other than their carbohydrate content, can influence the cariogenicity of foods. They include inhibitory levels of sugar or acid, the presence of acid buffers or enamel protective agents, flavor effects, and food texture (2).

MAJOR CARIOGENIC CARBOHYDRATE - SUCROSE

Sucrose is the major chemical food constituent responsible for the production of dental caries. The cariogenicity of sucrose is dependent on other factors including the physical form in which it is eaten, the other food ingredients with which it is compounded, the amount eaten and the frequency of eating (73, 74).

Clinical Observations on the Direct Relationship Between Caries and Carbohydrate Intake

Primitive diets versus modern diets. Although ancient man ate no refined carbohydrates, there is evidence that he was not entirely free of dental caries (75, 76). However, caries incidence was much lower than in present

man. That may have been attributable, partly, to the lack of refined sugar in the diet. Because of the coarse foods eaten by early man, there was a wearing away of the pits and fissures on the surfaces of the teeth. These are the areas of the teeth that are most susceptible to dental decay. Ancient man had well-formed dental arches and properly spaced teeth. In contrast, modern man, who strives for a desired cosmetic appearance, has crowded and unharmonious arches and improperly spaced teeth. That results in greater areas of food stagnation and caries development (3).

The first recorded observation that eating sweet foods might cause dental decay was made by Aristotle some 2,000 years ago, around 384-422 B.C. He observed that the eating of soft and sweet figs caused damage to teeth (3). Since that time, the natural diets of primitive man have been replaced by the softened and more refined diets of civilized man. The incidence of dental caries increased progressively with this change in dietary pattern. It is possible that environmental changes, the delay in maturation of teeth or other host factors may have had an influence on the increasing incidence of dental decay. However, the role of sucrose cannot be minimized (3, 12). Many primitive civilizations that have converted to refined type foods have been studied including the Bantu tribes of South Africa (77), the Eskimos in Greenland (78) and Alaska (79), and the Indians of James Bay in Northern Canada (80). The teeth of these people began deteriorating when the refined types of foods of civilized man were introduced into their diets (3). When the Tristau Da Cunha natives increased their sugar intakes from practically nothing to one pound per week, their incidence of dental caries increased by 50% (81).

War time population comparisons. Indirect evidence of a sucrose-dental caries interrelationship was obtained in studies conducted during the two world wars when most of the involved countries suffered from food shortages. The most renowned war time study was made by Toverud (82) on Norwegian children who, during World War II, were eating a diet low in sugar because of rationing (3). The main differences in food consumption and food habits of those children during the war as compared to before the war was as follows: increased consumption of fish and salted herring, vegetables, potatoes, cod liver oil, and high extraction flour; decreased consumption of refined and white flour, meat, fat, sugar and sugar sweets, fruits and reduced between-meal eating (83, 84).

Dental findings during and after World War II, as reported by Toverud (83), indicated a reduction of 35-60% in decayed, missing, filled (D.M.F.) teeth and of 60-80% in D.M.F. surfaces in Norwegian children 2 1/2-14 years of age. There was a time lag in caries development corresponding to the average time needed for lesions to reach the stage that they could be clinically diagnosed. In 12- and 13-year-old children, D.M.F. rates remained high during the first years of the war because of caries development before sugar restriction. Also decay rates were low for several years after a post-war increase in sugar consumption. That was caused, mainly, by the favorable conditions of early posteruptive maturation on the enamel surface.

Data in table 3 and figures 4 and 5, which were compiled from Toverud's 1961 paper, show that the caries decrease during and after World War II was caused by factors that were present during tooth formation.

TABLE 3

Number of DF surfaces (excluding occlusal) per 100 permanent molars in 1941, 1956, 1949. Exposure time for first molars in 7-year-old, and second molars in 13-year-old was from 1 to 3 years. Exposure time for first molars in 13-year-old was 7 to 9 years

	1941	1946	1949
UPPER JAW			
First molars, age 7	29.1	8.0	19.0
Second molars, age 13	17.8	3.2	12.5
First molars, age 13	136.8	115.6	75.7
LOWER JAR			
First molars, age 7	46.7	14.0	26.3
Second molars, age 13	35.5	7.2	19.7
First molars, age 13	105.3	67.8	60.3

