

THE RELATIONSHIP BETWEEN DIET AND DENTAL CARIES

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الأمراض الخبيثة الإنتقالية من أعضاء الجسم المختلفة إلى عظام الفك أو الأنسجة المحيطة به تعتبر نادرة الحدوث وقد نُشر منها عدد بسيط من الحالات . التشخيص ليس مشكلة ولكن مكان التورم الخبيث الأصلي هو المشكلة في أغلب الأحيان . وجود الورم الثانوي أو المنتقل بالفم من أوائل علامات وجود المرض مما يدل على انتشاره بطريقة موسعة ويكون له دلالة على سوء الحالة وغالبا ما تحدث الوفاة . هذه الدراسة تستعرض حالتين من هذا النوع وتوجه انتباه جراحي الفم والفكين إلى هذا النوع من المرض الذي يصيب الفك وتحدد العلاقة بينهم وبين الفروع الطبية الأخرى .

Dental caries is a disease of multifactorial etiology and results from a complex interaction between both cariogenic and protective influences acting on the teeth. Though diet plays a major role in the causation of dental caries it has been a common mistake to over-simplify the relationship. Dietary manipulation is also extremely difficult to achieve thus limiting its role in the prevention of caries. Accordingly, alternative preventive measures such as the use of fissure sealants and fluoride which enhance the resistance of the host to disease and are of proven efficacy are more likely to be successful in the control and prevention of dental caries.

The driving force for the development of preventive dentistry and its effective use in patient management has been the expanding understanding of the disease of dental caries itself. Dental caries is now recognized as a disease of altered ecology in which the host, oral microflora and diet interact to present a challenge too strong for the normal defense mechanisms. It is, however, wrong to regard caries as a simple, continuing acid demineralization of the tooth enamel. Although teeth may be exposed to acid environment frequently, caries does not always arise since a carious lesion is the result of a dynamic interaction of demineralization and remineralization, which are under the influence of a whole range of both cariogenic and protective factors [Fig. 1]. It is clear from this model that diet is only a part of a large spectrum of the caries process. Sugar and other fermentable carbohydrates in diet are but only one aspect of cariogenic potential with other factors. The frequency of food intake, its retentiveness, and buffering potential also play an important part. This paper argue the misunderstanding of dietary considerations. Overemphasis on any one aspect of diet, like sugar concen-

tration, can lead to a narrow-minded approach to prevention of dental caries which is unlikely to succeed.

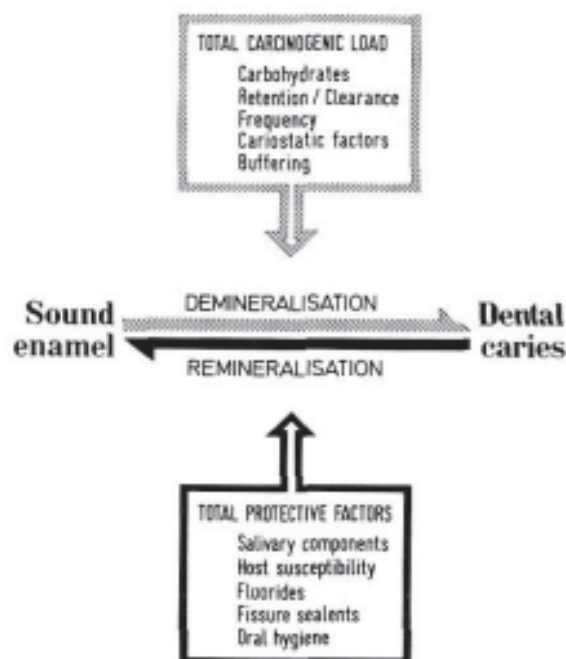


Figure 1. A dynamic model of caries

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The Host

Individuals vary in their susceptibility to disease, including dental caries. The reasons for this variability are not fully understood but some influences have been documented.

The size, shape and position of the teeth influences the washing effect of saliva and can thus allow additional protective effects of saliva on enamel or prevent self cleansing and encourage trapping of plaque micro-organisms and food particles. Orthodontic movement of teeth to more favorable positions can alter this only slightly.

