Public Health Nutrition: 4(2B), 569–591 DOI: 10.1079/PHN2001142

Dietary effects on dental diseases

Aubrey Sheiham*
Department of Epidemiology and Public Health, University College London, 1-19 Torrington Place, London WC1E 6BT, UK

Abstract

Dental caries is a highly prevalent chronic disease and its consequences cause a lot of pain and suffering. Sugars, particularly sucrose, are the most important dietary aetiological cause of caries. Both the frequency of consumption and total amount of sugars is important in the aetiology of caries. '*The evidence establishing sugars as an aetiological factor in dental caries is overwhelming. The foundation of this lies in the multiplicity of studies rather than the power of any one.*'[1]. That statement by the British Nutrition Foundation's Task Force on Oral Health, Diet and Other Factors, sums up the relationship between sugars and caries in Europe. There is no evidence that sugars naturally incorporated in the cellular structure of foods (intrinsic sugars) or lactose in milk or milk products (milk sugars) have adverse effects on health. Foods rich in starch, without the addition of sugars, play a small role in coronal dental caries.

The intake of extrinsic sugars beyond four times a day leads to an increase risk of dental caries. The current dose-response relationship between caries and extrinsic sugars suggests that the sugars levels above 60 g/person/day for teenagers and adults increases the rate of caries. For pre-school and young children the intakes should be proportional to those for teenagers; about 30 g/person/day for pre-school children.

Fluoride, particularly in toothpastes, is a very important preventive agent against dental caries. Toothbrushing without fluorides has little effect on caries. As additional fluoride to that currently available in toothpaste does not appear to be benefiting the teeth of the majority of people, the main strategy to further reduce the levels of caries, is reducing the frequency of sugars intakes in the diet.

Dental erosion rates are considered to be increasing. The aetiology is acids in foods and drinks and to a much lesser extent from regurgitation.

Keywords
Dental caries
Sugars
Erosion
Recommendations

Introduction

There are two major dental conditions, dental caries and periodontal disease. As diet has not been implicated in periodontal disease, this report relates to diet and caries. Factors following eruption of the teeth are much more important than pre-eruptive nutrition in the aetiology of caries[2]. Therefore this review is restricted to the effects of post-eruptive diet on caries. Dental erosion is increasing, so the report will also review the relationship between diet and dental erosion. Throughout the report, sugar refers to sucrose and sugars to total sugars. Most of the reports referred to relate to extrinsic sugars as defined by COMA[3] as 'Sugars not located within the cellular structure of food'. Non milk extrinsic sugars (NMES) are 'Sugars found in confectionery, soft drinks, table sugar. Biscuits, cake, fruit juices, honey and sugars added to recipes.'[3].

The burden of disease

Epidemiological analysis within the EU

Extent of the problem and the cost of treating dental caries

Dental diseases, particularly dental caries, are the most expensive part of the body to treat. Caries is indeed the most expensive human disease in terms of direct costs. For example, the direct costs of caries treatment in Germany was 20.2 billion, CVD 15.4 billion DM, diabetes 2.3 billion DM.[4]. In West Germany, the cost of dental care was 10.3% of the health budget in 1994[5]. The high cost of treatment is directly related to the progressive nature of dental caries. As a higher proportion of older people are retaining there teeth, and they are more susceptible to caries than younger cohorts[6], and they require more

*Corresponding author: Email a.sheiham@ucl.ac.uk

extensive treatment and retreatment of their fillings, the cost of treatment of caries is likely to increase.

Dental pain

Dental pain is mainly due to dental caries. Among a national sample of UK children aged 14–15, 49 per cent had experienced toothache[7] and 40 percent of UK adults had dental pain in the past year[8]. In England, 47 per cent of 8-year olds in North London had experienced toothache[9]. Dental pain caused crying in 17 per cent of children. 7.6 per cent of children had pain in the previous 4 weeks. Among these 45 children, this recent pain resulted in stopping playing in 26 per cent, eating in 73 per cent, sleeping in 31 per cent and in going to school in 11 per cent.

Dental caries in children

Despite the marked declines in caries in the past 30 years, dental caries in pre-school children remains a major dental public health problem in most EU countries. The caries condition of children of immigrants and refugees is particularly poor. Early childhood caries (ECC) affects one in ten of all 3–4 year olds in the UK. A higher likelihood of caries occurred in children given a sweetened comforter. These findings highlight that ECC '… *is a serious public health problem in disadvantaged communities in both developing and industrialized countries* …' [10]. A national survey in Britain indicated that 50% of children in Scotland aged $3\frac{1}{2}$ to $4\frac{1}{2}$ had experienced caries and of children with active decay, 30% had decay which extended into the dental pulp[11].

Marthaler *et al.*[12] in an extensive review reported on the prevalence and severity of dental caries in Europe between 1990 and 1995. The data reported here are mainly from their review. Dental caries in the primary teeth of school aged children aged 5–7 year ranged from 0.9 to 8.5 dmft. National averages below 2.0 occurred in Denmark, Finland, Italy, the Netherlands, Norway and England. Higher levels of decay were reported in Portugal (4.4), Lithuania (4.4), Hungary (3.7) and Scotland (3.0). In some countries the percentage of the caries which was untreated was between 71% (UK)[13–15].

Dental caries is common in 12 year olds. In countries with relatively low DMFT scores, 65% of children had experienced dental caries in their permanent teeth. The Scandinavian countries, England and the Netherlands were the only countries were approximately half the 12 year-olds had no decay experience. The majority of EU countries had DMFT averages below 3.0 at 12 years of age. Nine had DMFT scores above 3.0, the WHO goal for the year 2000. They include Austria, Iceland, Germany, Greece, Israel, Spain, Yugoslavia, Hungary (4.3) and Poland (5.1). The Baltic countries like Latvia (7.7) have high DMF levels. An indicator of the inability of current dental services to cope with the dental caries problem is the relatively high percentages of untreated dental caries lesions. The percentage of caries lesions untreated in 12 year olds was 29% in France, 45% in the UK, 46% in Hungary and 53% in Poland[13–16].

Dental caries in adults

Caries increases after the age of 20 years indicating that it affects children and adults[17]. Adults have caries incidence rates similar to those of children. Increases in caries with increasing age were reported in cohorts of Norwegians. The numbers of decayed and filled teeth increased from 3 at 13 years to 8 at 23 years and to 15 at 33–34 years[6]. Caries has declined and is occurring at later ages. The prevalence of DMFT was high in most young adults in Europe; more than 90% were affected. Caries severity increased with age. In Ireland and in the Netherlands the mean DMFT for 20–24 year olds was 9.5 and 11.3 respectively in 1985–1991. The DMFT in 16–24 year old United Kingdom adults was 10.8, and increased to 16.0 at 25–34 and to 19.0 at 35–44 years. The mean DMFT in EU countries after 1988 varied between 13.4 and 20.8 at 35–44 years. In France the DMFT was 14.6 in Germany 16.3 and in the United Kingdom, 18.9. In fluoridated areas of Ireland the DMFT was 18.9. The WHO classifies DMF levels at 35–44 years as high when the DMF is above 14.0 and very high, at 18.0 or more[18]. Most EU countries fall into these two categories.

Among older people with natural teeth free-living British people aged 65 years and older, the average numbers of teeth with caries experience was 15.5. In Ireland, the DMFT in older people was 26.5 in fluoridated areas[19].

Dental caries in immigrants and refugees

Dental caries levels among most of the young in immigrant groups in Europe are higher than the resident groups. Whereas disease levels are decreasing in the latter, it is increasing, particularly among pre-school children, in the immigrants[20]. There are more than 20 million immigrants and refugees in Europe. They carry a serious dental disease burden in addition to their other problems. The higher caries levels of Asians and other immigrant groups has been attributed to a higher intake of sugar containing drinks, including milk and a higher frequency of sugary products[21].

Root caries

The prevalence of root caries ranged from 7–11% among Finnish adults aged 30–39 years to 27–33% in the oldest group but the mean number of teeth affected was low, 0.32. Higher rates (15–80%) were reported in Sweden with 65-year-old men having an average of 0.9 teeth affected by root caries[17].

Tooth erosion

Dental erosion has been defined as an progressive irreversible loss of dental hard tissue by a chemical process, usually by acids other than those produced by plaque bacteria[2,22]. Erosion can lead to reduction in size of teeth and depending upon the severity and length of exposure, may lead to the total destruction of the dentition[22]. That requires extensive expensive restorative treatment. Rates of dental erosion appear to be increasing. The prevalence of erosion affecting dentine or pulp (severe erosion) increased from 3% in children in Great Britain aged $1\frac{1}{2}$ to $2\frac{1}{2}$ years olds to 13% in $3\frac{1}{2}$ to $4\frac{1}{2}$ years olds[11]. Among 5–6 year olds the prevalence of erosion was 52% in the primary incisors: 25% had erosion involving dentine or pulp and among 11–14 year olds the prevalence in permanent teeth was 28%; in 2% the erosion affected the dentine[23]. Based upon a review of population-based surveys Ten Cate and Imfeld[24] reported that from 8 to 13% of adults had at least one facial surface erosion lesion involving dentine. A further 30 to 43% had between 3.1 and 3.9 teeth affected by occlusal lesions extending into the dentine. The main causes are intrinsic or extrinsic acids. The intrinsic acids are from vomiting related to anorexia, bulimia, and cytostatic drug treatment. The extrinsic causes are dietary acids such as fruit acids, ascorbic and phosphoric acid in frequently consumed acidic fruit juices, squashes, sports drinks and carbonated beverages[22–25]. Many drinks have pH values below 4[22]. Smith and Shaw[26] have drawn attention to the erosive effects of baby fruit juices. Ten Cate and Imfeld[24], in summarising a workshop organised by ILSI stated that '*the potential risk factors for dental erosion are changed lifestyles and eating patterns, with increased consumption of acidic foods and beverages*.'

The current situation relating to dental erosion was outlined in the National Clinical Guidelines and Policy Documents 1999 for Paediatric Dentistry of the Dental Practice Board for England and Wales[27]. The report emphasised that erosion was a oral health problem. The causes were '*All acids, whether from within the body or from external sources, are capable of demineralising tooth tissue, and therefore of causing erosion*.' '*The intrinsic causes include intrinsic acids from gastric reflux and vomiting. Extrinsic causes are soft drinks, and some dry wines and alcopops*.' Although there is increasing evidence of the role of soft drinks in the development of erosion, it is not just drinks that contain acid. There are also other potential dietary sources such as fresh fruit, pickles, and sauces, lactovegetarian foods and yogurt '*Frequency of, rather than total intake may be critical in the erosive process. It is the titratable acidity of the drinks that is more important in causing erosion than just the actual pH*.' '*Another important consideration is that dental erosion is frequently associated with individuals with high standards of oral hygiene …*'[28].

Pathogenic mechanisms with observed relationship across Europe

To answer the question, why do we get caries, the pathophysiology of caries needs to be explained.

Dental caries is a process of enamel and dentine demineralization caused by various acids formed from bacteria in dental plaque. Caries '… *involves dissolution of the minerals of the tooth surface by organic acids formed from the bacterial fermentation of sugars derived from the diet*.'[1]. Saliva is capable of depositing mineral in porous enamel areas demineralized by the acids (remineralization). The deposit of impure hydroxyapaptite crystallites is an important protective property of human saliva. Saliva is always supersaturated with calcium as well as phosphate at pH 7. If a porous lesion is formed in enamel, repair by remineralization always takes place. However, remineralization is a slow process and has to compete with factors causing demineralization. If the remineralization process can effectively compete, repair of the enamel takes place. On the other hand, if the challenge is too great, it is below the critical pH, and demineralization dominates, porosity of enamel increases with lesion progression until finally a carious cavity forms[29]. The rate of demineralization is affected by the concentration of hydrogen ions (pH) at the tooth surface and the length of time, which is related to frequency that the pH of the plaque is reduced below the critical pH. In other words, caries occurs when the balance of dissolution and repair is tipped towards excess dissolution, when demineralization exceeds remineralization[1]. Fluoride enhances remineralization.