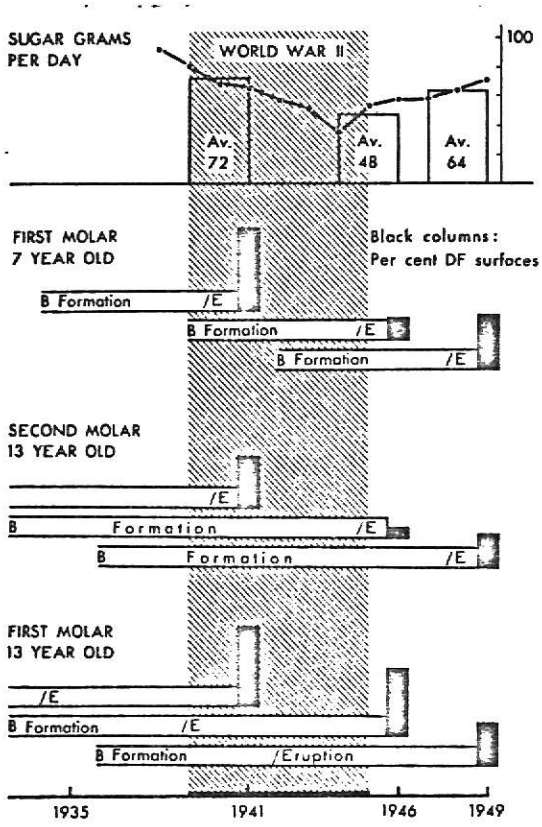

Fig. 4. Dental caries in upper molars as related to high versus low sugar intake in Norway: effects during tooth formation and after eruption. The upper part shows the yearly consumption of sugar (curve) from 1935 to 1949. Broad empty columns represent average consumption over the years 1939-1941, 1944-1946, and 1947-1949. Black columns show percentage of DF surfaces (excluding occlusal) in first and second molars of 7- and 13-year-old children (figures are given in Table 3). B = time of birth, E = period of eruption of the respective tooth.

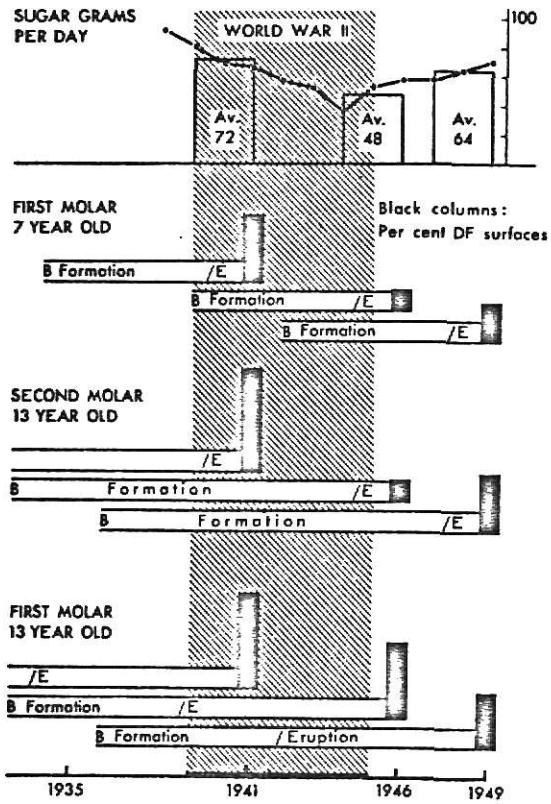

Fig. 5. Dental caries in lower molars as related to high versus low sugar intake: effects *during tooth* formation and after eruption. For explanation see Fig. 4. Data are given in Table 3.

From the figures, it is evident that the first molars in 7-year-old and the second molars in 13-year-old children showed a similar decrease in dental caries from 1941 to 1946. The low caries rates observed in 1946 in the second molars of the 13-year-old children were not caused by favorable influences of the war-time diet as those teeth were already formed when the war shortages became effective. As shown in the figures, the first molars, which were formed and mineralized during the sugar restrictive years, were not resistant to caries when increased sugar consumption resumed as indicated by the increased caries rates in 1949 (7-year-old children). In the first molars of the 13-year-old children, no detrimental effect of pre-war nutrition was found. Their low caries rates were attributed to favorable decreased sugar consumption influences during eruption. Toverud (83) found a close relationship between annual caries incidence and the sugar consumed in the same year or the two preceding years.

Takeuchi (85) conducted studies in a large population relating wartime sugar rationing to caries incidence. In Japan the sugar consumption dropped from approximately 15 kg per person per year to below 1 kg between 1943-1948. He reported that caries prevalence was lowest in the years following the period of lowest sugar consumption.

Other wartime population studies include those that were conducted in England, Japan and Norway (3). Results of those studies indicated that when sucrose was in short supply during the 1939-45 war, there was a reduction in caries prevalence. However, when sugar supplies were restored a few years after the war, there was a rapid rise in caries incidence (36).

Institutional studies. The Vipeholm Study conducted by Gustafsson et al. (86) in Sweden is the most extensive institutional study of the relationship between sugar intake and dental caries ever undertaken. Institutionalized patients with mental disorders were chosen because they were expected to be hospitalized for a long period of time. Following an initial dental examination to determine the suitability of subjects from both medical and dental standpoints, 436 adults were selected to participate in the study. The subjects underwent dental examinations at regular intervals and a medical examination at least once a year.

The initial stage of the investigation was conducted between the years of 1947 to 1949. During this preliminary period, referred to as Carbohydrate Study I, all patients in the institution were fed a basic diet, which contained a minimum amount of refined sugar, but it was not "sugar free" because of the natural sugar content of many foods.

In the second part of the study, Carbohydrate Study II (1949-1951), subjects were divided into four groups and fed diets that resembled those consumed in ordinary Swedish households, particularly in the amount of sugar eaten per day. With a few exceptions, the subjects were not allowed to eat between meals. The four groups were fed the following diets:

Group 1: A basic diet supplemented with fat to provide the recommended daily calorie allowance. No additional carbohydrate was fed. This was the Control Group.