Salivary components, too, vary considerably between individuals. These influence the microbial flora, immune status, plaque formation and enamel structure to generally provide a series of protective agents for oral structures. Stimulation of saliva can, therefore, be a critical factor in dental caries control. Saliva also plays an important role in the clearance of sugars from the mouth by dilution and reflex initiation of periodic swallowing. Dawes¹ came to the conclusion that unstimulated salivary flow rate, and the volumes of saliva in the mouth before and after swallowing are the two most important factors influencing clearance.

The concentration of sugars in the mouth appeared to be less important mainly because as sugar concentration increases, the stimulated flow rate increases which increases the rate of clearance. Interest has also been focused on the immunological aspect of caries and specific IgA antibodies to *Streptococcus mutans* have been detected in saliva by immune assays. The concentration of secretory IgA in whole saliva is significantly less in subjects with a high caries rate as compared with those with low caries experience.²

These local modifying influences of saliva and other host defenses are responsible for the inconsistent relationship between the drop in plaque pH brought about by the intake of carbohydrates and the caries incidence. Most of the host factors discussed here are inherent to the individual, many genetically determined and most only moderately amenable to manipulation, with the present state of knowledge.

Microbial Flora

Most researchers agree that bacterial organisms are capable of plaque formation and acid produc-

tion from a variety of fermentable carbohydrates.^{3,4,5} Studies on *Streptococcus mutans* strongly suggest its active involvement in initiation and progression of dental caries.⁶ Other organisms like *Lactobacillus acidophilus* have also been shown to be positively associated with dental caries.

The Role of Diet

Epidemiological studies have been used to try to establish the relationship of various types of diets and dietary components to caries incidence. It is agreed that foods which produce a pH drop below pH 5.5, known as critical pH, are considered detrimental to teeth. Some authors⁷ regard values below 5.7 as being damaging to teeth. Foods giving a pH drop between 5.5 and 6.0 are also dubious.⁸ This form of ranking is referred to as relative potential cariogenicity. The relative cariogenic potential of foods compared with known foods of high cariogenicity (sucrose) and low cariogenicity (Sorbitol) are of greater importance, however.⁹

Edgar¹⁰, using the plaque harvesting method, removed a representative sample of plaque from human mouths before and after consumption of foods and assessed the pH response of that plaque. The response followed a typical Stephen curve¹¹ and the potential acidogenicity of the foods was compared by measuring the minimum pH recorded and the area enclosed by the curve under the resting pH value. Acidogenicity in a range of snack foods is assessed in Table 1. Interestingly, some foods which are perceived to be "better for teeth" actually fare quite badly in such a ranking, compared with foods traditionally thought of as "bad for teeth", such as chocolate.

This study bore out the work of Rugg-Gunn et al,¹² who demonstrated that ingestion of chocolate or apples resulted in a similar pH response. Earlier work by Edgar et al¹⁰ using the same test procedure also showed that a wide range of foods containing either sugar, or starch, or combinations thereof are potentially acidogenic.

It is important to note that the concentration of fermentable carbohydrate in a food does not affect the pH drop in the mouth, although the period of time taken to return to normal pH levels may be related to concentration.^{13,14} This return to resting pH is as much related to the buffering capacity of the saliva as to the physical properties of the food itself. Thus, the retentiveness and, hence, clear-

Table 1. Acidogenic potential of various snack food (U.S.) grouped by category.

Group	Beverages	Fruits, etc.	Baked Goods	Sweets
Least acidogenic	1. Milk	Peanuts	Bread & Butter Graham Crackers	Caramels Sugared gum Chocolate Liquorice Sugarless gum Jellies Rock candy Clear mints
	2. Chocolate milk	Crisps apple		
	3. Carbonated beverages	Banana Sandwich cookies	Cream-filled cakes Orange	
	4. Apple juice Orange juice	Dates Raisins Sweetened cereal Plain sweet biscuits	Doughnut Bread & Jam Whole wheat bread Cakes	
	5.		Apple pie Chocolate Graham Angel Food Cake	
Most acidogenic	6.			Sourballs Fruit gums Fruit Lollipops

After Edgar⁸ (1981)

ance rate of a food is important.