Bioactive food components and caries

Caries is caused by acids produced mainly from the interaction of specific bacteria with sugars. So although the bacteria produce the acids and are considered a 'cause' of caries, the bacteria do not produce sufficient acids to demineralize enamel without sugars or sugars in combination with finely ground and heat-treated starch or with cooked starchy foods. As the pathogenic mechanisms of caries involves five main factors, tooth susceptibility, saliva, bacteria, dietary sugars and the time the sugars are in contact with the bacteria, this section of the report will outline the interactions and assess the role of nutrients.

The mouth is unique in the human body in having non-shedding surfaces (teeth) for microbial colonisation. That results in the accumulation of bacterial biofilms especially at stagnant sites around the dentition. Plaque-mediated diseases such as caries occur at sites with a pre-existing normal resident flora[30]. Caries results from imbalances in the resident microflora resulting from an increase of pathogens due to strong selective pressures from a major disturbance to the local habitat. A common feature of

disruptive factors is a change in the nutrient status of the site. Changes to the local environment can disturb the microbial balance found in health and select for organisms associated with caries[30].

Brathall[31] stated that 'Bacterial plaque is the only, immediate, direct factor causing caries. The level of bacterial activity in the plaque is determined by diet (sugars). In addition, the outcome of the bacterial activity in terms of causing damage to the teeth is controlled by several other factors such as fluorides, saliva secretion, buffer capacity, salivary antibodies which collectively are said to represent the resistance to caries development, ie the susceptibility of the host.' The microbiology of caries revolves around important roles of mutans streptococci and S. sobrinus in the bacterial community. The growth of some species of bacteria are controlled by environment, including pH and nutrition. Mutans streptococci will establish on the teeth in the absence of sucrose, probably because of their affinity to adhere to the teeth and fermentation of lactose. The acids produced by mutans streptococci are mainly influenced by the diet (sucrose), the cariogenicity in rats was related to the amount of sucrose in the diet.

Sucrose can enhance production of insoluble plaque matrix polymers by specific bacteria, including mutans streptococci and S. sobrinus. And the growth of those organisms is highly dependent on the presence of fermentable monosaccharides, provided directly by sucrose and glucose and indirectly through the degradation of complex carbohydrates, mediated by host enzymes (amylase) or the combined activities of other oral bacteria degrading plaque matrix or salivary mucins. Excessive exposure to monosaccharides provides a selective pressure *via* pH for rapid growth and retention (matrix), which favours mutans streptococci and S. sobrinus relative to the other acid-producing bacteria. Indirect provision of these monosaccharides while generating acid may not have such an immediate impact, although the slower liberation of monosaccharides may increase the time for acid production. However, excessive use of sucrose would probably give an equivalent period of low pH.

In summary, given sucrose, mutans streptococci and S. sobrinus can begin the caries process quickly, without intermediaries. Nevertheless there is a debate whether there is a specific sucrose-mediated glucan effect on mutans streptococci[32]. The presence of sucrose-specific glucosyltransferases (GTFs) in mutans streptococci led to a presumption of a unique relationship between these bacteria and sucrose. The establishment of mutans streptococci on the tooth surface results in a concentrated accumulation of acids *in situ* during bacterial growth and fermentation of dietary sugars. Sucrose is split into glucose and fructose by mutans streptococci invertase, fructosyltransferase or GTFs. These monosaccharides are easily fermented by most plaque bacteria *via* the glycolytic sequence to produce primarily lactic, but also to alcohol, acetic, and formic acids.

Thus it is clear that bacteria such as mutans streptococci interact with sugars. Regular periods of low pH in plaque select for bacteria like lactobacilli and mutans streptococci because they are more competitive at low pH compared with those organisms associated more with enamel health. Thus it is the low pH from sugars rather than the sugar itself that disrupts the plaque ecology[30]. There is an increase in the levels of mutans streptococci in volunteers who rinsed with low pH buffers[30]. The oral flora would be influenced by the diet, namely, it is a dynamic relationship, and some species would increase and others decrease. Mainly, it is the low pH generated from the diet rather than how much sugar per se, although the two are strongly correlated.

Mutans streptococci ranks highest among the potentially cariogenic bacteria, playing an important role in the initiation and progression of enamel caries[33]. Mutans streptococci have three unique caries-inducing properties; their ability to adhere firmly to tooth surfaces in the presence of sucrose by forming water-insoluble glucan (IG), and, second, to form acids by fermenting various dietary sugars[32]; a key discriminatory property of bacteria such as mutans streptococci and lactobacilli is their ability to grow and metabolise at the low pH values, they generate[30]. The rate at which the bacterial plaque produce acids depends upon the type and numbers of bacteria, and whether the bacteria have been 'primed' by frequent exposure to sugars. The rate at which pH returns to the resting level depends on the amount and duration of sugar exposure, the rate of salivary flow, and the buffering capacity of the saliva[34]. The formation of acids from sucrose by glycolytic activity of a group of streptococci and lactobacilli which are especially cariogenic, leading to a fall in pH at the tooth surface is very well established[35]. The acid response of the plaque to sugary food is altered if it is taken in combination with non-acidogenic foods.

The interaction of sugars and bacteria

Caries is associated with increases in the proportions of acidogenic and acid tolerant bacteria, especially mutans streptococci. These bacteria are able to rapidly metabolise dietary sugars to acid, creating locally a low pH. They grow and metabolise optimally at low pH and under such conditions become more competitive, whereas most bacterial species associated with enamel health are sensitive to acidic conditions[30]. It is indisputable that mutans streptococci's role in caries development is conditional upon a frequent intake of fermentable sugars[33]. They are strongly dependent on dietary carbohydrates[32]. Mutans streptococci are able to rapidly metabolize dietary sugars to acid, creating locally a low pH[30]. High frequency of fermentable carbohydrate intake with regular pH drops favour the proliferation of mutans

streptococci and there is a direct effect of sucrose on the numbers of these micro-organisms in the mouth[33]. Reports on the relation between self-reported sugar consumption and oral loads of mutans streptococci are equivocal. Some studies show only small changes in mutans streptococci counts with decreased sugar intakes whilst others report two- to five-fold reductions after 8 weeks[32,33]. They conclude that '*Individuals with a high risk of caries incidence are, almost by definition, exposed to a cariogenic diet*.' As most oral streptococci, including mutans streptococci, produce little acid from starch[33], the main dietary cause of caries is sugars. Acid production is very rapid with a minimum pH occurring 5–10 minutes following exposure to sugars. The demineralisation of enamel and later dentine is affected by the concentration of hydrogen ions (pH) at the tooth surface. The characteristic form of the pH response in plaque to sugars, plotted against time, is called the Stephan curve. The episodic nature of the Stephan curve indicates that frequency and duration of ingestion of dietary sugars are important aspects of caries[1]. As the pH falls calcium and phosphate ions are lost from the enamel. The drop in pH below 5.4, the critical pH, is sufficient to decalcify enamel and dentine. Glucose and fructose are fermented at a similar rate to sucrose. Most oral streptococci, including mutans streptococci, cannot produce acid from starch[33]. Starch can give rise to falls in pH only if hydrolysed by host amylase and microbial glucosidases.

In summary, sugars particularly sucrose, are the most cariogenic of carbohydrates in animal studies. The high affinity of mutans streptococci for sucrose coupled with the unique uptake of the disaccharide molecule of sucrose and mutans streptococci interaction with dietary carbohydrates particularly sucrose, to form acids. Complex carbohydrates such as starch interacts to a lesser extent with oral bacteria including mutans streptococci and produce less acids than sugars, suggesting that they are not as acidogenic as sucrose.

The influence of fluoride on the sugar/caries relationship

The biochemical mechanisms causing the dissolution of human enamel and dentine indicate that dietary substrates are necessary for caries to occur. Fluoride is the main factor altering the resistance of teeth to acid attack and interacting with sugars in plaque. Fluoride affects tooth structure during tooth development and post-eruptively and reduces caries in three ways:

Reducing and inhibition of dissolution of enamel. Fluoride has a strong affinity for apatite. Because of its small ionic size and its strong electronegativity, fluoride is incorporated into the enamel lattice and/or binds to crystal surfaces. By converting the apatite into fluorapatite, which is more stable than hydoxyapatite, fluoride reduces the dissolution rate of enamel by replacing hydroxyl groups in hydroxyapaptite to form fluorapatite.

Remineralisation is the process by which partly dissolved crystals are induced to grow by precipitation of mineral ions from solution, so that the process of mineral loss is reversed to some extent. Both saliva and plaque fluid are supersaturated at near- neutral pH with respect to hydroyapatite and can thus support remineralisation. At the pH and supersaturation levels found in resting plaque fluid, apatite crystal growth proceeds by formation of intermediate solids, especially octacalcium phosphate (OCP), which then hydrolyses to hydroxyapatite. The hydrolysis of OCP is accelerated and the product of crystal growth is fluorapaptite or fluoro hydroxyapatites (FHA) rather than hydroxyapatite. Thus remineralisation in the presence of fluoride will not only replace lost mineral but will also increase resistance to acids and to later demineralisation.

Effect of fluoride on bacteria. Fluoride affects plaque by altering the ecology of the dental plaque and reducing acid production. The pH fall is reduced in the presence of fluoride.

The main protective action of fluoride is topical, after the teeth have erupted. The inverse relation between fluoride concentration in drinking water and caries is well established. Topical fluoride, either in the form of toothpastes, rinses or varnishes, reduce caries in children by between 20–40%. These findings from over 800 controlled trials shows that fluoride, either in water or in toothpaste, is the most important prophylactic agent against caries[36]. Does fluoride affect the sugar/caries relationship?

Recent studies on diet and caries have been confounded by the widespread use of fluoride toothpastes. Nevertheless Hinds and Gregory[11] in England and Steckson-Blicks and Holm[37] in Sweden showed that the association between snacking and caries experience was only partially negated by the frequent use of fluoride toothpaste. Caries progressed through enamel and dentine in teenagers in a preventive programme which included a range of fluoride regimens[38]. In the two major studies of diet and caries, the use of fluoride toothpaste did not affect the relationship between sugar and caries[39,40]. In a cross-sectional study, Beighton *et al.*[41] on 12 year olds who were using fluoride toothpaste and with a low mean DMFS of 3.05 ± 3.85 and 5.72 ± 5.00, the DMFS scores were significantly related to the number of eating events at which sugar-containing foods or confectionery were consumed. In a study where the use of fluoride toothpaste was controlled, the strong positive relationship between frequency of sugary snacks and caries persisted[42]. The most objective view on the current sugar/caries relationship is Marthaler's[43]. He concluded, after reviewing the literature on declines in caries and associated factors, that '*… within modern societies which are aware and make use of prevention, the relation between sugar consumption and caries activity still exists*'. The main conclusion by Marthaler[43] is '*Recent studies*

have demonstrated that sugar – sucrose as well as other hexoses – continues to be the main threat for the dental health (1) *of the whole populations, some developed and many developing, and* (2) *for the individual in both developed and developing countries,* (3) *in spite of the progress made in using fluorides and improved oral hygiene*'. Marthaler[43] concluded that in Spain, Portugal, Greece, Hungary, Iceland, Poland, Yugoslavia, Albania and Italy the '… *high sugar consumption is still the most important determinant of caries prevalence*.' Although dental caries levels have declined in many European countries, a significant relationship between sugars and caries persists despite the regular widescale use of fluoride toothpaste.

A combination of fluoride and lowered sugar has a synergistic effect on caries reduction. Weaver[44] showed that the caries in 12 year-olds declined by 50% in both South (fluoride) and North Shields (no-fluoride) during wartime because of sugars rationing. In 1943 the DMF in 12-year-olds in North Shields was 4.3 and in 1949 it was 2.4. In South Shields the DMF was 2.4 in 1943 and 1.3 in 1949.

