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Nutrition, diet and dental public health

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Nutrition and diet can affect teeth in three ways: by affecting the structure of teeth (and thus appearance), by causing dental caries and by eroding teeth. The importance of dietary sugars in causing dental caries is clearly established and a reduction in consumption of non-milk extrinsic sugars recommended by government. The Health Education Authority has played a major role in promoting this aspect of dietary advice. The structure of teeth is influenced by nutrition; much of the evidence for this being published by Mellanby and colleagues in this country. Interest in this topic has increased recently and it seems probable that malnutrition enhances susceptibility to dental caries, and possible that it increases susceptibility to enamel defects especially in areas with moderate to high levels of fluoride ingestion. The prevalence and severity of dental erosion is likely to be increasing in Britain. Preliminary studies indicated that fluoride helps to protect teeth against erosion, but more research is needed.

Key words: nutrition, diet, dental caries, dental hypoplasia, dental erosion

Diet, fluoride and dental caries

During the past one hundred years the prevalence and severity of dental caries has increased, particularly in industrialised and developing countries, so that it is an important public health problem. This rise in the occurrence of dental caries has been associated with increasing use of sugar and refinement of foods (Hardwick, 1960; Rugg-Gunn, 1993). In many industrialised countries, caries experience reached its peak in the 1950s and 1960s, and has declined since then. Ten years ago, two international groups, the Boston Conference held in 1982 (Glass, 1982) and the joint working party of the World Health Organisation/Fédération Dentaire Internationale (1985) emphasised the very great importance of fluoride in the control of dental caries throughout the world. Of paramount importance in most countries was the widespread use of fluoride toothpastes.

Thus, in simple terms, the rise in caries throughout the world has been due to increasing consumption of sugar while the decline in caries has been due to the appropriate use of fluorides.

A more recent and more modest appraisal of changes in caries experience, particularly in Europe, took place at the ORCA congress in 1990. In most areas of Europe caries continues to decline, but two subsidiary points should be mentioned. First, Marthaler (1990) concluded that while fluoride seemed to be a very important cause of caries decline, some other factor seemed to be acting in some countrires. Improved diet was strongly suggested, for example in Finland and Switzerland, but lack of good epidemiological dietary data prevented more confident conclusions. Second, polarisation of caries experience has occurred in many countries with caries experience declining in the better off groups of society but getting worse in more deprived groups. This was recorded in the surveys of 5-year-old children in fluoridated Newcastle and non-fluoridated Northumber-

and, where caries experience increased in social class IV and V children in both areas between 1981 and 1987, but not in the social class I and II children (Rugg-Gunn, Carmichael and Ferrell, 1988). This has recently been echoed by Truin *et al.* (1991) in their surveys of children in The Hague.

Dietary sugars and dental caries

There is no shortage of evidence linking dietary sugars and dental caries, and it is worth recalling the many authoritative non-commercial statements on this issue in this country. One of the first was the report by the Committee on Medical Aspects of Food Policy (1969); then came the report *Eating for Health* (Department of Health and Social Security, 1978), the report of the Dental Strategy Review Group (Department of Health and Social Security, 1981), the report of the National Advisory Committee on Nutrition Education (1983), the British Medical Association (1986), the COMA report on *Dietary Sugars and Human Disease* (Department of Health, 1989), which was endorsed and targets for sugar consumption quantified in the COMA report on dietary reference values (Department of Health, 1991). The gross recommendations of this latter COMA report agreed very closely with those in the report of the World Health Organisation (1990). Unanimity between dental professional groups within the United Kingdom on this issue can be seen in policy documents published by the British Association for the Study of Community Dentistry, the British Society of Paediatric Dentistry and the British Dental Association. The policy document on dietary sugars issued by the British Dietetic Association (1992), follows the Department of Health (1989) line closely and recommends quite clearly that consumption of non-milk extrinsic sugars by the UK population should be reduced.

The task of providing those involved in community preventive programmes with accurate information in England falls within the remit of the Health Education Authority. Professor Holloway chaired the dental advisory committee of the then Health Education Council for a number of years and one of the fruits of his labours was *The Scientific Basis of Dental Health Education*, first published in 1976. In the view of many, this document proved to be a watershed so that, for the first time, we had the basic message agreed by the vast majority of experts in this country, clearly written down. The second edition was published in 1985 and the third edition in 1989.