Group 2: A basic diet with additional sugar (non-sticky form) in solution at meals. The amount of sucrose fed in this diet was higher than in any other diet. Observations made in this group indicated the relationship between total sugar intake and dental decay. This was designated as the Sucrose Group.

Group 3: A basic diet with additional sugar (sticky form) consumed at meals. Observations made in this group indicated the relationship between dental decay and the retention of sugar consumed in low concentrations at meals. This was designated as the Bread Group.

Group 4: A basic diet with additional sugar (sticky form) consumed between meals. Observations made in this group indicated the relationship between dental decay and sugar retention between meals, from preparations containing sugar in high concentrations. Group 4 was further subdivided into a Chocolate Group, Caramel Group, 8-Toffee Group and a 24-Toffee Group. The 8-Toffee Group received 8 sticky toffees between meals, whereas the 24-Toffee Group received 24 sticky toffees between meals daily.

Figure 6 shows the D.M.F. teeth per person as observed in the Vipeholm Institutional Study.

Results of the Vipeholm Study supported the following (86):

1. Sugar consumption can increase caries activities.

2. If the sugar is consumed in a sticky form which adheres to the teeth, the risk of sugar increasing caries activity is great.

3. If the sugar is consumed in a sticky form of high concentration between meals, the risk of sugar increasing caries activity is greatest.

4. Increases in caries activities under uniform experimental conditions varies from one person to another.

5. When sugar-rich foods favoring caries activity are withdrawn, the increased activity disappears.

6. Caries may continue to appear despite the avoidance of refined sugar, restriction of natural sugars and total dietary carbohydrates.

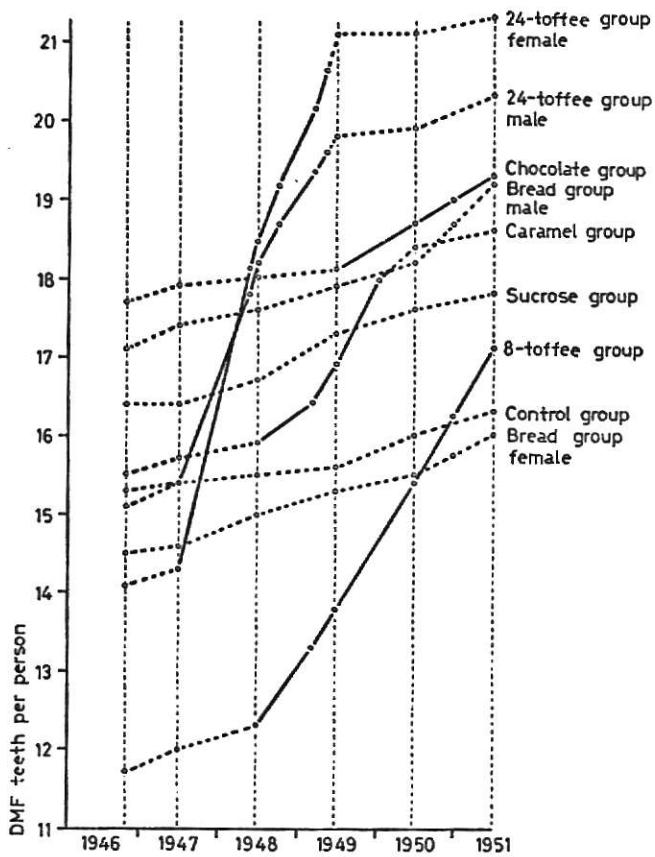

Fig. 6. Dental caries frequency 1946-1951. Sugar consumed at meals - - - -, sugar consumed both at and between meals _____.

Another important long-term institutional study was conducted at Hopewood House in New South Wales, Australia (87). That study extended over a 5-year period, 1957-1961, and included 81 children ranging from 4 to 9 years of age (12, 88). At Hopewood House, where the children were brought soon after birth, there was an unusually large proportion of uncooked food eaten. The diet consisted mainly of whole wheat grains, fresh and dried fruits, cooked and raw vegetables. Practically no meat, butter, eggs, cheese and milk were consumed, and honey and molasses were used as natural sweetening agents.

As shown in figure 7 (88), in an early dental survey of Hopewood House children in every age group, there appeared to be from 50 to 75% fewer caries than among children of similar ages in the state schools. However, the differences in dental caries incidence decreased with age. As the children reached the 13- to 15-year-old range, they began to leave Hopewood House for longer periods of time to attend secondary schools or to begin working. At that time they consumed high-sugar foods, white flour and other products that were available in the cities of Australia (89).

Boyd and Drain (90) summarized the observations made of institutionalized diabetic children, who were fed regulated diets. Those children showed low incidence of new dental caries, which was attributed to well-balanced diets, a minimum of refined sugar, and the discouragement of between-meal eating.

