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Dental caries in relation to nutrition: structural effects

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In discussing the relationship between nutrition and dental caries it is important to distinguish between the systemic effect of the absorbed food upon the developing tooth and the environmental effects of dietary habits. In this paper we are concerned with the former. There is no evidence to suggest that any physiological changes occur in the intact fully formed enamel. It is unlikely therefore that any change can take place in the composition of the fully formed enamel by active cellular processes. If nutritional factors are to influence subsequent caries then it is likely that they will do so during the formative period.

Dental caries is the result of bacterial attack on the surface of the tooth. Some teeth seem better equipped than others to combat this attack. It is pertinent to inquire how far this resistance can be attributed to nutritional factors present during the tooth's development.

Earlier work on the relation between nutrition, tooth structure and dental caries

Various attempts have been made to correlate imperfection of surface enamel structure with susceptibility to dental caries. Mellanby (1918, 1923) was one of the first to suggest such an association. After examining many hundreds of children's teeth she concluded that those teeth with comparatively mild surface hypoplasia were more susceptible to decay than those without such irregularities (Mellanby, 1934). Support for this hypothesis was provided by the observations of Davies (1939) on the first permanent molars in British schoolchildren, by Mellanby (1940) in Finnish children and by Bibby (1943) in a study of New England children. In each study there was some positive correlation between irregularity of enamel surface and dental caries. Contrary to these findings, Day & Sedwick (1934) reported no difference in the caries incidence in children with and without grossly hypoplastic teeth and Staz (1943) in a study of Johannesburg children concluded that hypoplastic teeth were less liable to decay than normal teeth.

There is therefore some evidence that poor surface structure may increase susceptibility to caries. It is in the interpretation of these results that controversy has arisen. There is evidence to show that deficiencies of vitamin D during tooth

development lead to hypoplasia in the guinea-pig and the dog (Howe, Wesson, Boyle & Wolbach, 1940; Mellanby, 1928). Such changes, however, could not be demonstrated in the rat (Weinmann & Schour, 1945). Several studies have indicated that an adequate vitamin D intake in growing children is associated with a reduced incidence of dental caries (Mellanby, 1934). Other workers have been unable to find any greater incidence of caries in rachitic than in non-rachitic children (Day, 1944). There is therefore no direct evidence to support the view that hypoplasia is necessarily due to a deficiency of vitamin D or that adequate vitamin D *per se* will prevent hypoplasia or reduce the incidence of dental caries.

Effect of wartime diet on the incidence of dental caries in children

Wartime rationing had a great effect on the dietary habits and the pattern of food eaten in many European countries. The greatest change was a reduction in the consumption of sugar, refined carbohydrates and sweets and also of meat. More potatoes and vegetables were eaten; the consumption of milk and fish was high and the flour used was of a high extraction rate. It is of interest, therefore, to examine the available information and to see what effect these changed conditions had on the incidence of caries in children.

During the 1939–45 war there was a progressive decrease in the incidence of caries in 5-year-old children in this country. This reduction continued until 1947–8 when the incidence again began to rise (Mellanby & Mellanby, 1948, 1950, 1951). Similar results were reported by Toverud (1949) for Scandinavia.

One interesting observation was that the reduction in caries did not immediately follow the fall in consumption of total sugar and refined carbohydrate. There was an apparent lag of about 2 years which suggested that the effect of alteration in diet was not merely upon the oral environment. Some authors believe that the reduction in caries incidence was due to an increased consumption of protective foods and vitamins (Mellanby & Coumoulos, 1944). However, in Finland, where the consumption of sugar was reduced to one-third of its prewar value and the general nutritional conditions were very bad, a similar reduction in caries was observed (Wilska, 1946). Sognaes (1947, 1948) has put forward the hypothesis that the resistance of the teeth was increased when the sugar intake was low during the period of tooth formation.

Parfitt (1954) has queried the apparent lag between decreased caries incidence and reduction of sugar consumption and again the delay in the appearance of an increase in caries after the restriction on sugar consumption was lessened. He re-examined Toverud's (1949) data concerning Norwegian children and computed yearly rates of caries incidence as distinct from absolute caries values. By this treatment he showed that there was an immediate increase in caries incidence rates, following the increased sugar consumption of 1945. It would thus seem that the wartime diet helped to reduce caries mainly because of the reduction in the consumption of fermentable carbohydrate. The evidence in favour of the increased consumption of protective foods being responsible for the lessened incidence of caries in the wartime children is very slight.

Use of purified diets in the production of experimental dental caries

Controlled experimental work on nutrition and caries must be carried out with laboratory animals. In the last decade the subject of nutrition and caries has been re-examined, caries-susceptible rats and hamsters and purified experimental rations of defined composition being used.

Sognaes (1948) studied the effect of such diets containing 67% sucrose and adequate in all known respects for growth and reproduction. When natural diets were given to rats and hamsters during pregnancy and lactation and the pups were maintained on the same diet the incidence of caries in the offspring was very low. When the natural diet was used throughout pregnancy and lactation and the offspring was weaned on to the purified diet the caries incidence was increased. If the purified diet was given to the mothers during pregnancy and lactation and to the offspring at weaning there was an even greater increase in the caries experience of the latter. This finding shows two distinct effects of the purified diet in increasing the caries incidence, one environmental and the other systemic (see Table 1).