The health education aspect of achieving a reduction in sugars consumption will not be considered here except to suggest a positive approach to dietary advice may be more successful than the rather negative approach generally pursued so far. Last year Mason and Judd (1991) reported an interventional study aimed at increasing the fibre intake of a group of 15 adult volunteers in London. Before the intervention, the fibre intake was low – 19 g per day on average. All subjects were given instruction on how to increase their fibre intake and reference cards to assist purchase and food choice. They maintained their new diet for a ten-week study period, during which their fibre intake had increased to an average of 40 g per person per day. Most interestingly, fat intake declined from 40 per cent to 34 per cent of energy intake. The authors only quoted total sugars consumption but, since milk and fresh fruit consumption increased markedly, there must have been a reasonable, about 30 per cent, reduction in intake of non-milk extrinsic sugars. Those in dental health education need to look at this study as it seems to produce generally beneficial results (although many would question the need for fibre intake to increase as high as 40 g per day) by using a positive dietary message as opposed to saying 'don't' all the time. At the end of the ten-week intervention, the subjects stated that they enjoyed the

new diet and wished to continue it. Mason and Judd (personal communication) said that the subjects found the high fibre diet very little more expensive than their previous diet.

Nutrition and dental health

The Health Education Authority publication (1989) *The Scientific Basis of Dental Health Education* quite rightly stresses the very great importance of the presence of dietary sugars locally in the mouth. The nutritional influence, fluoride apart, is rather dismissed. During the 1920s and 1930s the prevailing view was that dental caries, like rickets, was largely a deficiency disease. Chief among these British dental scientists was the very capable Lady May Mellanby, with whom Professor Holloway worked for a number of years. Her work deserves to be read more widely, although most of it is published in Medical Research Council reports, copies of which are becoming increasingly hard to find. May Mellanby's work spanned 50 years. In 1918 she reported that dogs reared on diets deficient in a 'fat soluble A accessory food factor' (which she subsequently recognised as vitamin D) had delayed development of teeth which were poorly aligned and which had very deficient, poorly calcified enamel (Mellanby, 1918). This defective enamel was termed hypoplasia which, according to Mellanby (1923) included not only defects observed by the naked eye, but also microscopic changes recorded only when the teeth were sectioned. She was aware that many children in Britain at that time had hypoplastic deciduous and permanent teeth and she considered it very likely that vitamin deficiency was the cause of the observed hypoplasia.

Mellanby attributed the observed improvement in the appearance of children's teeth in London between 1929 and 1943 (Mellanby and Coumoulos, 1944), to improved diet, notably the introduction of cheap milk in 1934 and the introduction of the war-time food policy. This included the provision of cod liver oil to pregnant and lactating women, infants and to young children and the addition of vitamins A and D to margarine and the fortification of bread with calcium carbonate. Mellanby postulated that vitamin D deficiency was responsible for the higher prevalence of hypoplastic teeth in children and that these teeth were more prone to decay, thereby concluding that vitamin D deficiency was a factor in causing dental caries. Data given in Table 1 are taken from her examination of 302 deciduous teeth collected from 218 children aged 2–13 years in Sheffield. Nearly all the hypoplastic teeth were carious, but only a quarter of the teeth without hypoplasia were carious. Several other studies have also found a positive relationship between the presence of hypoplasia and caries, usually in deciduous teeth (Rugg-Gunn, 1993). So, in spite of the imperfections of the Sheffield study, this aspect of Mellanby's

Table 1 Number of teeth that were of normal (good or sound) structure or hypoplastic, that were also caries-free or were carious. Data from Mellanby (1923).