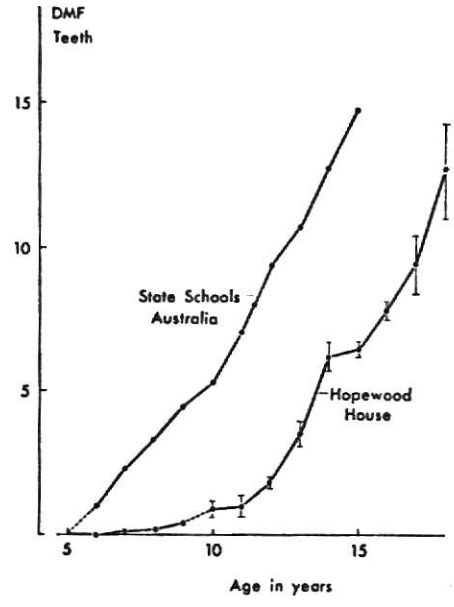

Fig. 7. Mean number of DMF teeth per child in State Schools of Australia and in children of Hopewood House (with standard errors of means). Harris (88).

Preschool and school population studies. Several studies have been conducted with preschool children comparing dental caries development with the use of the nursing bottle as a comforter at bedtime. Winter et al. (91) found rampant caries in 48 of 602 preschool children between one and four years of age from all social groups in a London borough. Caries were found, especially among children who had been bottle-fed on going to sleep. Also, prolonged bottle feeding and the use of comforters were associated with increased incidence of dental caries (36, 91). Often, comforters are dipped in sugary solutions or many are filled with a syrupy base containing vitamin C. The use of comforters exposes the teeth, constantly, to high concentrations of sugar, which is broken down to acids. It was found that these children

have a rapid breakdown of teeth, especially the upper front teeth (12). Winter et al. (91) found a relationship between the use of comforters and social class; the higher the social class, the less often comforters were used.

Palmer (92) observed the dietary habits at bedtime of British children 7-11 years old, and found that children who ate or drank food just before sleep had approximately 16% more decayed teeth than children who had cleaner mouths on retiring. These observations indicate that when a comforter is used or when food or drink is consumed prior to sleep, when the flow of saliva is negligible, carious activity will result.

Weiss and Trithart (93) conducted a study, which included 783 preschool children attending a health conference in West Tennessee to determine types of between-meal snacks and the relationship between the frequency of between meal eating and caries experience. The 24-hour dietary recall and the decayed, teeth to be extracted, or filled (D.E.F.) methods were used to determine the correlation. The most frequent between meal items consumed were gum, candy, soft drinks, pastries, and ice cream. The average frequency of eating between meals was 1.75 items per day, and the average number of D.E.F. teeth was 5.88.

The authors (93) found a direct and consistent relationship between caries experience and the frequency of eating items of high sugar content or high degree of adhesiveness between meals. As the frequency of eating those items between meals increased, there was a corresponding increase in the number of D.E.F. teeth per child.

In 1938 Read and Knowles (94) observed 3,000 children between the ages of 6 and 13. They found that only 6% of the 6-year-old children and 4% of the 13-year-olds were completely free of dental decay. The group of children who were caries-free ate well-balanced diets with adequate protein, fat, and vegetables. They consumed the recommended daily allowances of milk and fresh fruit, their meals were regular, and they did not eat between meals. Much of the food consumed by this group of children was hard chewing food. The carious group of children tended to consume insufficient amounts of vegetables and fresh fruits, and they ate significant amounts of sweets and fermentable carbohydrates daily. Data indicated that freedom from dental decay was associated with consumption of well-balanced diets; whereas, caries activity was related to consumption of inadequate diets that were high in fermentable carbohydrates.

In regard to the relationship between gum chewing and the number of D.E.F. teeth, several studies have shown no increase in dental caries from chewing sugar gum (95, 96, 97). Volker (97) studied the effect of chewing gum under carefully controlled conditions and found that gum chewing did not increase dental caries, but rather gum removed an average of 80% of the residual oral debris. Although chewing gum does contain sucrose and glucose, the increased salivary flow clears those materials from the mouth more rapidly than when they are introduced with other confections and foods. After the initial introduction of the chewing gum, the detergency action of chewing the gum tends to remove mechanically, the residual foodstuffs that might cause the production of dental caries.

Several studies have been conducted with children to determine the effect of sugar-coated cereals on the production of dental decay. During a two-year period, Glass and Fleisch (98) analyzed the consumption of regular and presweetened ready-to-eat cereals by 979 children aged 7-11 years to determine the relationship between caries incidence and cereal intake. All of the subjects lived in eastern Massachusetts communities where the amounts of fluoride in the drinking water were not significant. The children were encouraged to eat as much cereal as they wanted, but were requested to eat at least one serving of cereal daily. The average cereal consumption by type of cereal was estimated for each participant, and the children were then categorized as low, medium, or high consumers of cereals. Analysis of the data showed no association between caries incidence and cereal consumption.

Rowe et al. (99) studied the effects of consumption of ready-to-eat cereals on dental caries in 375 adolescent children for three years. Of those 375 children, 302 consumed ready-to-eat breakfast cereals regularly, whereas, 73 did not. Statistical analysis of the data showed no difference between the two groups in caries incidence. Other foods consumed at breakfast by the subjects were not reported in this study.