Table 1. *Comparison of caries susceptibility in hamsters, rats, and mice 'bred' on stock diet and on a purified diet**

Species	No. of animals	Diet in pregnancy and lactation	Posteruptive experimental period		Mean caries incidence No. of molars affected
			Diet	Length (months)	
Hamster	6	Stock	Purified	4	4.6
Rat	12	Stock	Purified	4	0
Mouse	15	Stock	Purified	4	0
Hamster	3	Purified	Purified	2	9.0
Rat	10	Purified	Purified	2	2.4
Mouse	21	Purified	Purified	2	0.4

* Table adapted from Shaw & Sognaes (1955).

A similar experiment was carried out with monkeys (Shaw & Sognaes, 1955). Ten animals were placed on a purified diet when they were about 2 years old. At this age the first and second deciduous molars were erupted, the first permanent molars would normally erupt within a few months and the second and third molars during the next 3½ years. Three young animals (aged 6 months to 1 year) were placed on a similar diet; their second deciduous molars and all their permanent molars were still in process of formation. In this group ten of the twelve second deciduous molars developed caries within 20 months after eruption. In the older animals whose second deciduous molars were already formed at the time of transfer to the purified diet none of these teeth was carious after 4½ years on the diet. To quote Shaw & Sognaes (1955), 'Thus, the teeth which were formed while the monkeys were in the jungles of India have not decayed even though the monkeys have been fed a purified ration with a sucrose content of 73% for as long as 4½ years post-developmentally. In contrast the teeth which developed in our laboratory while the animals were being fed the purified diet have a relatively high caries susceptibility'. (See Table 2.)

Table 2. *Incidence of dental caries in molars of rhesus monkeys maintained on a purified ration for varying periods during development and maintenance and after eruption of molars**

No. of monkeys	Approx. initial age (years)	Total period on purified ration (years)	Molar teeth erupted	Period on purified ration		Mean no. of carious molars/monkey
				Pre-eruptive (years)	Posteruptive (years)	
10	1.9	5.5	First deciduous	0	4.5	0
			Second deciduous	0	4.5	0
			First permanent	0.3	5.2	0.4
			Second permanent	2.3	3.2	1.0
			Third permanent	3.2	2.3	3.0
3	0.7	2.4	First deciduous	0	2.4	0
			Second deciduous	0.2	2.2	3.3
			First permanent	1.6	0.8	0

* Table adapted from Shaw & Sognaes (1955).

Very few monkeys were used in this experiment and therefore it is perhaps prudent to consider the results to be suggestive rather than conclusive. There is therefore evidence from three species of animal that a purified diet with a high sugar content is cariogenic and that the incidence of caries is greater when the diet is given during the development of the teeth. Conversely a natural diet when given during the developmental period reduces the incidence of caries on subsequent exposure to the purified diet. It is logical to postulate that some factor is present in the natural diet which increases the tooth's resistance to decay. This factor is unlikely to be any of the major minerals or vitamins with which the purified diet is well supplied. This suggestion does not exclude the possibility that the consumption of a high-sugar diet during tooth formation is also a factor in caries susceptibility (Steinman & Haley, 1957).

Sognaes & Shaw (1954) investigated the mineral fraction of the natural diet. The salt mixture of the purified diet was replaced by an equal weight of ash prepared from the natural diet. When this modified diet was given to rats during pregnancy and lactation and the offspring were weaned on to the original purified diet there was a significant reduction in the incidence of caries. When the ash-containing diet was given throughout the pre- and post-natal life there was a greater reduction in the incidence of caries. It would seem therefore that the ash of the natural food influences caries both developmentally and environmentally. Thus it is reasonable to conclude as a working hypothesis that the natural diet contains some factors in small quantity which are not contained in the purified diet and which when incorporated in the tooth during development protect the rat in some measure against dental caries. The nature of these factors awaits elucidation. It is of interest to note the recent report (Adler, 1957) on the effect of traces of molybdenum on dental caries. Drinking water containing 0.1 p.p.m. of molybdenum as ammonium molybdate reduced the incidence of caries in albino rats which had received a coarse maize diet. Caries produced on this diet is not strictly comparable with that produced on the high-sugar diets since fracturing of the molars is involved in the aetiology.