Side-effects of fluoride

There has been growing concern about the increasing problem of dental fluorosis. With the widespread use of fluoridated toothpastes, and accidental ingestion by very young children, in industrialised countries in the past 30 years there are more reports of unacceptable fluorosis of the front teeth[45,46]. Recent studies have reported fluorosis prevalence ranging from 3% to 42% in communities with negligibly fluoridated water and between 45% and 81% in areas with optimal fluoridation[46–52]. The association between fluoride in toothpaste and fluorosis has been demonstrated in children in low water fluoride communities[53,54]. The difference in prevalence of fluorosis in optimally and in negligibly fluoridated communities has narrowed considerably[52].

Some epidemiological characteristics about caries – the nature of caries and its distribution in populations

The progressive nature of caries

As with other chronic diseases, prevention ideally should stop or delay progression of caries for long enough for the severe manifestations of the condition not to manifest. In this section, two aspects of progression of caries are analysed; progression in patho-physiological terms in teeth and in epidemiological terms

Analysing the rates of progression of a disease provides evidence of the extent of the aetiological challenges and the resistance to them. If the challenge is sufficient to cause progression that would suggest that the resistance is not able to overcome the challenge. On an observational and policy level, it is clear that the rates of progression of dental caries have not been sufficiently slowed to allow dental policy makers worldwide to change the health education messages to the public to attend for a regular annual recall visits to diagnose and treat dental caries. In all European countries recall intervals remain annual.

Caries occurs on the occlusal (biting) and the approximal (fronts and backs of teeth). The majority of approximal caries lesions in permanent teeth progress slowly, with an average lesion taking at least 3 years to progress through enamel to dentine. At levels of caries common in EU countries, the rates of progression of caries in people from 11 to 22 years is as follows: the median number of new approximal lesions per 100 tooth surface-years (there are 156 tooth surfaces per person) were 3.9 from no caries to caries halfway through enamel, 5.4, from halfway through enamel to spread into the outer half of dentine, and from broken enamel-dentine border to obvious spread in outer half of dentine, 20.3[38]. The DMFT increased from 3.2 at 12 years to 7.0 at 22 years. The cohort was exposed to comprehensive topical flourides including rinses, fluoride lozenges and varnishes over many years. The authors conclude that '*The slow but continuous progression of approximal caries and the relatively high prevalence of enamel caries at* 21 *suggest that for the approximal surfaces in a long-term perspective, the fluoride supplements simply retard the progression of caries*'[38].

A further indication of the progressive nature of caries is the differing types of surfaces of the teeth affected as the severity of caries increases. At low levels of caries (DMF-S below 10) the main parts of the teeth attacked are the pits and fissures. As the caries attack increases, the disease extends to involve the approximal and smooth surface. The DMF increases with increasing age and so does the extension of the disease to more surfaces. In epidemiological terms, caries progresses with increasing age of groups of people. In all humans studied, the DMFT for children of 6 years is lower than those of 12 years and 18 years. The rates of progression of the disease varies according to the level of disease at the younger age.

Distribution of dental caries in populations

Some claim that at current levels of caries most of the dental caries load in EU countries occurs in a small proportion of the population[55]. Indeed the low prevalence of caries is considered to be a justification for using a high risk strategy limited to the small percentage who have the majority of caries. For example Pollard *et al.*[56] stated that '*This decline (in dental caries) now means that over half the children in Britain never experience dental caries and that* 60–80% *of the decay that does occur is found in* 20% *of the child population*.' This is an inaccurate statement as the possession of a caries-free dentition under the age of 10 is not a good predictor of zero caries incidence thereafter. Fejerskov and Baelum[55] warned against the term caries-free. That indicates

'… *only that no cavities have been found, not that the disease is not present*'.

Caries is considered to be '… *a* '*dichotomous disease*' *affecting mainly those in the lower social classes and other disadvantaged groups*.' Pollard *et al.*[56] claim that there is no persuasive argument to support reducing sugars intakes by the whole population. Such a policy would only benefit the minority with a high caries prevalence[56]. Therefore it is worth outlining the different policies or strategies to control dental caries.

Strategies to control dental caries

Rose[57] divides strategy approaches to control most chronic diseases into two distinct groups. Those aimed at the population and those in which certain sections of the population are identified, either as a group or as individuals, the risk approach. To decide whether to adopt a population or the risk approaches, Rose[57] poses the fundamental question, does a small increase in risk in a large number of individuals generate more cases than a large increase in risk in a few individuals? The criticisms of the high-risk approach is that it is both palliative and temporary. It neither addresses the underlying cause(s) of the problem nor prevents new cases occurring. The pivotal factor that should be used to determine the choice of preventive strategy is the distribution of disease within the population.

How do Rose's principles apply to preventing dental caries? Batchelor[58] tested Rose's concepts on caries distributions and concluded that at low levels of caries, the majority of lesions occur in a minority of individuals. As DMF levels increase beyond DMFT of 1 for any age group, the changes in caries levels are not confined to a group who some define as at 'high risk'. The majority of new caries lesions occur in the group defined at 'low' risk. While those individuals at an initial low DMF score had, on average, a lower annual caries increment than those individuals with a high DMF score, both groups developed caries. Any changes in caries levels are distributed throughout the population: a strategy limited to the 20% of individuals 'at risk' will fail to deal with the majority of the new caries. For example, per 100 7 year-old children, 67 of children had a DMF score of 0 and developed 2.28 lesions over a 4 year. Thus the number of new lesions was 152.7 (2.28 times 67). At the other extreme, although those with an initial DMF score of 7 or greater had a 4-year increment of 4.27, only 2 children per 100 were at this disease level. The number of new lesions would be only 8.5. Those who would be defined at low risk, with a DMF-S score of 0, accounted for 55% of the new lesions. Taking a 'high-risk' approach even if 100% successful would deal with less than 10% of lesions[58].

These studies show that contrary to some claims, caries does not only affect a minority of Europeans. The percentages of children with caries is high. Whereas 40% are caries free at 14 years and caries free levels continues to decline in older groups, in Scotland 79% and in Northern Ireland 85% of 15 year olds had experienced caries. The percentage of caries free young adults is very low and on average 15 teeth are DMFT in dentate people[18].

Major risk factors for dental caries

'The evidence establishing sugars as an aetiological factor in dental caries is overwhelming. The foundation of this lies in the multiplicity of studies rather than the power of any one.'[1]. That statement by the British Nutrition Foundation's Task Force on Oral Health, Diet and Other Factors, sums up the relationship between sugars and caries in Europe. Some claims are made that the causes of caries are multifactorial. 'Saying that dental caries is a multifactorial disease merely reflects the fact that bacteria, as well as sugar and other factors, are needed for the caries to develop. But refined sugars are a necessary factor for the occurrence of the disease. The other factors such as the amount and type of sugar, frequency of sugar intake, age of the dentition, and availability of fluorides are additional to sugars, not alternatives to them'[59]. When looking for causes of pathological states the main emphasis should be in identifying those factors that determine whether or not the disease develops[60]. To get caries you have to have certain types of bacteria present, and people have to eat refined sugars frequently. The first condition is satisfied, as far as we know, in most individuals. The second, however, varies and it is this variation that is responsible for a high or low caries incidence.

There are extensive reviews of the same body of literature on diet and caries[1,2]. All conclude that non-milk extrinsic sugars, particularly sucrose, are the main cause of caries. Nevertheless, some recent reviews have argued that, because of the widespread use of fluoridated toothpastes, the relationship between sugars and caries in children and young adults has declined[61], and they question whether sucrose reduction is important in food and health policy in industrialised countries. Burt and Ismail[62] correctly pointed out that '*total consumption of all sugars does not correlate well with caries experience in populations consuming high amounts of sugars, but it does so better where overall sugar consumption is lower, such as developing countries today*.'

The Committee on Medical Aspects of Food Policy (COMA) on Dietary Sugars and Human Disease[3] and Rugg-Gunn[2] and other expert committees have reviewed the evidence on sugars and dental caries. The positions of most expert committees which have reviewed the evidence on dietary sugars and dental caries is encapsulated in the conclusions of the COMA report on Dietary Reference Values for Food Energy and Nutrients for the United Kingdom[63]. They are; '*There is no evidence that sugars naturally incorporated in the cellular structure of*

foods (intrinsic sugars) or lactose in milk or milk products (milk sugars) have adverse effects on health. Apart from lactose in milk and milk products, extrinsic sugars in the UK, (principally sucrose), contribute to the development of dental caries…. There is evidence from laboratory studies for the potential cariogenicity of fermentable carbohydrates other than sugars, but the epidemiological evidence implicates non-milk extrinsic sugars as the major dietary component contributing to dental caries. Factors other than dietary carbohydrate, in particular fluoridation, are also important in determining the incidence of dental caries.'

Laboratory and epidemiological studies support the relationship between diet and caries. Since the identification by Stephan that the fall in the plaque pH below 5.5 following the ingestion of sugary foods was a central mechanism to caries development, the results of many investigations have confirmed the strong positive association between the frequency and/or quantity of sugars intake and the occurrence of caries[39,40,64,65]. The sugars intakes of children with high and low DMFT showed very large and systematic differences in sugar intake according to the level of caries in a large survey in the USA in 1970. The data on 2514 adolescents shows that there was twice as high intake of sugar-foods in adolescents with high than those with low DMFT. Any increase in sugar-food consumption was associated with a comparable increase in DMFT[66]. These results were not entirely reproduced in later longitudinal studies. There were weaker associations between amount of sugars and caries in the two well-controlled longitudinal studies of diet and caries. Both found only small positive associations between sugar intake and caries[39,40]. In the UK study the correlation between caries increment and weight of daily intake of sugars was +0.143. There were considerable differences in caries increment between the highest and the lowest sugar consumers. Children consuming the most sugar (>163 g/day) developed 5.0 DMFS during the 2 years, 0.9 DMFS per year more than the children (3.2 DMFS over 2 years) who had the lowest sugar intake (<78 g/day)[39]. Both studies used total sugars intake, combining intrinsic and extrinsic sugars. This may have concealed the more important influence of extrinsic sugars[1].

Rose's[57] observations on the difference between determinants of disease occurring in individuals and determinants of populations' health can be applied to the epidemiology of caries and sugars. For example, the correlation between individual cholesterol intake and heart disease is weak whereas the populations' cholesterol levels are significantly associated with their levels of heart disease. Turning to caries and sugar, there is a relatively low correlation between an individual's sugars intake and caries increment[39]. But in comparisons between high- and low-consumption populations, associations between sugar consumption and the incidence of caries are high ($r = +0.7$ to 0.8)[67,68]. Thus, although dietary sugar is the main determinant of a population's incidence of caries, measures of sugar intake in industrialised countries generally fail to identify high risk individuals; the level of consumption is too high, and moreover the variation in sugar consumption within populations is too low, to show any measurable effect on caries incidence[69,70]. Burt *et al.*[40] pointed out that some of the older studies have failed to demonstrate a correlation between amount or frequency of sugar intake and dental caries activity. Marthaler[43] explained the 'failures' by the fact that the samples were too small. '*Consequently, the power of the experiment was insufficient to level out inter-individual variation.*' Indeed, Burt and Szpunar[71] concede that in their study[40] the low relative risks of caries in higher sugar consumers was the small variances in the patterns of both diet and caries and that a substantial increase in either the number of participants or the length of the study would have been required to establish clearer relationships. In addition some of the older studies had obvious methodological weaknesses. The fact that there were relatively small variations between subjects' intakes of sugar in most of the longitudinal studies[71] must be considered when assessing those studies. They illustrate a basic principle in epidemiology, namely that if all people are exposed to cigarette smoke then the relation between smoking and cancer will not be apparent[57].