Test for Cariogenicity of Foods

Bowen⁵ ranked the "cariogenic potential index" (CPI) of a selection of human snack foods by feeding laboratory rats via a gastric tube thus bypassing the mouth. Sucrose was used as a reference food and given a CPI of 1.0. Foods with a score of less than 1.0 are considered less cariogenic than sucrose, while those with a score above 1.0 were thought to be more cariogenic. Interestingly, the concentration of sucrose on a breakfast cereal made little or no difference to the CPI. The results, given in Table 2, show that potato chips (crisp) actually score higher than chocolate bars. Comparable results were obtained by Navial¹⁵ using the noncariogenic gel food procedure, and also by Mundorff et al.¹⁶

It is clear from most of these experiments that any foodstuff containing fermentable carbohydrate has the potential to cause significant amounts of acid to be produced at certain sites in the dentition, which can be followed by demineralization of the enamel and subsequent caries. However, the significance of acid production as an index of cariogenicity has

been questioned by various investigators^{17,18} who have shown that the total amount of titratable acid produced by a food does not necessarily parallel the amount of enamel it will dissolve.

However, the cariogenic potential of a food will

Table 2. Selected rankings of cariogenic potential index (CPI) in rats using human food as snacks.

Food Tested	CPI
Sucrose	1.0
Filled Chocolate Cookie	1.4
Cereal (14% Sucrose)	1.1
Cereal (8% Sucrose)	1.0
Cereal (60% Sucrose)	0.9
Coated Chocolate Candy	0.9
Potato Chips	0.8
Caramel	0.7
Chocolate Bar	0.7
Cereal (2% Sucrose)	0.5
Starch	0.5
Sucrose + 5% Dical	0.4
No meals by mouth	0.0

After Bowen⁵1978

be influenced also by a number of other factors, including the ability of foods to remain in the oral

cavity and, in some cases, the sequence of food intake. When the relationship between food and dental caries is examined, not only foods in themselves are important but also their relationship with other items of diet regarding its nature, timing and order.

Cariostatic Factors in Food

Some components of foods may be cariostatic. Proteins may remineralize enamel or reduce the rate of crystal dissolution.^{19,20} Some fatty acids have been shown to reduce caries in rat studies.²¹ Some minerals, such as phosphorous, have been shown to have a marked protective effect.^{21,22} The protection afforded by fluoride is well documented and has lead some researchers to refer to dental caries as a fluoride deficiency disease.^{23,24} These materials are all components of various foods. Furthermore, some cariostatic agents have been isolated from cereals²⁵ and cocoa.^{26,29} These factors all influence the level of caries caused. So the level of fermentable carbohydrate in food will not be directly related to the degree of caries caused.

Food Retention

Tests carried out by Bibby^{14,20} illustrate that, contrary to popular opinion, foods that are perceived to be "sticky", such as caramel, tend to clear from the oral cavity faster than many other foods. As Table 3 shows, after 15 minutes, white bread was retained in higher quantities in the oral cavity than cake, chocolate or hard mint. After 30 minutes, more residue from raisins was present than from caramel. Raisins have consistently been shown to be cariogenic.^{15,16}

Beverages, which are perceived to clear quickly from the mouth, actually sustain a low pH level for a long period as a "sticky" confectionery.¹¹

Eating Pattern/Frequency

At a population level, average amounts of sucrose consumed per capita relate to the average level of caries in the population.³¹ However, more detailed studies show the relationship to be less consistent.

Table 3. Representative figures for food retention in mouth after eating.