The cereal studies suggest that eating sucrose during a meal with other foods does not increase dental caries. This may be attributed to the increased salivary flow at mealtime and swallowing the food before all sweetness is extracted from it. Usually, sweets consumed between meals are eaten more slowly than those eaten with a meal, and therefore there is longer contact with the teeth (12). The consistency of the food

plus accompanying foods, such as milk, may affect the cariogenicity of sucrose (98).

Effects of Physical Form

Although the coarseness of foods is believed to have a cleansing effect on smooth surfaces of the teeth by a mechanical action during mastication and by increasing salivary flow to flush away undesirable material in the dental plaque, few experiments have produced data to support this belief (17). Slack and Martin (100) studied about 200 children for two years. The experimental results indicated that the group of children who ate hard apples after meals did not show a statistically significant decrease in caries incidence over the control group who did not.

Hartles and Leach (17) found that in subjects chewing excessive amounts of hard apples or raw carrots over a one hour period, plaque was removed from the easily accessible upper two-thirds of the teeth. There was no effect on plaque removal from the gingival margins. The cleaned surfaces normally are free from dental caries.

It has been reported that apples (101) help remove food particles and clear the mouth of sugar. Apples, carrots, celery or other fibrous foods may help curb the appetite for sweets and confections, and in this way could reduce caries incidence. However, if an apple is eaten before retiring to bed, the plaque pH is lowered for three hours afterwards, and thus promotes dental decay.

Review of primitive diets (77, 78, 79, 80) suggested that the hard and coarse foods of the diet wore away the pits and fissures of the teeth

making them shallower and more resistant to dental decay. In the meantime, with the consumption of a natural diet of hard and coarse foods, dental plaque formation was reduced. From studies conducted by Newman (192) on teeth from persons whose diet was typically modern, it was concluded that plaque formation on tooth surfaces depended mainly on dietary texture.

Another factor that must be taken into consideration is the retention of food on the tooth surface. The Vipeholm Study conducted in Sweden by Gustafsson et al. (86) showed a rise in dental caries activity with the addition of sucrose in the form of sticky foods such as toffee between meals. Dunning and Hodge (103) demonstrated that when sugar sweetened chocolate flavoring was added to fluid whole milk and consumed by school-age children there was no statistically significant increase in caries incidence. Liquid foods in comparison to sticky or retentive foods tend to have lower cariogenicity (3, 6).

Effects of Frequency of Eating

The study conducted by Toverud (83) of the eating habits of people in Norway before, during and after World War II and the Vipeholm Study (86) conducted with mental patients who were fed controlled diets, indicated that between-meal eating of sweets was a major factor in the development of dental caries. In the Vipeholm Study it was shown that when sweets, particularly sticky or hard-sucking types, were eaten between meals from 8 to 24 times, dental caries increased rapidly. Although the study revealed that less retentive sugar (in solution or in a food product eaten at meals) decreased the number of caries, an equally important conclusion from the study was that between-meal eating of sweets was more cariogenic than at-meal eating.

The results of the Vipeholm Study regarding the high cariogenicity of frequent between-meal eating of sweets was explained further by Stephan's (11) in vitro tests. In those tests the speed and duration of acid production in dental plaque after exposure to sucrose was determined (104). It was found that it took 20-30 seconds, after sucrose came in contact with plaque, to produce an acid potential that lasted 20-30 minutes causing tooth decalcification. Therefore, if an individual consumed sweets every 30 minutes there would be a continuous acid environment on the tooth surface. The result would be a continuous carious process.

A recent examination of per capita sugar consumption in the United States and Canada provides further evidence for the role of between-meal eating of sweets in the development of dental caries. The United States Department of Agriculture and the Canadian Sugar Institute reported that consumption of sugar has not fluctuated markedly since 1921 (figure 8). However, during that time, there was an increase in the caries rate (30). Apparently, factors other than sugar intake are involved. Bibby (2) suggested that the use of sugar in so many types of manufactured foods and snack foods is the detrimental factor. Sugar is used in almost all types of manufactured foods, but the amount included in food products and beverages consumed between meals increased most rapidly. Figure 9 shows this shift, as well as the shift away from direct consumer use of refined sugar (38).

Information on caries prevalence and food use is available for groups by sex. Girls have more permanent tooth caries than boys (105)