		Normal structure		*Hypoplastic*	
Tooth type	*No. examined*	*Caries-free*	*Caries*	*Caries-free*	*Caries*
Incisors	47	34	5	0	8
Canines	29	1	0	12	16
1st molars	88	1	5	1	81
2nd molars	138	0	1	0	137
	302	36	11	13	242

work can be accepted. Mellanby then embarked on a series of clinical trials in which the effect of giving 'abundant fat soluble vitamins and calcium' to children was tested (Mellanby, 1934). Some of the group sizes were unfortunately rather small but, nevertheless, initiation and spread of dental caries was lower and the number of previous carious lesions which showed hardening was higher in the children who received the supplements. Some of the effect must have been post-eruptive since the trials were too short to show any pre-eruptive effect and Mellanby did not fully explain this aspect of the mode of action of vitamin D.

There then followed a longer (two year) trial carried out in three children's institutions in Birmingham. The three groups of children received supplements of either treacle, olive oil (which was low in vitamin D) or cod liver oil (high in vitamin D). They were initially aged 9.5 years and were dentally examined every 6 months during the two year trial. The results were analysed statistically by Young (1937) who reported that children who received cod liver oil had the smallest caries increment. A second trial was undertaken in which two groups of children initially aged 10 years received either vitamin D or olive oil supplements over 1.5 years. There was, again, a statistically significant difference in favour of the vitamin D group, for erupting permanent teeth, but not for teeth already erupted. The results of the Birmingham investigations were summarised by Young (1937) thus: 'they show conclusively that the relatively high vitamin D content of food can do much to diminish the incidence of caries if the vitamin is given during the development of teeth. That a beneficial effect may be obtained if the vitamin is given at a fairly late stage of development and that even when it is given after eruption of teeth the onset and spread of caries is delayed'.

Bunting (1935) and Jay (1940) both working in America stated quite clearly that they could not accept that caries could be controlled by the addition of calcium phosphate or vitamins, and in England Weaver (1935) also found Mellanby's evidence unconvincing, criticising the way she presented her data and indicating areas of her arguments which conflicted with other evidence. Subsequently, Mellanby (1937) modified her views on the beneficial effects of vitamin D but still maintained it was *a* factor in the resistance to dental caries rather than *the* factor. Mellanby's ideas cut little ice during the 1950s, '60s and '70s mainly because sugar was such a dominant factor.

However, a new wave of recent reports supports several aspects of Mellanby's work. Ferne *et al.* (1990) described the dental health of low birth weight children born during one year at two hospitals in London. The 110 low birth weight children were examined at the age of 5 years. Ninety-three normal birth weight children from the same locality and matched for age, sex, race and socio-economic status formed the control group. Race and sex had no effect on the prevalence of enamel defects which were much more prevalent (77 per cent) in the low birth weight children than in the control children (37 per cent). Although causes of low birth weight were not given, poor nutrition is likely to have been one factor.

The extensive data of Enwonwu (1973) in Nigeria, Sweeney, Saffir and Leon (1971) and Infante and Gillespie (1974) in Central and South America have indicated that nutrition linked to diarrhoeal diseases is positively related to the occurrence of enamel hypoplasia. It should not be assumed that this is irrelevant to Britain, as results from an interventional study on vitamin D supplementation during pregnancy in Edinburgh by Cockburn *et al.* (1980) show. Pregnant women received daily supplements of 10 μg of vitamin D2 from the twelfth week of pregnancy. Their plasma calcium concentrations were higher than those in a control group of pregnant women who did not receive a supplement. Infants of the women who had received the supplements had higher plasma calcium and lower plasma phosphorus concentrations on the sixth day of life and a lower

Table 2 Plasma concentrations of calcium (Ca) and inorganic phosphorus (Pi) of 56 patients before treatment for hereditary vitamin D-dependency rickets (VDDR), X-linked hypophosphatemia (XLH) and hypoparathyroid pseudohypoparathyroid conditions (HP/PHP), together with the number of patients with hypoplastic teeth. Data from Nikiforuk and Fraser (1981).

	VDDR	*XLH*	*HP PHP*
Plasma Ca	low	normal	low
Plasma Pi	low	low	high
Rickets	severe	yes	no
No. of patients	10	25	21
No. with hypoplastic permanent teeth	10	0	15
No. with hypoplastic deciduous teeth	1	0	

incidence of hypocalcaemia. Sixty-one of the infants in the trial were examined 'blind' in their third year by a paediatric dentist who observed that fifteen out of thirty-one (48 per cent) of the children of the control group mothers had hypoplastic teeth, compared with only two out of the thirty (7 per cent) of the infants born to mothers in the supplement group. The conclusions of the study were that the control mothers, who were deficient in vitamin D, gave birth to infants more prone to hypocalcaemia and defects of dental enamel.