Sucrose consumption was associated with the trends in the increase and decline of caries in Japan and in Britain. Miyazaki and Morimoto[72] (1996) reported a positive significant relationship of 0.91 between the DMFT in 12-year-olds and per capita sugar consumption per year in Japan between 1957 and 1987. The market share of fluoridated toothpastes was 10% until 1986 and has increased since then. Downer[68,73] used data from 13 extensive nationally based point prevalence dental surveys of British 5 and 12 year olds. The sucrose data from both sugar intake figures in the COMA[8] report and from the Sugar Bureau. Neither included glucose, honeys, and non-food use and wastage. The Spearman correlation between sucrose consumption and mean dmft the populations of 5-year-old was 0.62 and for DMFT of 12 year olds, 0.84. The relationship between sucrose availability and mean DMFT at 12 years was linear. These two, detailed analysis based upon well-defined caries diagnosis and sugar available for consumption, puts in perspective the relationship between national data on caries and sugars.

In an ecological study using data from 47 countries, Sreebny[67] found a correlation coefficients for sugar supplies and dmft of 0.31 and 0.72 for DMFT. Marthaler[43] calculated from Sreebny's data that for each 25 g of sugar per day, one tooth per child would become DMF. In the 47 countries studied there was a correlation of $r = 0.72$ between sugar supply per capita per day and caries at 12 years of age. In statistical terms a correlation of 0.72 means that 52% of the variation between the reported

data on caries are explained by the variations of the average sugar supply[43]. On the other hand Woodward and Walker[74] using DMFT data for 12 year-olds from 90 countries, 61 developing and 29 developed, found that the relationship between sugar and DMFT was not significant. The shortcomings of their interpretations have been highlighted by Nadanovsky[75] who argued that the reason why the association between sugars and caries was not high using ecological data, was that all the industrialised countries have such high levels of sugars consumption that changing sugars intake by a few kilograms per year does not affect the caries attack. Nevertheless over 28% of the variation in DMFT was explained by sugars in Woodward and Walker's study[74]. When problems of comparing DMFT derived from different examiners are considered and the inaccuracy of sugar consumption data, and the fact that children up to the age of 12 are only one fifth of the total population so the sugar consumption is dominated by those over 12 years old and that the dental data were not from representative samples, the important distorting effects of confounders such as availability of fluoridated toothpaste and frequency of sugar intake, then a 28 coefficient of determination is very impressive indeed. The data from Woodward and Walker highlights an impressive ecological association between sugar and caries. Their data strongly reinforce the view of a 'safe' level of sugar. Almost all countries with sugar levels below 18.25 kilograms/head/year had DMFT below 3.0 at 12 years (23 out of 26 countries). Only half the countries (36 out of 64) with sugar levels of 18.25 kg or more had DMFTs below 3.0. When the 10 kg/person/year is used the difference is even more striking. Over 78% of countries with sugar levels below 10 kg had a DMFT below 2.0. On the other hand only 30% of the countries with sugar consumption above 10 kg had a mean DMFT below 2.0 at 12 years. Thus the caries in populations is much lower when sugars consumption is below 18 kg/person/year.

Ruxton, Garceau and Cottrell[76] from The Sugar Bureau used the data from Woodward and Walker[74] and Sreebny's[67] studies to assess whether there was a relationship between declines in caries and declines in sugar supply in 67 countries between 1982 and 1994. They used a simple scatter plot and no statistical analysis to control for confounding factors. Their plot showed that whereas the DMFT decreased in 18 countries which had declines in sugar supply, in 25 countries dental caries declined despite increases in sugar supplies. In three countries sugars declined and caries increased. However, in a further 18 countries caries levels increased with increases in sugar supplies. So, overall the relationships between sugar supply and increased caries was supported in 36 countries and not in 28[76]. Considering the limitations of analytical methods used and the reliability of the data on caries and sugar supply in this analysis, and the fact that for 17 countries the data points around the axis were within 1 DMFT and 10 g/person/day, the conclusions are questionable that there are no reliable information for setting population-based targets for sucrose or sugars in general.

Relationship between frequency and amount of sugar intake and caries

There is a debate about whether frequency or quantity of intake of sugars is more important in causing caries. The discussion is complicated by the fact that the two are highly associated in human diets[2]. Evidence from numerous studies suggests that caries rates in humans are related to the amount of sugar consumed and the frequency of eating between-meal sugary products. COMA[63] stated: '*In free-living people these three variables, mass, concentration and frequency, are all closely and positively related to each other.*' A WHO expert committee on Diet, Nutrition & Prevention of Chronic Diseases[77] concluded that '*Numerous epidemiological studies conducted at the population level suggest that there is a direct relationship between the quantity and frequency of sucrose consumption and the development of caries.*'[77]. Despite those conclusions most of the emphasis on the sugar/caries relationship has been on the frequency of sugar intake. There is conclusive evidence of a high correlation between the frequency and the amount of sugar intake. Studies indicate that when more sugar is consumed, the frequency increases. The correlation between frequency of sugary foods of intake per day and weight consumed per day, by children aged 12 to 14 years was +0.77[2,39]. All the correlations for a number of food groups were above +0.75, showing that as the amount of sugary foods consumed per day increased, the frequency of intake also increased (Table 1)[2,39]. The correlations were higher for the amount and frequency of all sugared drinks (0.86). Similar high correlations for amount and frequency of drinks between meals (+0.97) was reported by Ismail *et al.*[78] for American children and for frequency and total sucrose intake for South African Black people, Indians and Whites[79]. The correlations were between +0.78 and 0.84. Similar correlations

Table 1 Correlations between frequency and weight of intake of dietary items which are high in sugars, observed in 405 English children aged 11–14 years. All correlations are positive and significant[39]

Sweets	0.74
Confectionery	0.77
Chocolate	0.85
Biscuits and cake	0.80
Biscuits, cake, puddings	0.71
Sweet puddings	0.86
Sugared tea	0.98
Other hot drinks	0.93
Sugared drinks	0.86
Sugared cordials	0.79
All foods and drinks	0.32
All foods with >10% sugars	0.59

between frequency of sugars was reported by Rodrigues[65]. There was a highly significant relationship between daily frequency of sugar intake and the daily weight of intake at nursery and caries increment, after adjusting for all the other variables. In addition, children with a frequency of sugar consumption of 4–5 times per day at nursery were 6 times more likely to develop high levels of caries over one year, compared to those having the lowest frequency[80,81]. Daily frequency of sugar intake at nursery showed a dose-response trend with the risk of having high caries increment.

Ismail *et al.*[78] reported a significantly higher risk of caries in persons aged 9 to 29 years for both the amount and frequency of soft drinks consumed between meals. Those consuming 450 grams/day of soft drinks between meals were 1.86 more times likely to have a high DMFT. The odds ratios increased from 1.28 for those drinking soft drinks once a day between meals, to 1.87 for twice and 2.79 three or more times a day. Ismail *et al.*[78] caution against considering sugary drinks less damaging to teeth than sticky sugary foods.

At frequencies of intake of sweets and between meal sugars which are common in Europe at present there are statistically significant relations between frequency of sugars intake and caries. Rugg-Gunn *et al.*[39] found higher correlations between caries increments and total sugar intake than between frequency of sugar intake. Szpunar *et al.*[82] confirmed the association between amount but not with frequency. They found that children whose energy intake from sugars was one standard deviation above the mean had twice the risk of developing approximal caries relative to those with one standard deviation below the mean.

The two large longitudinal studies on caries and sugars[39,40] were conducted on children with relatively high average intake of sugars of about 100 g/per/day (i.e. 36.5 kg/year). As the study by Burt *et al.*[40] is often mentioned, a more detailed analysis of their study is reported here. In the USA, Burt *et al.*[40] showed that sugar intake, especially sugary snacks were positively correlated with caries. The initial and final DMFS differed between the children grouped by sugar intake. Children with the lowest sugar intake of sugar from all sources (109 g), had a final DMFS of 5.86, whereas those with the highest sugar intake had a DMFS of 8.39. The small difference in age at baseline cannot explain the difference of 2.51 DMFS as the average increment of new caries per year was equal or less than 0.23 DMFS. Marthaler[43] has pointed out that there were not many children in the study with low sugars intake and there was only limited variability in natural and added sugar of the food consumed by the children. That would give a low correlation. In addition, the ratio, 175/109 g is 1.6 and the corresponding DMFS ratio 8.16/5.86 is 1.4. This agrees fairly well with regression line in Sreebny's[67] study in which passes through the origin. Sreebny[67] had shown a strong positive correlation of $r = 0.72$ between sugar supplies and dental caries.

Most recent studies have found larger associations between frequency than amount of sugars ingested and caries. The dmft in young children who were fed four sweetened snacks and drinks a day was 1.67 compared to 1.25 in those fed them once a day[42]. The British National Diet and Nutrition Survey assessed the relation between diet and dental caries in 1500 preschool children[11]; 22% of children with low intakes of confectionery had caries and 40% of children with a high intake. Grindefjord *et al.*[83,84] reported a significant relation between caries and intake of sweets and with consumption of sugar-containing beverages in a longitudinal study of 3 year olds. Holbrook *et al.*[85] reported a mean dmfs of 10.7 in the 56% of children who consumed sugars 4 or more times a day compared to dmfs 2.6 in those consuming sugars less frequently. They found a threshold effect. Above the threshold of ≥4 sugar intakes per day or ≥3 between meal snacks, the levels of caries rose markedly. In an earlier cross-sectional study on 4 year-old urban children Holbrook *et al.*[86] reported a doubling of the dmft in children with intakes of sugar of more than 30 times a week, which is comparable to ≥4 sugar intakes per day. Frequency of sugars intake was also important in teenagers[70]. Between meal sugar consumption and frequency of candy consumption were significantly related to approximal caries. The r^2 ranged from 0.17 to 0.32 for the significant relationships. Bjarnason *et al.*[87] in a detailed dietary study of 12–13 year-olds in Iceland showed that there was a higher incidence of caries surfaces with frequent intake of buns and cakes with meals and confectionery. These findings were confirmed in a 3 year longitudinal study of Swedish 15–18 year olds[88]. The correlation between sweet consumption and the incidence of posterior approximal caries was 0.25. The r value increased to 0.70 in children with poor cleanliness and considerable dental plaque and low salivary flow. They stated that the '… *explanatory value of about* 49%,… *is remarkable and seems to be the highest value reported for any caries-etiologic factor in observational studies*'[89]. The authors concluded that '… *consumption of sweets should still be considered an important caries-related factor* …'[89]. Recent investigations in the Netherlands show a strong relation between the consumption of sweets and caries experience in school children[90]. Jamel *et al.*[91] reported a strong positive relationship between numbers of cups of sweet tea consumed per day and caries (+0.43 for urban group). The correlation between weight of sugar in tea and DMF was +0.71 for rural groups. Caries levels were related to preference for sweetness ($r = +0.58$). A study on 6014, 14-year-olds in 20 districts in England reported a highly significant relationship between consumption of sugared drinks and carbonated drinks and caries (Jones *et al.*[92]). Children adding two or more teaspoons of sugar to hot

drinks were 1.87 times more likely to have caries than those who added less than two teaspoons[92]. Kleemola-Kujala and Rasanen[93] reported that with increasing total sugar consumption the risk of caries increased significantly only when children did not clean their teeth properly.

Some studies did not find a relationship between frequency of sugary intakes and caries[94]. Bergendal and Hamp[95] did not find a relationship between diet and caries in 95 teenagers but the numbers of subjects in subgroups was too small for rigorous analysis.

The findings from the studies reviewed here suggest that both the frequency of consumption and total amount of sugars are important in the aetiology of caries because frequency of intake and amount of sugars ingested are closely related. An increase in frequency of sugary intakes of more than 4 per day increased the risk of caries.

The influence on caries of different carbohydrates

Oral bacteria utilize sucrose, glucose, fructose and other simple sugars to produce organic acids in sufficient concentrations to lower the pH of plaque to levels that may result is demineralization of enamel. It is only from sucrose that most bacteria are able to synthesize both soluble and insoluble extracellular polymers which facilitate attachment of bacteria, especially mutans streptococcus, to it. Unlike other disaccharides, sucrose can serve directly as a gycosyl donor in the synthesis of extracellular polymers. Sucrose has been shown to be more cariogenic than monosaccharides and other disaccharides in animal experiments in rats infected with mutans streptoccoci.