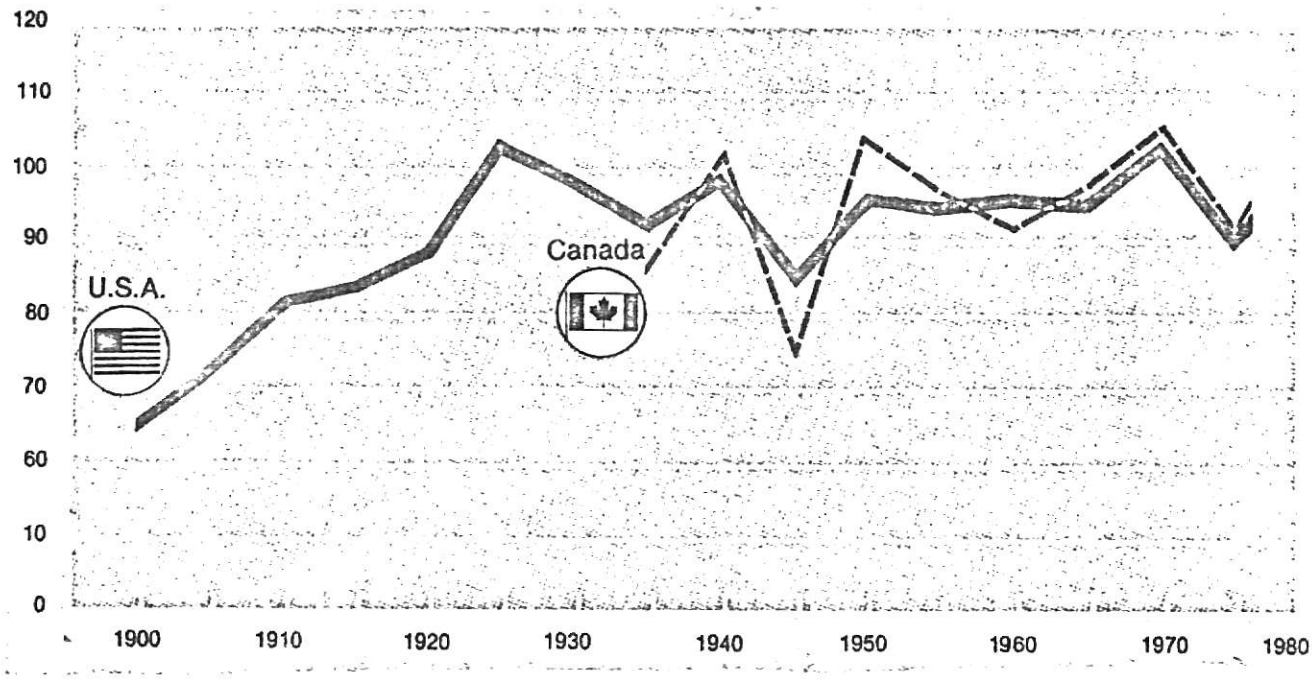

Fig. 8. Per capita consumption of sugar in the U. S. and Canada (1900-1976).

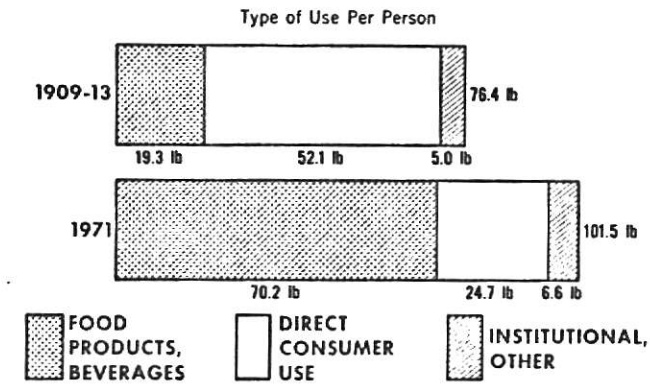

Fig. 9. Per capita per year use of refined sugar in industrially prepared food products and beverages, direct consumer use, and use by institutional and other users, selected periods.

of the same age although they eat less sugar and baked goods. Two factors were suggested for the higher caries rate in girls: earlier eruption of permanent teeth and more frequent snacking. Therefore, it appears that the form of the sugar and frequency of its intake are more important than the amount eaten.

PREVENTION OF DENTAL CARIES

Dental treatment can control and eliminate dental caries temporarily by extraction of diseased teeth, removing diseased portions of teeth, or repairing the damaged teeth. Susceptibility of a tooth to caries can be reduced by the placement of sealing agents on the occlusal surfaces or by the application of fluorides to the enamel surface (13). However, dental treatment has little effect on inhibiting the progress of the disease if neither mechanical nor chemical measures are employed.

Several measures have been suggested for the prevention of dental caries in our society. First, oral hygiene should be emphasized. The teeth should be cleaned immediately after eating, and the mouth should be rinsed with water, at least, after consuming any carbohydrate if brushing and flossing of the teeth is not possible (14). Secondly, new carbohydrate sweeteners could be used to sweeten foods so that the sucrose content can be reduced or eliminated (106). Thirdly, continued research should be conducted not only to dicover new artificial sweeteners, but also to find additives, such as phosphates, which make highly cariogenic foods less destructive to the teeth (12). Last, but not least, nutrition education should be used to modify food preferences and eating patterns so as to effect a decrease in dental caries incidence.

Use of New Carbohydrate Sweeteners

Although sucrose is used in foods for sweetness, it possesses many other characteristics that account for its widespread use. Sucrose helps to preserve food by its osmotic action, it assists moisture retention, improves appearance and influences texture. Most of these properties cannot be provided by non-caloric alternatives such as saccharin. However, one-third ounce of saccharin is equal to one pound of sucrose in sweetening power (107). Because consumers do not change their food habits readily, the first step in reduction of fermentable carbohydrates might be to encourage the replacement of sucrose-containing products with products containing sugar substitutes. The substitution of polyols such as sorbitol, mannitol, and xylitol for sucrose as a sweetener may lead to significant reduction in dental decay in human beings (108).