The importance of hypocalcaemia as a cause of enamel hypoplasia was clearly shown by Nikiforuk and Fraser (1981) in Toronto, Canada, in a series of eloquent investigations involving the thorough examination of 56 patients over a 25 year period. These patients belonged to three categories of disorders of calcium and phosphate homoeostasis. Ten had hereditary vitamin D dependency rickets (VDDR), 25 had x-linked hypophosphatemia (XLH) and 21 belonged to a hypophosphatemia/pseudo-hypophosphatemia group of disorders (HP/PHP). Table 2 indicates that plasma calcium levels were low in the VDDR and the HP/PHP group of children, but normal in the XLH children. Plasma levels of inorganic phosphate, though, were low in the VDDR and XLH groups but high in the HP/PHP group. All these values referred to their condition before treatment. None of the 25 children in the XLH group (which had normal plasma calcium levels) had hypoplastic deciduous or permanent teeth, while most of the 31 patients in the VDDR and HP/PHP groups (who had low plasma calcium levels) had hypoplastic teeth. Thus enamel hypoplasia occurred only in children who had hypocalcaemia, and no relation was observed between enamel hypoplasia and plasma phosphate levels.

Mellanby's belief that vitamin D deficiency was a major factor accounting for the high caries prevalence in temperate climates was based partly upon her knowledge that caries was rare in the tropics where vitamin D levels were expected to be adequate because of high exposure to sunlight. This issue is not completely dead in Northern communities who experience little sunlight for several months of the year. Some schools in Alberta, Canada, have installed 'full spectrum' lighting in classrooms and Hargreaves and Thompson (1989) reported lower caries increment over two years in 31 children who were exposed to this light (with high ultra violet output) compared with 52 children in classrooms with conventional lighting. The differences between the caries increments (0.6 and 2.1 DMFS, respectively) was substantial, and the results of other studies are awaited with interest.

In the Northumberland study (Rugg-Gunn *et al.*, 1984), which related diet of adolescent children to their two-year caries increment, one of the highest correlations observed

was for vitamin D intake. However, the relationships between weight of dietary vitamin D intake and caries increment was only observed in the boys ($r=-0.24$, $p=0.0004$) and not in the girls ($r=+0.01$, $p=0.5$) and because it was not observed in both sexes, the finding was rather dismissed as a quirk, despite the very low p value in the boys. However, if there are enough 'quirks' (which researchers should record), the subject should be investigated further. Mellanby's pioneering work is not yet finished.

The issue of hardness of water is not completely dead either, although very much less important than the fluoride content of water. In America, Mills (1937) and East (1941) reported moderate inverse relationships between water hardness and caries experience of children. Waters with appreciable levels of natural fluoride tended to be hard leading most people to conclude that water hardness is of negligible importance compared with the fluoride content of water. This conclusion seems justified when data presented by Dean, Arnold and Elvove (1942) are subjected to partial correlation analysis (Table 3). The correlation between caries experience and fluoride level is negative and high and is not altered when the level of water hardness is controlled. The negative correlation between caries experience and hardness is weaker and not statistically significant, and little altered when the fluoride content of water is controlled.

However, a South African study by Ockerse (1944) contradicts this conclusion, to the extent that not only did he find high negative correlations between caries and water fluoride levels and between caries and water hardness but by partial correlation analysis he showed that the relation between caries and fluoride was not influenced by differences in water hardness and that the relation between caries and hardness was not influenced by fluoride levels. Whether these dental effects of differing water hardness levels are principally pre-eruptive or post-eruptive is unclear.