Changes in tooth composition by dietary means

The mineral composition of teeth is by no means constant. The calcium content of normal rat enamel may vary between 29% and 35%; variations were observed also in phosphorus content (Hartles, 1951). Sobel & Hanok (1948) showed that, in rats, dietary variations in calcium and phosphate were reflected in their serum concentrations and also in the composition of the teeth being formed. This was not the only factor governing composition since there were differences in the mineral phases of bone, enamel and dentine of the same animal. There are present 'local factors' which determine the exact composition of the deposited mineral (Sobel, 1955). When rats were fed on a diet with a high calcium : phosphorous ratio, the mineral in the enamel and dentine had a high carbonate : phosphate ratio. A diet with a low calcium : phosphorus ratio resulted in a correspondingly lower carbonate : phosphate ratio in the teeth. Cotton-rats whose teeth had such an experimentally produced high carbonate : phosphate ratio had a much higher incidence of caries than those animals with a low carbonate : phosphate ratio (Sobel, 1955). It is worthy of note that the teeth of the two series of rats were histologically indistinguishable. Experiments on the mineral phase of human enamel have shown that acids dissolve carbonate preferentially (Logan & Taylor, 1938; Hardwick, 1949). Carbonate also dissolves more rapidly from the teeth of cotton-rats having a high carbonate : phosphate ratio than from those with a low carbonate : phosphate ratio (Sobel, 1955). These results therefore support the view that a tooth whose mineral phase is more easily soluble in acid is more likely to decay. We think it important to stress the point, that although teeth appear similar in structure to the histologist they may have small chemical differences which may influence their subsequent caries experience. These differences may arise as a result of variation in humoral and local factors during the developmental period.

Fluoride as a nutritional factor in the formation of caries-resistant teeth

There is some reluctance to classify fluoride as an essential nutrient. There is no evidence that the fluoride ion serves any purpose in animals save that of conferring increased resistance to dental caries. Protection is always relative and must be viewed from the level of the population rather than from that of the individual. It can now be stated quite categorically that in any community whose teeth have developed while it was consuming an optimal amount of fluoride, such as is provided by a drinking water containing 1 p.p.m., the incidence of dental caries will be less than in a similar community where the intake of fluoride is very low.

The amount of fluoride deposited in human enamel increases as the fluoride content of the drinking water increases (McClure & Likins, 1951; Jenkins & Speirs, 1953; Isaac, Brudevold, Smith & Gardner, 1958). This effect may be due to an increase both in the amount of fluoride incorporated in the enamel during formation and in that acquired after eruption by surface adsorption. There is evidence to support the view that the incorporation of fluoride into the tooth structure makes the enamel more resistant to dissolution by acids formed during carbohydrate breakdown by the oral flora. Enamel samples taken from the deciduous teeth of children who

had received since birth a drinking water containing 1·2 p.p.m. of fluoride were less soluble in acid than samples taken from children who had not received a fluoridated water (Finn & DeMarco, 1956). Isaac (1956) has shown that the solubility in acid of both deciduous and permanent enamel is inversely proportional to the fluoride content.

It seems likely that maximum protection against dental caries will be obtained when the individual receives fluoride both during the period of tooth development and after eruption of the teeth. In the formative period the fluoride is incorporated into the enamel as it is laid down and post-eruptively the surface of the enamel will acquire fluoride from the saliva. Thus there is justification for concluding that fluoride is a necessary component of the tooth which helps to increase its resistance to dissolution by acids. The evidence concerning fluoride and dental caries is well reviewed in the report of the Commission of Inquiry on the Fluoridation of Public Water Supplies (Stilwell, Edson & Stainton, 1957) presented to the New Zealand Government.

Conclusions

The part played by adequate nutrition in preventing dental caries is still obscure. The reduction of caries observed in children in several European countries during the 1939–45 war is unlikely to have been caused by an increase in the consumption of protective foods. It is more likely to be related to a reduction in the consumption of sugar and to less eating between meals.

There is experimental evidence to show that the resistance of the rat tooth can be increased by nutritional means. Evidence for man is unsatisfactory owing to the difficulty of conducting definitive experiments during the period of tooth development.

The only nutritional factor that has been proved to be of value in reducing the incidence of dental caries in man is the fluoride ion. It may well be shown that other minor components incorporated in the tooth structure during development will increase its resistance to the carious process. In the meantime every effort should be made to ensure that full use is made of the knowledge we possess.

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Dental caries in relation to nutrition: environmental effects

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Ever since the classical work of Miller (1890), which demonstrated the decalcification of teeth incubated with saliva and carbohydrate foods, it has been widely believed that the acids formed from carbohydrate by mouth bacteria are the major factor in the development of caries. The local effect of sugar has been proved by the finding of Kite, Shaw & Sognaes (1950) that caries did not occur in susceptible animals when they received a high-sugar diet by stomach tube. It has been suggested that the initial attack on the enamel is made by proteolytic bacteria (Pincus, 1939; Gottlieb, 1947) but this view became less likely when it was discovered that some of the organic matter of enamel was soluble in acid (Stack, 1955) and could therefore be removed without proteolysis. A compromise theory suggests that the breakdown products of proteolytic action remove the calcium salts by forming complexes at neutral or even alkaline pH (Atkinson & Matthews, 1949). A similar concept has recently been developed by Schatz and his colleagues in what they call the 'proteolytic-chelation' theory (Schatz, Karlson, Martin & Schatz, 1957). They emphasize that proteolysis, and the metabolism of many substances, may result in the formation of complexing agents, and suggest that the formation of such substances by salivary organisms from the organic matter of the teeth could lead to decalcification without acid. Lura (1957) has suggested in addition that food and salivary constituents might be the precursors of complexing substances. These