Food	Clearance Time		
	5min.	15min.	30min.
Peanuts	4.9 (mg)	3.3 (mg)	2.6 (mg)
DentyneGum	5.0	3.9	3.1
7 Up	6.3	2.4	2.1
Chocolate Milk	7.4	3.8	1.9
Potato Chip	12.3	4.9	2.5
White Bread	16.1	10.0	3.6
Raisins	16.8	5.7	3.0
Sponge Cake	18.8	6.0	4.2
Caramel	19.0	6.8	3.0
Milk Chocolate	19.0	6.8	3.0
Cracker (oil sprayed)	23.8	8.5	3.7
Hard Mint	31.9	9.4	2.5
Cracker (plain)	33.6	10.4	3.3
Sandwich Cookie	35.0	8.4	4.9

After Bibby¹⁴ (1981)

In the classical study often referred to as the Vipholm study,³² inmates of a Swedish Medical Institute were fed increased sucrose or other foods in different patterns and caries experience was monitored. Groups of patients receiving high levels of sucrose (up to 330g/day) with other meals experienced minimal increase in caries. But if smaller quantities of sucrose were consumed between meals, very high levels of caries ensued. The relationship was not, therefore, between the quantity of sucrose and caries but rather between frequency of intake and caries experience. This relationship, which has been confirmed in human and animal research sheds light on why population studies do not demonstrate a clear and consistent relationship between sugar consumption and caries.

Experience from primitive and developing cultures with little access to sucrose but abundant access to starch is often cited as evidence that sucrose and not starch results in dental caries. This evidence purports to be strengthened by the fact that introduction of Western type diet (including sucrose) immediately results in development of dental caries. It can be argued that introduction of a Western type diet is accompanied by increased affluence and an altered eating pattern. Such changes also include differences in the use of cooked

starches as much as differences in use of sucrose. Frequency of intake of any food increases dramatically and so potentially does dental caries, it is also interesting to note that caries has been shown to be associated with a diet consisting of sago starch, in a group of people in Papua New Guinea.³³

Sugars and their Role in Caries

In Western society, eating frequency has generally increased and snacking has become an accepted aspect of life. This change took place over a long period of the nineteenth and early twentieth centuries when the incidence of dental caries increased. However, in the past twenty years while the sucrose usage has not changed, dental caries has dramatically decreased. In the case of 5 year olds, the percentage with tooth decay fell from 73% in 1973 to 48% in 1983.³⁴

Nearly, all foods contain some fermentable carbohydrate. Reducing the frequency of eating just one of these foods, or reducing the concentration of sugars in a food, is unlikely to have a significant effect on the incidence of caries. It has been assumed tacitly both by practicing dentists and by too many dental investigators that the cariogenicity of individual foodstuffs is directly proportional to their content of sucrose or other fermentable carbohydrates. There is no quantitative data to support this belief. Actually the effects of high sucrose concentrations in increasing the rate of food clearance of some foods from the mouth and in inhibiting the fermentation process make it seem improbable that high sugar content of itself would be particularly damaging to teeth.¹⁴ The Vipeholm study has been mentioned as *an evidence for cariogenicity of sucrose* though investigators have questioned the reliability of a single clinical study from a mental institute.³⁵ There are a number of contradictory studies that have not been widely recognized. King et al,³⁶ in English children for example, found they could substantially increase sugar, as sucrose, in the children's diet without increasing caries.

While we must accept the belief that sugar and other fermentable carbohydrates play a major role in the causation of caries, it has been the common mistake to over-simplify the relationship. Preventive dental care in the form of fluoride therapy is likely to be more successful than dietary manipulation, which is notoriously difficult to achieve.

Considering the already substantial decline in the incidence of dental caries in the Western World³⁷ where frequency of eating has generally increased, it is reasonable to assume that alternative preventive treatments particularly fluoride are highly effective and are more likely to aid in prevention of caries.

Conclusion

All fermentable carbohydrates and foods containing fermentable carbohydrate, including sugars, have the potential to become the dietary component of the caries process. However, it is the frequency of consumption rather than the amount consumed which is associated with potential dental caries incidence. Dental caries can be effectively controlled by good oral hygiene, use of fissure sealants and fluoride, thus, enhancing the resistance of the host to the disease. The effectiveness of these procedures has been clearly demonstrated in many parts of the world^{38,39} and it would be unfortunate if the public hopes were raised to believe that diet control alone would solve the problem of dental caries.

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