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NUTRITION AND TEETH

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Nutrition and the teeth

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The fact that this Symposium has been arranged to discuss the way in which the food we take can influence the development and calcification of our teeth is an indication that there are still many gaps in our knowledge. It is a subject that has attracted many research workers and a mere review of all the work that has been done on this highly complex problem would fill several volumes. In a short paper I can only attempt to deal with a limited field and record in a few words the results of work that may have taken months or years of arduous labour and much imaginative thought.

The human species is not a good medium to use for any controlled experiments, especially when they have to be carried on over a long period of time, and most of the knowledge we have regarding the effect of various diets upon the calcification of bones and teeth is the result of investigations carried out upon animals that develop more quickly and can be killed at the appropriate time and so provide an opportunity for a detailed examination of the tissue being investigated. There is, however, a good deal of evidence that often the results obtained in one species are not valid for another and a good deal of caution must be exercised in applying the results of animal experiments to the human race and in assuming that the reactions would be the same. In spite of the volume of work that has been done and the valuable information gained from animal experiments, we have little definite knowledge regarding the way in which calcification in man is affected by the various factors in his diet, and when I settled down to the task of trying to present a picture of it I found myself with a very large canvas but very little paint.

It is generally agreed that once teeth are calcified and erupted into the mouth the enamel is capable of little change and any effect diet may have upon the breakdown of enamel, as found in that ubiquitous disease dental caries, must be due to local action in the mouth rather than to any effect it may have as the result of metabolism in the body. The way, however, in which enamel is destroyed by local factors, such as food, saliva and the organisms they support, is still far from being understood but, whatever the line of attack, I think it is reasonable to assume that the resistance of a tooth to any attack will be influenced by the way it has developed and the extent to which calcification has taken place. It is to those factors in the diet that are more specifically related to calcification that I propose referring in this paper.

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Most of the knowledge we have regarding calcification and development generally is based upon observing the effects produced in animals by the removal of certain factors from a diet known to be adequate and satisfactory for normal growth and development or on the equally valuable experiment of adding substances to it. The importance of studying the effect of adding to a diet as well as that of subtracting is apt to be lost sight of, and yet in countries with an unlimited food supply it may be the predominating factor. To quote from an article by C. G. King (1949), the Scientific Director of the Nutrition Foundation, New York, 'Many competent students of public health and nutrition already regard an excess intake of calories as one of the most damaging forms of malnutrition in the United States'. I well remember being very impressed many years ago by some experiments that Professor Wolbach was conducting in the nutritional laboratories at Harvard. He showed me a number of very healthy looking monkeys that were being fed on a basal diet. In the next cage was a miserable looking specimen. On my inquiring the reason he told me that this monkey was still receiving the same basal diet as the others, upon which it had previously thrived, but for some time it had also had in addition as much carbohydrates as it would take.

The basal diet we were subjected to during the war, although thought to be deficient in calorific value, kept us surprisingly healthy, and the absence of epidemics was impressive. There is some evidence that the abolition of rationing and our return to a fuller and unrestricted diet has not been an unmixed blessing. Lady Mellanby's work (Mellanby & Coumoulos, 1946; Mellanby & Mellanby, 1954) shed some light on this question. She inspected the teeth of a large number of London schoolchildren, both during the war and more recently. It would appear that the enamel calcified during the period the child was receiving the restricted wartime diet was better, and showed less signs of hypoplasia, than was found in the postwar period when food became more plentiful.

Basically we know that calcification of the bones and teeth depends upon an adequate intake of calcium and phosphorus with a sufficient amount of vitamin D to ensure absorption of the calcium. Some vitamin C is also necessary which, although there is no evidence that it is directly concerned with calcification, is recognized as being essential for the normal development of the connective-tissue matrix in which calcification takes place. The effect of supplementing the diet of young children with these substances has been remarkable. Rickets, a condition that has as its main features gross abnormality in the development and calcification of bone and teeth, was until the early twenties a common disease in the industrial areas of this country. It has now been almost completely wiped out by supplementing the diet of young children with milk, cod-liver oil and orange juice.