Glucose syrups. Glucose syrups are products resulting from partial hydrolysis of starch in which starch has undergone in the food factory some of the changes it would otherwise go through early in the course of normal digestion. When (for six months) baboons were fed a diet rich in glucose syrup, they produced less calculus on the teeth than when they were fed an isocaloric diet containing sucrose (107).

When glucose syrup is used in food preparation in combination with glucose or fructose, many of the characteristics of sucrose are produced, except that of sweetness. By boiling glucose syrup the sweetness of sucrose can be obtained, but some of the other characteristics are lost. It is less brittle and becomes more sticky when moist. Those differences may not be serious obstacles in food acceptance to the consumer, but

they cannot be ignored. The replacement of sucrose by glucose syrup in less concentrated products leads to a reduction in osmotic pressure. Therefore, unless preservatives are added, the risk of microbiological spoilage is increased.

Fructose. Fructose or levulose, the sweetest of all known sugars, already has a long production history (106). Fructose was produced at first from inulin-containing plants like the Jerusalem artichoke, dahlia, or chicory. The early methods were expensive and have been replaced today by the more economical method of separating glucose and fructose from invert sugar solutions by ion-exchange chromatography. Variations of this new method have lowered the price of fructose, drastically, the last few years and has opened up food use markets.

Fructose has a spectrum of properties that justify the prediction that it will capture a growing part of the total carbohydrate sweetener market. It is sweeter and more soluble than sucrose, and has the ability to improve fruit flavors. In addition, fructose is metabolized without initial need for insulin, does not induce insulin release, and is less cariogenic than sucrose.

Sorbitol. Sorbitol occurs naturally in some fruits, but commercially it is prepared by hydrogenation of glucose (107). In many countries, sorbitol is used extensively in foods with the non-caloric sweetener, saccharin. On a weight basis, sorbitol has only half the sweetness of ordinary table sugar, and therefore, usually has its sweetness supplemented by saccharin. It provides as many calories as sucrose. Sorbitol's rate of absorption is slower than sucrose; thus, it has been used in foods for diabetics where a rise in blood sugar level must be avoided. Oral bacteria

do not ferment sorbitol readily, and that is why it is considered a non-cariogenic sugar substitute.

Cornick and Bowen (109) observed in a two-year study with 8 monkeys that the ingestion of sorbitol was followed initially by the formation of plaque which had a syrupy consistency. It was noted also that the numbers of Streptococcus mutans in plaque declined markedly when sorbitol was substituted for sucrose, even though microorganisms ferment sorbitol. Prolonged ingestion by primates did not lead to the development of a plaque flora with an enhanced ability to metabolize sorbitol. All available evidence indicates that sorbitol is substantially less cariogenic in animals than sucrose.

A possible explanation for the lower cariogenicity of sorbitol may be found in the manner in shich it is metabolized by microorganisms. The breakdown of sorbitol produces mainly formic acid and ethanol; in contrast the metabolism of glucose results in the formation of lactic acid, primarily (109).

Unfortunately, most animal studies have shown a high morbidity following continued sorbitol ingestion; the surviving animals suffered from severe diarrhea and reduced growth rate (109). Only a limited quantity can be taken in food by many human beings, because it has a laxative effect (107).

Xylitol. Another promising carbohydrate sweetener is xylitol, a polyol obtained by hydrogenation of xylose (106). Xylose can be produced from xylane-containing plant materials.

Like fructose, xylitol was used first for parenteral nutrition and as a sweetener in diets for diabetics. Xylitol is as sweet as sucrose,

and because of its higher heat of solution has a pleasant cool taste. It holds great promise for production of noncariogenic confectionery products. A new industrial plant in Finland, which uses birch residues from the wood-working industry, is increasing the world production of xylitol and making a lower priced product available.

Mannitol. Mannitol occurs naturally in seaweed, and commercially it is extracted from it by reducing the sugar mannose (107). Mannitol has about the same characteristic properties as sorbitol. Cost is one of the main limitations in the wisespread use of mannitol and sorbitol.

Anti-caries Additives

Fluoride is the only nutrient that has been proven to increase caries resistance. By fluoride accumulation in surface enamel and its deposition during mineralization, the tooth becomes resistant to dental decay (17).

Dietary phosphate, vitamin B6, protein, and fats all have been associated with protecting teeth against caries, whereas, a number of other nutrients, including thiamin and selenium, have been associated with cariogenicity (30). Thiamin deficiency was correlated with a lowered caries incidence only because of a decrease in appetite (17).

In animals, dietary phosphate has been shown to have a protective influence against caries, even in the presence of a high sucrose diet (30). In the human population, however, clinical trials of dietary phosphate supplementation for caries control have not produced highly successful results. However, a general association was found between phosphates, and to a lesser extent calcium, and decay inhibition (107).

As the salivary concentration of phosphate and calcium ions falls, the apatite crystals of the tooth enamel dissolve more readily.