Three trials on human caries have compared sucrose with other sugars. In the Turku experiment in which xylitol, sucrose and fructose were compared, apart from the lower effects of xylitol, the findings were indecisive; the cariogenicity of sucrose and fructose were similar[96]. Whilst some aspects of the Turku studies have been criticized[97], the differences in caries between the xylitol and sucrose and fructose are considerable. Xylitol was the only sugar that the xylitol group used. Their consumption of starch was not controlled.

A study compared a normal sucrose diet with one where invert sugar completely replaced sucrose[98]. Children eating invert sugar had a lower level of caries. Studies on sorbitol[99] and hydrogenated glucose syrup (Lycasin) suggest that they are non-cariogenic[100].

The role of dietary starch

After an extensive review on the relationship between starchy foods caries Rugg-Gunn[2,101] and Rugg-Gunn and Nunn[102] concluded that:

- Cooked staple starchy foods such as rice, potatoes, and bread are of low cariogenicity in humans.
- The cariogenicity of uncooked starch is very low but, since this is seldom eaten by humans, this finding is of little relevance.
- Finely ground and heat-treated starch can cause dental caries, but the amount of caries is less than caused by sugars.
- The addition of sugar increases the cariogenicity of cooked starchy foods. Foods containing cooked starch plus substantial amounts of sucrose, appear to be as cariogenic as a similar quantity of sucrose.

Similarly the COMA panel on Dietary Sugars and Human Disease[3] concluded that 'Simple starchy foods, intrinsic sugars in whole fruit and milk sugars are negligible causes of dental caries. Non sugar bulk and intense sweetners are non cariogenic or virtually so.'[3]. Therefore the panel recommended that: 'In order to reduce the risk of dental caries, the Panel recommends that consumption of NMES by the population should be decreased. These sugars should be replaced by fresh fruit, vegetables and starchy foods..'[3]. And a later COMA on Dietary Reference Values[62] concluded that 'There is no evidence that intrinsic sugars or milk sugars have adverse effects on health.'[62]. Furthermore a WHO expert committee on Diet, Nutrition and Prevention of Chronic Diseases concluded that '… an extensive review of evidence showed that cooked staple starch foods such as rice, potatoes and bread appear to be of low cariogenicity'[77].

Less refined starchy foods may have properties which help to protect teeth from dental caries. These properties, include: (a) a fibre content so that the food has to be chewed vigorously which aids removal of the food from the mouth and increases salivary flow thus raising plaque pH, and (b) possible protective factors – mainly organic phosphates (almost exclusively phytate) – which may protect the teeth from dissolution.

Information on the potential cariogenicity of a carbohydrate can be obtained from a range of experiments including: incubation studies, plaque pH studies, enamel slab studies, animal experiments, human epidemiological studies and clinical trials. The cariogenic potential of a carbohydrate can be judged using information from these experiments.

First, plaque pH experiments. It is important to realise that these investigate acidogenicity not cariogenicity. Certain 'protective factors' in starchy foods would not affect acidogenicity, but would decrease cariogenicity since they affect solubility not acid production. Almost all of the plaque pH experiments investigating starch either use the sampling method or the indwelling glass electrode method of measuring plaque pH. The sampling method has tended to indicate that cooked starch, or starchy foods, are less acidogenic than sugar or high sugar foods and that uncooked starches are virtually non-acidogenic. On the other hand, indwelling glass electrode experiments show that starch depresses plaque pH below the critical pH 5.5. Whether this accurately reflects what

occurs naturally in humans is open to question. Indwelling glass electrodes '*tend to give an all-or-nothing response to foods – any carbohydrate-containing food leading to a maximum drop in pH*'[103]. Indeed, Edgar[103] considers that '*the pH response seen with glass electrodes might be hyper-responsive.*' '*This feature makes the application of the method to evaluating relative cariogenicity of snack foods difficult, as bread, judged to be low relative cariogenicity by other methods, appear highly cariogenic, and the technique is mainly used to verify the low cariogenicity of some sugar substitutes*' [103]. These facts should be taken into consideration when assessing the claim made by Edgar[104] that after reviewing the same data '*… it would appear that while fresh fruits (as consumed in a normal diet) do not contribute detectably to caries, they exhibit properties which do not exonerate them in terms of cariogenic potential were they to be consumed at a frequency similar to other foods which contribute to the cariogenic load*'. Edgar's consideration that the cariogenic potential of fresh fruits, if eaten as often as sugars would be similar to other foods, is conjecture and not based upon evidence.

Enamel slab experiments are a little closer to the human situation since demineralization and remineralization usually occur in vivo. Experiments which have investigated the cariogenicity of starch have indicated that cooked starch is about one quarter to a half as cariogenic as sucrose[105,106] but mixtures of cooked starch and sucrose are more cariogenic than sucrose alone[107]. Many animal experiments have been undertaken with rather variable results. Raw starches appear to have very low cariogenicity while cooked starches cause some dental caries; less than that caused by sucrose. Mixtures of starch and sucrose cause more dental caries than starch alone and the amount of dental caries was positively related to the amount of sugar in the mixture. The degree of processing of starch in manufactured foods influences the development of dental caries in rats, due to the partial hydrolysis of starch (for example in fried potato chips and extruded starchy snack foods)[102]. Cooked starch causes about half the caries caused by sucrose in rats[106] but mixtures of cooked starch and sucrose are more cariogenic than sucrose alone[107]. Starch (−0.06) and lactose (0.01) were not correlated to caries in rats whereas glucose (0.43), reducing sugars (0.30) and sucrose (0.18) were highly significantly related[108]. In an extensive series of studies on monkeys in captivity were fed a range of diets Cohen [109,110] found that '*… in all the experiments I have carried out I have never found it possible to induce caries in monkeys without the addition of sucrose to the diet.*'. Lehner, Challacombe and Caldwell[111] induced caries in the Rhesus monkey. Carious cavities started developing in deciduous teeth within 4–8 weeks on a diet with sugars and increased to a maximum at week 72.

Animal experiments are useful in giving some indication of the cariogenicity of foods in humans, but caution in their interpretation is necessary. Although laboratory studies show that starchy foods can reduce pH of plaque[112], epidemiological studies suggest that starches are of low cariogenicity in humans.

Human observational and intervention studies provide the most valid evidence of cariogenicity. On a world-wide scale, whereas there is a strong positive correlation between availability of sugar and dental caries, no such relation is seen for starch availability and caries. People eating low sugar, high starch diets tend to have very low levels of caries. They are the only types of study which actually record the development of caries in people and as such should provide the most valid estimate of cariogenicity. For millenia starchy foods were the staple diet of humans with there was very little caries. '*Even now, in countries consuming high starch, low sugar diets, caries remains low. It appears that only when sugar consumption increases does caries increase. Starchy foods have become more processed – flour is heat treated and finely ground, which breaks down the starch granules and some of the long-chain starch molecules- which may make them more cariogenic. Frequency of eating may have increased also (although there is no evidence for this), but it is unlikely that these aspects explain the great rise in caries which has occurred in developed countries – the rise in consumption of sugars remains by far the most reasonable explanation.*'[102]. In the Hopewood House study, where children were fed a lactovegetarian diet with a little molasses and honey but high levels of starch, children had one tenth the caries compared to non-institutional populations. The children's oral hygiene was poor and they had no fluoride[113]. In the Turku study[96], the participants continued to eat starch, yet the xylitol group developed very little caries. These studies corroborate the findings from studies of people with hereditary fructose intolerance (HFI). Indeed these studies provide evidence of a direct link between sugar sucrose ingestion and dental caries and the relatively low cariogenicity of starch. Persons with HFI, who could not tolerate sucrose or fructose, had strikingly reduced dental caries experience[114]. In a longitudinal study of caries and diet in adolescents, Rugg-Gunn *et al.*[115] found no correlation between starch consumption and caries when controlling for sugar. Children with high starch/low sugars intake had lower caries increments than children with low starch/high sugars intake. Further evidence on the low cariogenicity of starch comes from wartime dietary restriction. Dietary restriction in Norway and Japan resulted in an increased consumption of starch, unrefined flour and a decreased consumption of fat, meat, sugar and flour of low extraction. The caries rates decreased during wartime and increased sharply after the rise of sugar in the post-war diets[116,117].

Further evidence of the relative importance of sugars in

caries compared to starches come from studies on the effects of rationing during wartime. Weaver[44] reported a halving of the caries levels between 1943 and 1949 in 12 year olds living in North and South Shields. The main decrease during that period was in sugar consumption and not starch. The Weaver study highlights an important finding. Namely, that there is a synergistic effect of combining decreasing sugar and optimum fluoride. In the low fluoride North Shields, caries declined from 4.3 to 2.4 whereas in fluoride rich South Shields the caries level in 1943 (2.4) was half that in North Shields because of fluoride. Nevertheless, there was a further decline to 1.3 after sugar rationing Weaver[44]. Similar results were reported by Künzel and Fischer[118]. They showed that the beneficial effects of water fluoridation varied by levels of sugars consumption. These studies demonstrate that dental caries can be reduced to low levels by the combined effects of fluoride and low sugars.

Zeisenitz and Edmondson[119] claim, without presenting any evidence, that caries rates increased in developing countries such as Nigeria and China because the frequency of fermentable carbohydrates, starches and sugars, have increased. Chinese and Vietnamese have eaten cooked starch for many years, yet their caries rates were low[120,121] and Ethiopians and South American Indians who eat cooked starches such as wheat, rice, maize and quinoa have low caries rates[121]. There are some well controlled studies showing that there is a low caries level despite frequent consumption of cooked starches with low sugars intakes. One of the best studies of increases in dental caries with change of diet is the Tristan da Cunhans[122]. Prior to 1940 the diet was low in sugar but they did eat cakes, bread and biscuits. Yet the caries rates in children and adults was very low. That shows that in modern groups eating cooked starch relatively frequently, the caries levels were low. Similarly, as stated earlier, Hopewood House children consumed high levels of cooked starches frequently and yet they had low caries rates[113]. The Turku study showed very clearly that low increments of caries occur when sugars were replaced with Xylitol. No changes in consumption of cooked starch was introduced. All the foods consumed by the participants were supplied by the study team[96] yet the xylitol group developed very little caries.

Workers in the confectionery industry have higher caries rates than other workers. The DMFT of confectionery workers was 15.6 compared to 9.1 in comparable textile workers[123]. The caries rates in the confectionery workers increased more, the longer they worked in the industry. Similar results have been reported in Japan[124] and Denmark[125].

The most convincing evidence on the low cariogenicity of cooked starches in contemporary humans are the data from 47 countries assessing the correlations between dental caries experience, sugar consumption, and starch consumption. The correlations between caries and cereal were reduced to near zero when sugar was controlled for, indicating that cereal availability did not affect caries experience. The statistically insignificant partial correlations, after controlling for sugars consumption in 47 countries were −0.03 and −0.13, between total cereals, measured as calories per day or percentage of total calories; 0.05 and 0.03 for wheat and −0.24 and −0.26 for maize[2]. The strong positive relation between caries experience and sugar availability was unaffected by standardizing the data on cereal availability, thus indicating that sugar availability had a real effect on caries experience[2,67,126]. Similarly, in a longitudinal study of caries and diet in British adolescents, Rugg-Gunn *et al.*[115] found no correlation between starch consumption and caries when controlling for sugar. Children with high starch/low sugars intake had lower caries increments than children with low starch/high sugars intake.

Evidence shows that milk sugars exhibit low cariogenicity and do not pose a threat to dental health, therefore the extensive evidence that suggests that dental caries is positively related to the amount of sugars in the diet and the frequency of their consumption largely applies to non-milk extrinsic sugars.

Maltodextrins and glucose syrups

Research indicates that maltodextrins and glucose syrups are cariogenic, however, initial studies on some synthetic oligosaccharides have suggested reduced cariogenicity compared to sucrose[127].