Work currently in progress in Lima, South America, by Alvarez, Navia and colleagues from Birmingham, Alabama, and in India by Johansson and others from Umea, Sweden, provides further insights into nutrition and dental caries in young children. Alvarez *et al.* (1988, 1990, 1991) have carried out a series of investigations into nutritional status, times of exfoliation and eruption of teeth, and caries experience, in Peruvian children. Their latest study included a total of 1,481 children aged 1–13 years. The plot of decayed missing and filled deciduous teeth shifted to the right by about 2.5 years in the malnourished children compared with the normal children. The authors concluded that

Table 3 Correlations between mean caries experience (DMFT) and water fluoride concentration, water hardness and water calcium concentration in 21 cities in America. Data from Dean *et al.* (1942).

Bivariate correlations	
DMFT v. fluoride	−0.86*
DMFT v. hardness	−0.30
Fluoride v. hardness	+0.14
Calcium v. hardness**	+0.99*
Partial correlations	
DMFT v. fluoride (controlling for hardness)	−0.86*
DMFT v. hardness (controlling for fluoride)	−0.36

* $p<0.01$.
** For 14 cities only.

malnutrition delayed tooth development, and so affected the age distribution of dental caries, resulting in an apparent increase in caries experience in deciduous teeth at the age of 7 years. They emphasised that this difference in eruption did not explain all the difference in caries experience.

Johansson *et al.* (1992) from Sweden have shown that malnutrition in Indian children can lead to increased risk of caries by affecting salivary glands, so that flow rate is reduced and composition of saliva changed.

The relationship between water fluoride concentration and developmental defects of enamel in two countries of different climate and nutritional status is currently being investigated by Nunn and colleagues from Newcastle upon Tyne. Anecdotal reports suggested that the level of opacities in Sri Lankan children was higher than in English children living in communities with comparable water fluoride levels. Of course, from the publications of Galagan and Vermillion (1957) and others in the USA, 50 years ago, one would expect more fluorosis in high fluoride areas in the warmer climate of Sri Lanka, but it was felt that the observed differences in prevalence and severity of opacities in Sri Lanka and England were larger than would be expected due to differences in water intake alone. Our epidemiological comparisons rather confirm this view (Figure 1). Children aged 12 years, who had lived continuously in areas which received drinking water containing 0.1, 0.5 or 1 ppm fluoride, were examined clinically by one examiner using the DDE index on ten tooth surfaces. Even in areas with very low water fluoride levels, there were more teeth with opacities in Sri Lanka than in England. The differences were greater in the 0.5 and the 1 ppm areas (Nunn, Ekanayake and Rugg-Gunn, 1992).

Fluoride excretion is a reasonable guide to fluoride ingestion for any given age, although the proportion excreted varies with age (Murray, Rugg-Gunn and Jenkins, 1991), and Rugg-Gunn and colleagues have subsequently compared the fluoride output in the urine of children aged 4 years living in the same 1 ppmF areas in Sri Lanka and