Further confirmation as regards the importance of vitamin D is given by the classical work of Lady Mellanby (1929) who produced hypoplasia in the teeth of puppies by feeding them on diets deficient in vitamin D, in spite of the fact that they were receiving calcium in an amount that proved adequate when vitamin D was restored to the diet. The hypoplasia she produced in the puppies was similar to that found in the teeth of children who had suffered from rickets. Though vitamin D

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does enable the body to make the best use of even a minimal amount of calcium, she and many other workers investigating the complex problem of calcification, particularly Irving (1944–5, 1957) and Schour & Massler (1945*a*,*b*), stressed the importance of not only an adequate amount of calcium and phosphorus in the diet but of their ratio to each other. A marked excess of either will lead to defective calcification. The main danger, and the one most likely to occur so far as the human diet is concerned, is for it to be deficient in calcium without a corresponding fall in the phosphorus intake. Helen Mellanby (1952) considered this imbalance to be the probable cause of the defective calcification of the enamel she noted in certain parts of Newfoundland where the diet contained little milk or other source of calcium but where, on the other hand, the phosphorus content was raised by the liberal consumption of fish.

Although some experiments on animals have shown that certain trace elements such as manganese, zinc and cobalt are necessary for the calcification of their bones and teeth, there is no evidence that they are sufficiently lacking in any human diet as to require any special consideration.

Sir Edward Mellanby (1944) in his Croonian lecture described the neurological effects the absence of vitamin A from the diet had upon experimental animals. These signs he attributed to the overgrowth of bone, particularly at the base of the skull where it compressed the cranial nerves. No doubt a complete absence of vitamin A in the human diet would also lead to the abnormal development of the bones and probably of the teeth. Fortunately, most natural foods containing vitamin D also contain vitamin A and if we ensure that an adequate amount of vitamin D is present in the human diet we can, for all practical purposes, assume that it will also contain a sufficient amount of vitamin A. If we are dealing with a normal healthy child receiving a diet adequate in calories, the inclusion of an adequate amount of calcium, with vitamins D and C, should take care of calcification. Experimental rickets can be produced in rats by feeding them on a diet low in phosphorus, especially if it is also deficient in vitamin D (Irving, 1944–5). It is, however, unlikely that any natural diet containing the requisite amounts of proteins, fats, carbohydrates and calcium would not also contain at least the minimal amount of phosphorus necessary for calcification.

The actual amount of calcium required daily to satisfy the needs of a human being is by no means certain. Sherman (1920), by a number of balance experiments in which the intake and output of calcium showed a reasonable approach to equilibrium, found the mean daily intake to be 0·45 g. He added a somewhat arbitrary margin for safety of 50% and stated the daily requirement for ordinary conditions of life to be 0·65 g. Leitch (1936–7) reviewed and supplemented the work of Sherman and arrived at a somewhat similar figure. Unfortunately, natural foods, apart from milk and milk products, are not particularly rich in calcium, as is shown in the following table (Wishart, 1938):

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Food	Calcium (mg/100 g edible substance)
Cheese	931
Milk	120
Oatmeal	69
Green vegetables	45–67
White bread	27
Potatoes	14
Meat	12

In this country the main source of our calcium is milk, and to ensure an adequate intake a woman during pregnancy, when she has to provide for the needs of the foetus as well as her own, should receive not less than 1 pt. a day. A similar amount is required by the infant and growing child to provide for the active calcification taking place. Massler & Schour (1946) consider, as the result of their investigations, that there are certain critical periods when calcification of the teeth is particularly susceptible to injury, namely during the first 10 months of life and again at $2\frac{1}{2}$ and 5 years.

Milk, of course, is not the only source of calcium and, indeed, in some countries it is a rare commodity. In Singapore and Malaya the amount of milk consumed is negligible yet there is little, if any, rickets and the teeth of the children I inspected seemed to be well calcified. The children are, however, mostly breast fed, often into the 2nd year, and a common food taken by the mothers is a small shell-fish which is ground up and eaten whole. The sunlight acting upon their skins ensures an adequate amount of vitamin D.

The whole question of our calcium income and expenditure is bristling with unsolved problems and, notwithstanding the large volume of literature dealing with the subject, our knowledge of calcium metabolism is still fragmentary. Calcium, to be of value, must be presented to the gut in a soluble form. The mere fact that it is present in the food is no criterion that it will be absorbed and become available to take part in the process of calcification. Oatmeal and wholemeal bread contain calcium to the extent of 50–70 mg/100 g and yet, as was first pointed out by Sir Edward Mellanby (1921), they are poor sources of calcium, which he attributed to some anti-calcifying substance in cereals.