Refined cereals

Because sugars and refined cereals are usually associated in diets it is difficult to separate the effects of the two. Sreebny[126] found a positive correlation of 0.45 between availability of wheat and the prevalence of caries in 47 countries. When Rugg-Gunn[2] reanalysed the same data using partial correlation analysis, on the removal of sugar the correlation disappeared. However, when the influence of wheat was removed, the correlation between sugar consumption and caries levels only reduced from 0.70 to 0.60, indicating that sugar plays a much greater role in the aetiology of caries than the availability of wheat[1].

Fruit

Fruit is sometimes implicated as a cause of caries. '… in experimental conditions, with the fruit being a major dietary constituent, fruits may cause caries, however, as consumed as part of the mixed human diet there is no evidence to support its cariogenicity.'[127]. Observations from human studies show a negative relationship between fruit consumption and the incidence of dental caries, which supports the view that intrinsic sugars are of low cariogenicity. Clancy *et al.*[128] found a negative correlation between DMFT and frequency of intake of apples, fruit juice and sugarless gum. And Rugg-Gunn

et al.[39] found no correlation between intake of apples or other fruit and dental caries increment. Grobler and Blignaut[129] found that a high intake of apples or grapes was related to higher caries in adult farm workers. However, most of what they claimed was caries should be queried as 20 of the 24 DMFT teeth were missing. The cause of the loss of such a high number of teeth was not ascertained.

In summary the conclusions by Rugg-Gunn[2,101] mentioned above are valid. Starches and fruit have no or very low cariogenicity.

Fluoride and dental caries

The use of fluoride is the most effective tested method of controlling dental caries. Fluoride can be ingested from water, tea, and a range of foods[2]. A common non-dietary source of fluoride in industrialised countries is toothpaste. Fluoride in toothpaste is considered to be the most important reason for the decline in caries in Europe[130]. Despite the reductions in caries and the widescale availability of fluoride in toothpaste and other vehicles, caries persists and progresses. The benefits of fluoride must be weighed against the disbenefits. In many industrialised countries socially unacceptable levels of enamel fluorosis with discolouration of the front teeth is increasing even in communities with no artificial water fluoridation. The main source of the fluoride is from ingested fluoridated toothpaste. No costings are available on the treatment of the discolouration. As additional fluoride to that currently available in toothpaste does not appear to be benefiting the teeth of the majority of children, the main strategy to further reduce the levels of caries, is reducing the sugars levels in the diet.

Oral cleanliness and dental caries

There is a widely repeated premise that dental caries occurs only after plaque has accumulated on susceptible tooth surfaces in individuals who eat sugar frequently[131]. But the relationship between toothbrushing and oral cleanliness on the one hand and dental caries on the other is equivocal[132]. Bellini, Arneberg and von der Fehr[132] concluded that toothbrushing habits had a questionable effect on caries.

The position regarding toothbrushing and caries is summed up by the expert group advising the Health Education Authority in England. Here is what they recommend: '*Although caries cannot develop without the presence of plaque, plaque removal by toothbrushing cannot in itself be advocated for caries prevention. Normal brushing inevitably leaves some plaque in fissures and other stagnation sites where caries occurs, and plaque rapidly begins to reform on cleaned tooth surfaces. While toothbrushing is important for maintaining gingival health, numerous studies have failed to establish a clear association between toothbrushing and caries incidence. However, brushing with a fluoride toothpaste is the most important method of delivering fluoride to the tooth surface. Other suggested methods for plaque removal such as eating fibrous foods including apples and carrots are ineffective. Plaque can be suppressed using an antiseptic, but its acceptability for public use and its effectiveness for caries control have not been established.*'[133].

The relative ineffectiveness of toothbrushing in preventing caries is very clear. In practical terms, it is physically impossible for the toothbrush bristles to remove bacterial plaque from the depths of fissures and pits and from below the contact points between the teeth. On the other hand there are many population studies where populations with poor oral cleanliness and abundant dental plaque have low levels of caries. For example, in the Hopewood House study, the children had abundant plaque and very low levels of caries as long as only small amounts of sugars were eaten[134]. The relationship between oral cleanliness and caries is summarised by Sutcliffe[131]. After an extensive review he concluded that:

- '*Although tooth cleaning with unmedicated agents may be expected to reduce caries experience, the lack of consistent epidemiological corroboration of the relationship has led to questioning of the value of oral hygiene practices against caries.*
- *Relatively few controlled prospective studies have been undertaken and the results point towards a weak positive association between plaque and caries.*
- *Tooth-brushing with fluoridated dentifrices has been shown to be an effective caries preventive measure. The effectiveness of fluoride toothpastes has been shown to improve with increased brushing frequency and if the minimum amount of water is used to rinse after brushing.*'

Secular trends in EU dental caries patterns and projections of future caries

Dental caries has decreased in children and young adults in industrialized countries. For example, in England and Wales in 1973, 65% of 8 year olds had experienced caries; by 1993 the figure had dropped to 17%. In 12 year olds the mean number of DMF teeth decreased from 2.9 in 1983 to 1.2 in 1993[73]. Similar changes have occurred in most industrialized countries. As stated earlier, the different parts of teeth, pits and fissures mainly on biting surfaces and fronts and backs (approximal) of teeth differ in their susceptibility to caries. The greatest change in prevalence of caries has occurred in approximal tooth surfaces resulting in many children having caries confined to the pit and fissured surfaces of first molars. As caries prevalence falls, caries on the least susceptible surfaces (approximal and smooth surfaces) decreases by the greatest proportion, while the most susceptible surfaces (pits and fissures) have the smallest reductions[135].

The consensus of expert analyses of the decline, agreed by 55 experts[130], is that the widespread use of fluoride, especially fluoride in toothpastes, was the main factor. Nadanovsky and Sheiham[135,136] found that all the countries where substantial declines in 12 year-old DMF-T occurred also had improvements in several general health indicators.

Changes in sugars consumption are frequently dismissed as having contributed to the declines[61]. That viewpoint, which was not based upon a scientific analysis of data, was challenged by Downer[73] who showed that the rise and fall in caries in children in England and Wales '*… was the concurrent increase and reduction in the sugar challenge, mitigated after the early 1970s by the preventive effect of fluoride toothpaste*'[68]. Downer[68,73] has presented detailed evidence that there was a high correlation ($r = +0.85$) between sucrose consumption and caries levels in 12-year-olds in the United Kingdom and that sucrose made an significant contribution to the trends in caries over the past 50 years. There was a close correspondence between sucrose availability and caries until around 1970. After 1970, caries levels fell rapidly with the introduction of fluoride toothpaste[73]. König[138] argues that as caries has declined and sugar supplies have remained the same in the Netherlands, sugars are not a cause of caries. That is equivalent to concluding that if lung cancer decreased and smoking did not, then smoking is not a cause of cancer. His conclusion does not address the fact that dental caries does not occur without sugars. Despite König's claim that sugar supplies have been the same in the Netherlands, they have declined. They decreased from 42.5 kg to 38.5 kg between 1965 and 1985 and there was as correlation of $r = +0.92$ between sugar supplies and caries levels in 12 year olds in the Netherlands.

Marthaler[43] after asking the question 'Is the relation between dietary sugar and individual caries activity vanishing in countries where fluorides are used extensively?' said that 'whereas sugar has lost its dominant role as the primary determinant of average prevalence, … the studies demonstrate that within modern societies which are aware and make use of prevention, the relation between sugar consumption and caries still exists.' One of the reasons why the importance of sucrose is decreasing is that it is increasingly substituted by fructose, glucose and dextrose. In the USA sucrose constituted half of all sugars consumed in 1984 and in Ireland the average glucose consumption had risen to 23 g per capita per day[43].

The decline in dental caries appears to have stopped in most EU countries. Fejerskov and Baelum[55] came to the conclusion that '*A gradual decline in caries incidence has occurred over 40 years and has stabilized at a 'national' plateau*'. Poulsen[139] concluded that in Denmark '*the constant decrease in dental caries in Danish children and adolescents observed during the 1970s and early 1980s has now come to a halt*'. His analysis shows that the caries increment during preschool ages remained the same from cohort to cohort. Among 12-year-olds a plateau was reached around 1991 with about 45% being caries free and with a mean DMFS of 1.8–1.9. The DMFS in 15-year-olds has hardly changed since 1993 when the DMFS was 4.00 and 60% of children were caries free[53,139]. In the Netherlands the caries rates in 6 year olds had stopped declining[140]. Pitts *et al.*[16] came to similar conclusions about trends in the UK. They found an apparent slow down in improvements in caries and static levels of DMF.

Probable projections of future prevalence of caries

As indicated above, caries is a progressive disease and that although the prevalence and severity is moderate in the very young, the severity increases with age. There are trend lines for each level of caries. Groups with the particular level of caries at 6 years of age follow a rising trend line with a predictable dental caries increment. The slopes of the trend lines change as the caries levels decrease[135]. What is the significance of the trend lines? Some trend data are from countries such a Denmark, where there are annual national reports of caries. It is unlikely that the levels of fluoride available can account for the year-by-year decreases and the constancy of the trends. Two explanations for these trends are that:

1. There may be a change in the oral ecosystem, such as changes in numbers and virulence of the cariogenic organisms, at the tooth surface and the mouth.
2. The reasons for the changes may be environmental. Arguably the dominant changes in the environment which affects caries is fluoride, or changes in diet or social factors such as changes in breast feeding patterns and sugars consumption[137]. For a given cariogenic challenge there is a specific intra-oral pattern of caries. The pattern is changed by either altering the pathogenic challenge or increasing the resistance of tooth surfaces. The strength of the dental caries challenge affects the site specific caries pattern within the mouth.

The trend lines also indicate that as people age, they get more caries. The epidemiological trends of caries suggests that each new age cohort will have lower levels of caries and that caries will occur at later ages than at present until they reach a plateau for caries in children. The increase in severity in older people is related to the fact that with increasing age, the root surfaces of the teeth become exposed and they are susceptible to caries. In addition, there are a fair proportion of older people who have a reduced salivary flow, dry mouth. That is a risk factor for caries, as the beneficial buffering effect of saliva against acids is decreased. Therefore the pool of caries in older people will increase.

Unless broader measures are introduced to reduce the

cariogenic challenge, new caries increments will continue with increasing age, but at a lower rate than when caries levels were high. Caries will affect mainly pits and fissures on molars. The rate of progression of demineralization of enamel and dentine will decrease. That will allow longer intervals between screening and dental check-ups. More people will retain more teeth and therefore more root surfaces will be at risk of root caries.

Dental caries will continue to be a major public health problem in Europe. In the short term future dental caries will continue to be a disease of children and young adults. In the longer term future, within 20 years, instead of being a disease mainly of children it will be a disease of adults. Later still, as the cohorts of middle aged adults become older people and there will be higher proportions of older people in the next decades, severe caries will be seen mainly in the old.

Life cycle features

Infants and pre-school children

As reported earlier, early childhood caries (ECC) is related to the use of a sugar. Caries in early childhood has been considered a 'dieto-bacterial disease'[141]. Bowen[141] maintains that '*diet influences … the virulence of cariogenic microorganisms*'. Persson *et al.*[142] linked diet at age 12 months with caries at 3 years. In an extensive review of 40 cross-sectional observational studies Rugg-Gunn and Edgar[143] found that in most studies there were significant correlations between caries experience and sugar consumption for children under five years old. Among children $3\frac{1}{2}$ to $4\frac{1}{2}$ years olds in Great Britain, 30% had decay experience and among those with active caries, 30% had some decay which extended into the pulp[11]. These patterns are widespread in Europe indicating that dental caries in pre-school children is still a major problem. In addition to dental caries, 50% of the incisors of the 5–6 year olds are eroded by acids from drinks[11].

Older people

Three factors increase the susceptibility of older people to dental caries.