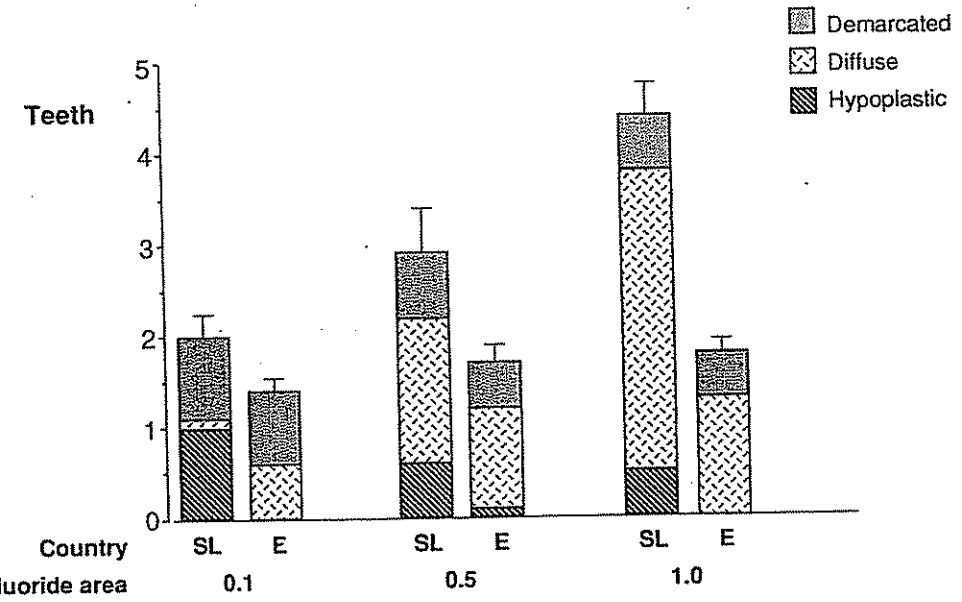

Figure 1 Mean number of teeth per person with developmental defects of enamel in 12-year-old children living in Sri Lanka (SL) or north-east England (E) and receiving drinking water containing either 0.1, 0.5 or 1.0 ppm F. Developmental defects of enamel were classed as demarcated, diffuse or hypoplastic. The bars above each column indicate one standard error. Data from Nunn *et al.* (1992).

North East England as were used in the above opacities studies (Table 4). The amount of fluoride excreted over 24 hours in Sri Lanka was a mean of 0.55 mgF, which was statistically significantly higher than a mean of 0.42 mgF in England. The numbers of children were modest, nevertheless, the authors doubt whether the modest difference in the urinary fluoride excretion (excretion from sweat can be discounted) if it accurately reflects fluoride intake, can adequately explain the large differences observed in prevalence and severity of dental enamel opacities. Whether other nutritional factors play a role in enhancing the prevalence of opacities is worthy of further study. The marked differences in the occurrence of hypoplastic lesions rather than diffuse type lesions (which is the type usually associated with fluoride) may be of relevance. If malnutrition has a significant role in enhancing the development of enamel defects above that caused by fluoride ingestion, then the levels of systemic fluoride appropriate for areas with endemic malnutrition need to be considered carefully. Galagan and Vermillion (1957) considered water fluoride level and air temperature pertinent to the occurrence of fluorosis – to these two might be added 'prevalence of malnutrition'.

Dental erosion

The last area to be considered, albeit very briefly, is dental erosion. Like fluorosis it is related to diet, also like fluorosis it is not yet a public health problem in the United Kingdom. Nearly 20 years ago, Levine (1973) published an article entitled 'Fruit juice erosion, an increasing danger?' while a clinician in Manchester Dental Hospital. Judging by the number of referrals and the clinical impressions of general dental practitioners, this prediction was correct. However, a search through the literature revealed only one epidemiological study of dental erosion and that was in Switzerland (Lussi *et al.*, 1991). The inclusion of both enamel opacities and dental erosion in the 1993 national survey of children's dental health in the United Kingdom is to be welcomed, so that their occurrence, and perhaps public health importance, in this country can be measured.

Professor Holloway was one of the first to assess acid drinks and tooth erosion in a scientific way (Holloway, Mellanby and Stewart, 1958). Amongst the several conclusions from their experiments, they stated that: 'These experiments established quite clearly that

Table 4 Mean and standard deviation of volume of 24 h urine, fluoride concentration in urine, and weight of fluoride excreted in urine in 24 h, in 53 children in Sri Lanka and 44 children in England; children aged 4 years. Values of Student's *t* and probability statistics, testing for differences between countries, are also given.

		Sri Lanka	*England*	*t(p)*
Volume, 24 h urine (ml)	mean	504	449	
	s.d.	198	196	1.38 (0.17)
F concentration (ppm F)	mean	1.19	1.02	
	s.d.	0.63	0.42	1.55 (0.12)
Weight of F excreted in urine over 24 h (mg)	mean	0.55	0.42	
	s.d.	0.30	0.19	2.68 (0.009)[a]

[a] *t*-test for unequal variances.

fruit drinks sold in Great Britain caused erosion of rats' and dogs' teeth *in vivo* and of human and dogs' teeth *in vitro*', and that fluoride reduced erosion.

That was 34 years ago and, to my knowledge, no one has provided any other worthwhile information on the protective role of fluoride in dental erosion. It is time they did, as it is of considerable practical significance. It is necessary sometimes to look back, so as to look forward. In this brief glance backwards at research into nutrition, diet and dental health, it is obvious that Professor Holloway has had a major impact in many aspects, enabling us to look forward to better dental health for all in the future.

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