Bruce & Callow (1934) found that the phosphorus in cereals was less effective than an inorganic form in curing rickets they had produced in animals by feeding them on a diet deficient in phosphorus. They suggested that the phosphorus present in the cereals as phytic acid probably combined with the calcium to produce an insoluble salt. The deleterious effect of phytic acid was confirmed by Harrison & Mellanby (1939) when they showed that puppies fed on an adequate diet could be made rachitic by adding phytic acid to the otherwise satisfactory diet. Further work by McCance & Widdowson (1942–3) and Krebs & Mellanby (1943) led to addition of calcium carbonate to the loaf, at present to the extent of 14 oz. to 280 lb. of flour, to combat the rachitogenic action of phytic acid and so provide some calcium in a soluble and absorbable state.

I have already referred to Lady Mellanby's work which emphasized the importance of vitamin D in ensuring that the best use was made of any calcium contained in the

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diet. It would seem from the results she obtained by feeding puppies on diets deficient in vitamin D that, irrespective of the amount of calcium and phosphorus in the diet, the bones and teeth failed to calcify completely and the enamel showed varying degrees of hypoplasia. Mellanby, Coumoulos & Kelley (1957), when making a survey of the teeth of a large number of London schoolchildren, found that in many the enamel showed some signs of hypoplasia. This effect, they contend, was primarily due to the lack of an adequate amount of vitamin D in the diet. Though definite proof of such an opinion is lacking, it is supported by the fact that the enamel is usually well calcified in those children who have received a supplement of cod-liver oil during the calcifying period.

The manner of action of vitamin D is not settled. McGowan, Cunningham & Auchinachie (1931) suggest that it primarily attacks the lipids of the tissues with the liberation of phosphoric acid, the lack of which in the blood is a notable feature of rickets. It also prevents the loss of calcium by the bowel by causing its deposition as phosphate. The human daily requirements for vitamin D are not precisely known. Once calcification is complete it would appear that very little is required to maintain health. According to Evans (1941), growing children need between 2000 and 5000 i.u., but adults require only about 200–500 i.u.

The main sources of vitamin D in this country are fish, liver, butter and egg yolk. With such limited sources of supply one can appreciate the likelihood of its being deficient and the importance of supplementing the diet with concentrates such as cod-liver oil. In tropical countries where the skin can be exposed to sunlight the need is not so urgent for we know that ultraviolet light acting on the skin can synthesize vitamin D by its action on 7-dehydrocholesterol which is present in the superficial tissues.

Though it is recognized that a human diet must contain some vitamin C for the maintenance of health and that a lack of it produces scurvy, the knowledge we have regarding the part it may play in the process of calcification is largely based upon animal experiments. Wolbach & Howe (1926) made a detailed study of the effect produced by feeding guinea-pigs on a scorbutic diet. They demonstrated that such a diet had a profound effect on the calcification of the bones and dentine but noted little change, if any, in the enamel. The dentine lost its normal structure and was laid down in a very irregular manner, which they attributed to faulty development of the connective-tissue matrix rather than to any interference with calcification *per se*. Irving (1957) in his recent monograph on calcium metabolism states 'The fundamental cause of these changes is still a matter of dispute, but it would probably be generally agreed that they are due to the inability of the osteoblast to secrete a calcifiable matrix'.

How far these animal experiments can be applied to man is still in doubt and we have no definite information as to the minimum amount of vitamin C the diet should contain to ensure normal calcification. The amount required to maintain a level of saturation that can be estimated by the amount secreted in the urine is about 50 mg daily. The sources of supply are citrus fruits, green vegetables and liver.

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In conclusion, I would reiterate that calcium metabolism is a highly complex problem that is far from being solved. It is affected in many ways but, whatever a diet may or may not contain provided it contains an adequate amount of calcium, it appears from the evidence we have that in man the only important single factor is vitamin D.

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Teeth in earlier human populations

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It is particularly fortunate, at least in human beings, that the teeth are situated in the skull. The archaeologist of the past was far from particular about what was thrown away, provided it did not yield one of the standard measurements, which has resulted in many post-cranial remains finding their way into the museum dust-bins of the world. However, the teeth remained, and odontological aspects of anthropology are now beginning to stimulate considerable interest.

The purpose of my study has been to try to ascertain the influence of food on the oral conditions of earlier man. There are, of course, numerous difficulties, especially in the statistical treatment of results. Caution must be taken against any possible selective bias in a cranial series (Krogman, 1938), and mean ages of groups should be similar (Stewart, 1931; Krogman, 1935). Where possible I have guarded against inaccuracies from these sources. Although in some groups there are slight differences between the sexes in the frequency of dental disease, it was thought sufficient to combine the data in this analysis. With periodontal disease, skull material is likely

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