1. Older people have a higher taste threshold for sweet[144].
2. Levels of xerostomia (dry mouth) increase with increasing age. In particular, dry mouth is associated with many drugs used by older people.
3. There is a high prevalence of exposed roots of teeth. Exposure increases vulnerability to caries. The pH required to demineralize exposed dentine is higher (pH 5.8) than for enamel (pH 5.5). Therefore, roots of teeth are more susceptible than enamel to acids from sugars and starches.

The following factors contribute to the increasing rates of dental caries in older people:

- The progressive nature of dental caries – even with slow rates of progression,
- demineralization undermines the enamel and creates cavities in later life on the coronal parts of the tooth.
- More teeth are present than in previous decades so more teeth at risk.
- Recession of the gums, getting long in the tooth, exposes more tooth, and particularly the roots of teeth. The cementum covering the roots is more susceptible to caries and demineralizes at lower pH than enamel.
- Poorer saliva flow – the buffering capacity of saliva against acids decreases.
- Increased numbers of older people taking medicines that leads to dry mouth.
- Increased intake of sugars, sometimes to stimulate saliva

As more older people are retaining more teeth into later life, the numbers of teeth at risk of caries will increase[6]. The risk is increased because older people with dry mouth use strong gustatory stimuli such as sugars to increase salivary flow. Sugars used frequently in older people with reduced salivary flow and buffering capacity will result in caries on the vulnerable exposed tooth roots[145]. In an extensive national study of a randomly selected sample of 437 free living older people aged 65 years and older and with natural teeth in Great Britain, Steele *et al.*[145] reported that those with a high frequency of intake of sugar-rich foods were significantly more likely to have new root caries than those with low intakes. Frequent intake foods rich in sugars more than doubled the chance of having caries on roots (OR = 2.4). They concluded '*frequent sugars intake is an important contributor to the development of root surface decay, even when all other major contributory variables are taken into account*'[145].

Dose response relationships between caries and sugars

This question is central to the argument about the level of extrinsic sugars that are compatible with low levels of dental caries. Newbrun[146] suggested that the relationship between caries and sugars was a S-shaped curve, rising steeply when the sucrose-containing food is eaten frequently by children when newly erupted teeth are more susceptible. When sugars are eaten less frequently or if the teeth have been longer in the mouth, or if most of the susceptible tooth surfaces are already decayed or filled as in adults, then it may take a higher level of sucrose to cause caries and the resulting caries will be less extensive – the curve will shift to the right. The rate of increase also depends on the availability of fluoride. Sheiham[147] suggested that where fluoride is present in drinking water at 0.7–1.0 ppm, or over 90 percent of the

toothpastes available are fluoridated, the dose-effect curve shifts to the right and the 'safe' level of sugars increases. Following the rise in caries, the curve flattens out, so that increasing the sucrose content of the diet beyond a certain level does not increase caries to an appreciable extent. This explains the findings in some human studies of a weak relationship between total sugar consumption and caries: the sugar intakes of all the individuals in the population are so high that they lie on the upper flat part of the S-shaped curve[147,148]. Significant lowering in caries rates occur only when sugars levels are on the slope and not on the flat of the curve. The benefits of prevention are larger at high levels of sugars below the 'saturation level'. Newbrun's proposal of an S-shaped curve was based upon animal and human studies and is probably correct. Evidence suggests that in populations without fluoride, below a consumption rate of 10 kg sugars per person per year (27.4 grams per day), the caries rate is very low; increasing levels of sugars are followed by increasing levels of caries; at 15 kg per person per year the intensity of the caries attack increases. Above 35 kg a year the curve eventually flattens out, and further increases in sugars do not lead to appreciable increases in caries.

The main studies on the dose-response relationship are those carried out by Takeuchi[117] in Japan and by Sreebny[149]. Wilska[150] was the first to show a relationship between caries and amount of sugar consumed. In countries where the annual per person sugar consumption was below 20 kg, high proportions of adults were caries-free. Above 20 kg the percentage with caries was over 98%. The most comprehensive evidence of the dose-response relationship between sugar and caries comes from studies of the changing levels of caries when sugar consumption decreased and then increased, as occurred during and after the 1939–45 war. Toverud[116] and Takeuchi[117,151,152] and his co-workers have carefully documented the effects on caries of a wartime decrease in sucrose. Takeuchi found that the S-shaped dose-response reached a plateau at 35 kg per person per year. Takeuchi showed that the annual caries incidence rate was positively correlated with the annual sugar consumption in Japan ($r = +0.8$) with increases in sugar consumption from 0.2 kg to 15 kg per person per year. Takahashi[153], using data from Japan collected between 1941 and 1958, showed that at 13.2 kg of sugar per person per year the annual caries incidence in first molars was 17%, at 8 kg the incidence was 10% and at 5 kg, 6%. When sugar consumption decreased to 2 kg the incidence was 2% and when no sugar was available the incidence was zero. When cavities did occur at low sugar levels, they were small and progressed very slowly. What is most significant is that Takeuchi found that, when the sugar consumption rate went beyond 15 kg per person per year, the caries occurred in the first posteruptive year and the attack rate increased[154]. These studies show that when the annual sugar consumption increased above 15 kg, caries intensified. The incidence rate of caries increased more rapidly when annual sugar levels rose – the incline of the dose-response curve became steeper – suggesting a more intensive dietary challenge. Wartime data from Norway and Britain support the Japanese findings. Schulerud[155] concluded that when Norwegian children aged 6–12 years consumed about 28.5 g of sugar a day (10.4 kg per person per year), a good state of dental health was achieved in comparison to pre-war levels. In Britain, evidence that annual levels of sugar consumption below 10 kg per person are compatible with good dental health is provided by a comparison between children evacuated from Jersey during the war and those remaining. Children on the island of Jersey had about 8.3 kg of sugar per person per year until 1944. They had markedly healthier teeth than children evacuated from the island to England. The average number of carious teeth was 1.8 among Jersey children and 5.1 among evacuees aged 5 to 7[156].

Buttner[157], using data from 18 countries in 1959 on the relationship of sucrose consumption and the average number of decayed, missing, and filled (DMF) teeth of children of 10–12 years old, revealed a high positive correlation ($r = 0.95$). Countries with sugar consumption levels below 20 kg per year had very low DMF scores. Sreebny[149] also found a strong positive correlation between per person sugar supplies and dental caries for 12-year old children in 47 populations ($r = 0.72$). The data clearly suggest that low caries rates are associated with a low availability of sugar, and higher rates with larger sugar supplies. For the 21 (mainly underdeveloped) countries with a daily sugar supply per person of less than 50 g, the caries level was less than 3 DMF. For seven countries at the other extreme – a daily supply of 120 g (43.8 kg per person per year) – the DMF was greater than 5. Sreebny considered that 50 g of sugar per day (18.25 kg per person per year) may represent an upper limit of 'safe', or at least 'acceptable' sugar consumption. Künzel[158] in studies on 200 000 Germans found an increase in caries in fluoride-free Plauen when the sugar consumption increased from 19 kg in 1950, to 27 kg in 1955 and 39 kg in 1979. The DMF increased from 4.2 at 11–15 years to 5.5 and then declined with partial water fluoridation to 3.4 in 1979. In fluoridated Karl-Marx Stadt the lowest DMFT at 11–15 years and at 39 kg sugars was 3.4[158].

The dose-response relationship is supported by a recent analysis of caries trends and sugar consumption in Japan by Miyazaki and Morimoto[72]. The caries rate in 12 year-olds increased as per capita sugar consumption increased up to a peak at 29 kg/year in 1973. Thereafter, sugar consumption decreased and so did caries levels, the correlation was 0.91. And the recent study by Downer[68] which reported a linear relationship significant between caries in children and sucrose availability supports a dose-response relationship.

Rodrigues[65] reported that after adjusting for confounding, children having more than 32.6 grams of extrinsic sugar daily (12 kg per child per year) at nursery were 2.75 times more likely to have high caries increment compared to those who consumed up to 32.6 g/child/day. There was a 29% higher risk of high caries increment among children who consumed more than 32.6 g of sugar daily at nursery. Those children with the highest overall sugar intake were 6.2 times more likely to have a high caries increment.

In summary, the studies reviewed show that when annual sugar availability exceeds 15 −20 kg per person per year dental caries increases with increasing levels of sugars intake. When the caries rates reported by Rugg-Gunn *et al.*[39] in their low-sugars group (<78 g sugars/day) are considered, they developed 1.5 new caries surfaces a year, then levels of sugars intake of about 60 g/day as recommended by COMA[63] for teenagers and adults appears to be a reasonable recommendation.

Recommended intakes of sugars

The following are recommended targets for non milk extrinsic sugars intakes:

- Citing its own 1986 report on sugars, the FDA accepted the fact that added sugars contribute to tooth decay in 1993. The Surgeon General's Report on Nutrition and Health stated: '*Frequent consumption of sugars, especially sucrose, promotes formation of dental plaque, the key predisposing cause of both caries*' '*Evidence exists that sugars as they are consumed in the average American diet contribute to the development of dental caries, suggesting that the general public should reduce its sugar consumption.*'[159].
- Free Sugars – Lower Limit 0% of energy, Upper Limit 10% of energy[77].
- '*… average intake of non milk extrinsic sugars should not exceed about* 60 *g/day or* 10 *per cent of total dietary energy.*'[63].
- '*Quantified targets for extrinsic sugars consumption are set in a minority of cases (27 reports). However the commonest recommended level is* 10% *of total energy intake as a maximum level.*' [160,161].

Most authoritative international consensus document(s) on quantitative relationship between the specified nutrient and the disease or risk profile for that disease.

Twenty three national reports set targets for added sugars, the average being 10 percent or less of calories[161]. The following are a list of recent consensus reports which concluded that caries levels will be low when dietary sugars are below 10% of total calories:

1986 Netherlands, Ministry of Health 0 ≠ 10%
1987 Australia, Department of Health 12%
1987 Finland, Nutrition Board 10% or less
1989 Poland, National Institute, less than 10%
1990 WHO 1990 – Diet, Nutrition & Prevention of Chronic Diseases less than 10%
1991 United Kingdom, Department of Health, COMA 10%
1996 Nordic Nutrition Recommendations. The upper limit of 10% fabricated sugars for children and adults with a low energy intake.
1997 Swedish. Most 10% from purified sugars.

Preventability

Known intervention and observation studies

Fluoride

The majority of trials to prevent caries have involved fluorides. The reductions in caries with fluorides range from 10 to 40%. At lower levels of caries, the reductions are smaller in terms of numbers of tooth surfaces prevented from caries. Many of the trials, which were conducted in the 1970s and 80s, have limited application now that the most people are using fluoride toothpaste and the levels and intra-oral patterns of caries are different. A large meta-analysis, which is due to be published soon, indicates that combinations of fluoride vehicles, at low levels of caries, are not very effective in further reducing caries levels in children[162]. Furthermore, in countries such as Denmark, where a range of fluoride methods have been used, the decline in caries has stopped[139].

Sugars

There is some intervention studies on reducing sugar consumption in the 1950s. As they were not well controlled, they were not reviewed. There are three intervention studies of the effect of changing the types of sugars on caries; the Turku studies and four quasi-experimental observational studies on children where sugar intakes were reduced; Hopewood House Study, the Malmo study, the Synanon Study and the Recife study.

The Turku studies investigated the effects of either almost totally replacing or partial replacement of dietary sucrose with xylitol, a natural polyol sweetener. In addition to the first Turku studies where sucrose was replaced by either fructose or xylitol, there are a number of intervention studies using xylitol chewing gum. Later there were some WHO commissioned demonstration trials in Hungary, Thailand and French Polynesia. Whilst there are some shortcomings in the later studies, the total replacement study showed that xylitol was non-cariogenic in adults[96,163].

The Roslagen Study. The cariogenic effects of sweets made with sucrose were compared sweets made with a hydrogenated potato starch containing a mixture of sorbitol, maltitol, maltotritol and higher saccharide alcohols, Lycasin. Compliance was inadequate and the

dropout rate from the study was too high (50%) to allow adequate evaluation[100].