A calcium sucrose phosphate and inorganic calcium phosphate complex, called Anticay, was added to processed carbohydrate foods such as flour, sugar, honey, jams and canned fruits, at a level of 1 per cent of the carbohydrate content (107). Other phosphate- and calcium-containing additives for sugar and carbohydrate-containing foods are being studied as potential decay-reducing agents. One of the most promising at the present time is calcium glycerophosphate.

Nutrition Education

Food preferences and eating patterns are socially conditioned (110). The attitudes of people toward food are strongly influenced by what they have learned as children and what the environment teaches. Advertising has a considerable impact on food habits (13). Magazine advertising, TV commercials and free samples of products are attempts to persuade the consumer to buy a new product. Many homemaking magazines picture the ideal homemaker as one who keeps the cookie jar filled. Sociability and hospitality are associated with the sharing of food, often cariogenic in nature.

The objective of nutrition education, in the prevention of dental caries, is to impart knowledge to individuals so there is a decrease in the consumption of cariogenic foods. Successful nutrition education means a change in the present behavior of consuming large quantities of fermentable carbohydrates in a retentive form between meals (110). One

suggestion for modifying present food habits is to decrease the availability of cariogenic foods. Food restrictions, such as no sweets between meals, placed on children by parents, dentists, teachers, or others would help modify the oral environment in a positive direction (13).

School menus could be developed to exclude fermentable carbohydrates as much as possible. School vending machines could be stocked with fruits and low carbohydrate foods instead of soda pop and candy bars (111). Instead of consuming high-sucrose foods throughout the day, one could limit the ingestion of such foods to one time per day, preferably at meal time. When snacks are consumed in the home, at school, at a friend's house, at the neighborhood grocery store or drug store, noncariogenic snack foods should be encouraged.

Other recommendations have been made for the modification of the social acceptability of cariogenic foodstuffs. Television advertising might indicate the item's sugar content, and warn viewers about the possibility of tooth cavities. The use of warning labels on packaged candy may be another way of using education to reduce candy consumption. The price of candy, carbonated beverages, and other similar foods might be increased by taxation of sugar as was tried in Sweden (112).

SUMMARY

Dental decay is a dieto-bacterial disease. Tooth vulnerability, bacterial virulence, nutrient substrate suitability for bacterial proliferation, as well as the chemical and physical properties of the saliva, influence the initiation, development and extent of the dental decay process. Since teeth begin calcifying before birth and calcification

may not be completed until 20-25 years of age, dietary adequacy is important both pre- and postnatally.

Research studies indicate that sucrose is the major chemical food constituent responsible for the production of dental caries. However, the increased consumption of carbohydrates in the diet is not the exclusive determinant of caries activity. The physical form in which they are eaten, the other ingredients of the food with which they are compounded, the amount eaten and the frequency with which they are eaten affect the susceptibility of the teeth to decay.

Several measures have been suggested for the prevention of dental caries. Oral hygiene should be emphasized. Dietary measures include less frequent use of sugar-containing sticky foods between mealtimes and before retiring, and the use of artificial sweeteners to reduce the sucrose content of foods. Research should continue to discover new carbohydrate sweeteners and food additives to counteract the caries-producing potential of carbohydrates. Water fluoridation for both communal and school supplies should be promoted. Through nutrition education, food preferences and eating patterns should be modified to decrease the consumption of cariogenic foods.

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ACKNOWLEDGMENTS

The author is sincerely grateful to Dr. Kathleen Newell, her Major Professor and Associate Professor of Foods and Nutrition, for assistance and encouragement in preparation of the report.

Appreciation is expressed to Dr. Dorothy Harrison, Professor of Foods and Nutrition, and Dr. Faith Roach, Assistant Professor of Dietetics, Restaurant, and Institutional Management, for participating on the advisory committee and offering constructive suggestions for the report.

The author wishes especially to thank her parents, Mr. and Mrs. Darrell Fahrenkrog, for patience, understanding and moral support throughout the period of graduate study.

THE ROLE OF DIETARY CARBOHYDRATES IN DENTAL CARIES

by

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AN ABSTRACT OF A MASTER'S REPORT

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Foods and Nutrition

KANSAS STATE UNIVERSITY
Manhattan, Kansas

1977

Dental caries is one of the most prevalent diseases affecting mankind today. It is a modern disease that has increased in occurrence with the development and adoption of more civilized and sophisticated life styles, including greater consumption of refined type foods and changed dietary habits.

Dental decay is a dieto-bacterial disease. Tooth vulnerability, bacterial virulence, nutrient substrate suitability for bacterial proliferation, as well as the chemical and physical properties of the saliva influence the initiation, development and extent of the dental decay process. Since teeth begin calcifying before birth and calcification may not be completed until 20-25 years of age, dietary adequacy is important both pre- and postnatally.

Research studies indicate that sucrose is the major chemical food constituent responsible for the production of dental caries. However, the increased consumption of carbohydrates in the diet is not the exclusive determinant of caries activity. The physical form in which they are eaten, the other ingredients of the food with which they are compounded, the amount eaten and the frequency with which they are eaten affect the susceptibility of the teeth to decay.

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