The Malmo Study. In a two-year trial, sucrose was compared with one where sucrose was completely replaced by invert sugar. There was less caries in children eating invert sugar[98].

Observation in non-experimental studies

Hopewood House Study. Children aged 5 to 13 in this institution in Australia were followed up for 15 years. Children were fed a lactovegetarian diet with a little molasses and honey. Comparison was made with non-institutional Australian children and New Guinea villagers. The 13 year olds had a mean DMF of 1.6 compared to the general populations DMF of 10.7. When the children were relocated and changed to the Australian diet their caries levels increased rapidly indicating that the preventive effect of a low sugar diet is not long-lasting[113].

Synanon Study. In a study with no control group, 73 children aged 11–17 years were exposed to a preventive programme, which included fluoride supplements since birth, daily brushing and flossing and the elimination of sugar from the diet[164]. The mean DMFS was very low for the 5–10 year olds (0.53). It is not possible to ascribe the decrease to any single aspect of the programme.

Recife Study. The objective of this study was to test the effect of adoption of dietary guidelines on sugars by nurseries on levels of sugar consumption and on one year dental caries increments in low-socio-economic 3-year-old children. There was a statistically significant difference in frequency and weight of sugar intakes between children attending nurseries adopting and not using guidelines ($p < 0.001$). Children at nurseries adopting guidelines consumed less than half the amount of sugar a day than children at non-adopting nurseries; 22.9 vs 53.5 grams. Children attending nurseries not using guidelines on reduction of sugar intake were 4.8 more times likely to develop caries in one year than those attending nurseries with guidelines. Attending nurseries not using guidelines on reduction of sugar intake increased by 3.6 times, the risk of developing a high caries increment. Children who had a daily sugar intake of more than 32.6 grams had nearly 3 times the risk of developing a high caries increment than those consuming less than 32.6 grams[65].

The findings from these few studies suggests that reductions in sugars consumption at nurseries would have a significant effect on dental caries in pre-school children. Substitution of sucrose by other sweeteners in confectionery and drinks will reduce caries.

Conclusions

1. Sugars, particularly sucrose, are the most important dietary cause of caries in children and adults. At current levels of sugars intake in Europe, there are statistically significant relationships between the frequency and amounts of sugar intake and caries severity. The quantity and frequency of extrinsic sugars intake are strongly related. Both the frequency of consumption and total amount of sugars is important in the aetiology of caries. Health education messages should emphasize that relationship and not concentrate mainly on snacks, confectionery and drinks as the main causes of caries.
2. The intake of extrinsic sugars beyond four times a day leads to an increase risk of dental caries.
3. There is no evidence that sugars naturally incorporated in the cellular structure of foods (intrinsic sugars) or lactose in milk or milk products (milk sugars) have adverse effects on health. Foods rich in starch, without the addition of sugars, play an insignificant role in coronal dental caries.
4. There should be an independent review, by statisticians, of the data from the controlled trials on caries and sugars. A question that is not addressed by most authors who question whether there is currently a significant relationship between sugars and caries is 'If the sugars levels are not significantly affecting caries at present, what is causing caries to progress at rates sufficiently rapidly to control the increase in caries, with increasing age, in all populations in Europe'?
5. The current dose-response relationship between caries and extrinsic sugars consumption and data from the observational studies on teenagers suggests that the sugars levels should not exceed 60 g/person/day for teenagers and adults. For pre-school and young children the intakes should be proportional to those for teenagers; about 30 g/person/day for pre-school children.
6. Research should be carried out to analyse the claim that there is a sugars/fats seesaw. Some argue that if sugars levels are decreased, there will be an increase in fats consumption with a concomitant increase in CHD and obesity.
7. Fluoride, particularly in toothpastes, is a very important preventive agent against dental caries. At low levels of caries in people using fluoride toothpaste, the preventiveness of combinations of fluorides, is low. As additional fluoride to that currently available in toothpaste does not appear to be benefiting the teeth of the majority of children, the main strategy to further reduce the levels of caries, is reducing the sugars levels in the diet.
8. Dental caries and its consequences cause a lot of pain and suffering. The treatment is expensive and palliative. The costs account for between 4 to 11% of the health budgets of European countries. On a

population basis, dental caries is the most expensive part of the human body to treat.

9. Caries has declined in Europe and is occurring at later ages. Caries is far from being a negligible health problem in a segment of the population.
10. The decline in dental caries has stopped in some EU countries where levels of DMF are 1 at 12 years of age.
11. Caries increases with increasing age. Dental caries will continue to be a major public health problem in Europe in the medium-term future.
12. Although the distribution of caries is skewed to the left, most of the caries increases occurs in the majority, who have low DMF, and not in a minority with high levels of caries.
13. Dietary aetiological factors are still causing dental caries in a significant proportion of population.
14. Caries levels in young immigrants and refugees are increasing.
15. Dental erosion rates appear to be increasing.

References

1 Arens U, ed. *Oral Health Diet and Other Factors*. Amsterdam: Elsevier, 1998.
2 Rugg-Gunn AJ. *Nutrition and Dental Health*. Oxford: Oxford University Press, 1993.
3 Department of Health. *Dietary Sugars and Human Disease. Committee on Medical Aspects of Food Policy. COMA. Report No 37*. London: H.M. Stationery Office, 1989.
4 Kohlmeier L, Kroke A, Pötzsch J, Kohlmeier M, Marin K. *Ernährungsabhängige Krankheiten und ihre Kosten*. Nomos Verlagsgesellschaft: Baden-Baden, 1993: 327–8.
5 Schneider M, Beckmann M, Biene-Dietrich S, Gabanyi M, Hofmann U, Köse A, Mill D, Späth B. *Gesundheitssysteme im internationalen Vergleich*. Augsburg: Eigenverlag, 1998: 142–3.
6 Holst D, Schuller AA. Oral Health Changes in an Adult Norwegian Population – A Cohort Analytical Study. *Community Dent. Oral Epidemiol.* 2000; **28**: 102–11.
7 Todd JE. *Children's Dental Health in England and Wales 1973*. London: HMSO, 1975.
8 Kelly M, Steele J, Nuttall N, Bradnock G, Morris J, *et al. Adult Dental Health Survey. Oral Health in the United Kingdom 1998*. London: The Stationery Office, 2000.
9 Shepherd MA, Nadanovsky P, Sheiham A. The Prevalence and Impact of Dental Pain in Eight Year Old School Children in Harrow, England. *Br. Dent. J.* 1999; **187**: 38–41.
10 Davies GN. Early Childhood Caries: A Synopsis. *Community Dent. Oral Epidemiol.* 1998; **26**(1 Suppl): 106–116.
11 Hinds K, Gregory JR. *National Diet and Nutrition Survey: Children Aged 11/2 to 41/2 Years. Volume 2*. Report of the Dental survey. London: HMSO, 1995.
12 Marthaler TM, O'Mullane DMO, Vrbic V. The Prevalence of Dental Caries in Europe 1990–1995. *Caries Res.* 1996; **30**: 237–55.
13 WHO WHO Oral Health Country/Area Profile Programme. http://www.whocollab.od.mah.se/index.html, 1999.
14 Pitts NB, Palmer J. The Dental Caries Experience of 5-, 12- and 14-Year Old Children in Great Britain. Surveys Coordinated by the Br. Association for the Study of Community Dentistry in 1991/92, 1992/93 and 1990/91. *Community Dent. Health* 1997; **11**: 42–52.
15 Pitts NB, Evans DJ. The Total Dental Caries Experience of 5 Year-Old Children in the United Kingdom. *Community Dent. Health* 1997; **14**: 47–52.
16 Pitts NB, Evans DJ, Nugent ZJ. The Total Dental Caries Experience of 12-Year-Old Children in the United Kingdom. Surveys Coordinated by the Br. Association for the Study of Community Dentistry in 1996/7. *Community Dent. Health* 1998; **15**: 49–54.
17 Fejerskov O, Baelum V, Ostergaard ES. Root Caries in Scandinavia in the 1980s and Future Trends to be Expected in Dental Caries Experience in Adults. *Advances in Dent. Res.* 1993; **7**(1): 4–14.
18 WHO. *Monitoring Dental Caries in Adults Aged 35–44 Years. WHO/ORH/Caries.35-44/96.1*. Geneva: WHO, 1996.
19 O'Mullane D, Whelton H. *Oral Health of Irish Adults 1989–1990*. Dublin: Stationery Office, 1992.
20 Bedi R, Uppal RDK. The Oral Health of Minority Ethnic Groups in the United Kingdom. *Br. Dent. J.* 1995; **179**: 421–5.
21 Verrips GH. *Child Dental Health and Ethnicity in the Netherlands*. Leiden: TNO Institute for Preventive Health Care TNO, 1993.
22 Meurman JH, ten Cate JM. Pathogenisis and Modifying Factors of Dental Erosion. *European J. Oral Science* 1996; **104**: 199–206.
23 O'Brien M. *Children's Dental Health in the United Kingdom*. London: H.M. Stationery Office, 1994.
24 Ten Cate JM, Imfeld T, Dental erosion, summary. In: Ten Cate JM, Imfeld T, eds. *Etiology, Mechanisms and Implications of Dental Erosion. A Workshop Organized by ILSI Europe Oral Health Task Force and ISLI North America Oral Health Technical Committee*. European J Oral Sciences, 1996; 104 (Part II): 241–4.
25 Jarvinen VK, Rytomaa II, Heinonen M. Risk Factors in Dental Erosion. *J. Dent. Res.* 1991; **70**: 942–7.
26 Smith AJ, Shaw L. Baby Fruit Juicd and Tooth Erosion. *Br. Dent. J.* 1987; **162**: 65–7.
27 Dental Practice Board for England and Wales. National Clinical Guidelines and Policy Documents 1999 Paediatric Dentistry. Eastbourne, UK, 1999: 24–9.
28 Millward A, Shaw L, Smith A. Dental Erosion in Four Year Old Children from Differing Socioeconomic Backgrounds. *J. Dentistry for Children* 1994; **61**: 263–6.
29 Arends J, ten Bosch JJ. In Vivo de- and remineralization of dental enamel. In: Leach SA, ed. *Factors Relating to Demineralization and Remineralization of the Teeth*. Oxford: IRL Press, 1985: 1–11.
30 Marsh PD. The control of oral biofilms: new approaches for the future. In: Guggenheim B, Shapiro S, eds. *Oral Biology at the Turn of the Century*. Basel: Karger, 1998: 22–31.
31 Bratthall D. Dental Caries: Intervened-Interrupted-and Interpreted. Concluding Remarks and Cariography. *European J. Oral Sciences* 1996; **104**: 486–91.
32 van Palenstein Helderman WH, Matee MIN, Van der Hoeven JS, Mikx FHM. Cariogenicity Depends more on Diet than the Prevailing Mutans Streptococci Species. *J. Dent. Res.* 1996; **75**: 535–45.
33 van der Hoeven JS, van Palenstein Helderman WH. Microbial specificity and dental caries. In: Guggenheim B, Shapiro S, eds. *Oral Biology at the Turn of the Century*. Basel: Karger, 1998: 43–55.
34 Mandel I. Impact of Saliva on Dental Caries. *Compendium in Continuing Education Dental Supplement* 1989; **13**: 476–81.
35 Edgar WM. The Physicochemical Evidence. *J. Dentistry* 1983; **11**: 199–205.
36 Murray JJ. *Appropriate Use of Fluorides for Human Health*. Geneva: World Health Organization, 1986.
37 Sticksen-Blicks C, Holm A-K. Dental Caries, Tooth Trauma, Malocclusion, Fluoride Usage, Toothbrushing and Dietaery

Habits in 4-Year-Old Swedish Children: Changes Between 1967 and 1992. *Int. J. Paediatric Dentistry* 1995; **5**: 143–8.
38 Mejáre I, Kallestal C, Stenlund H, Johansson H. Caries Development from 11 to 22 Years of Age: A Prospective Radiographic Study. *Caries Res.* 1998; **32**: 10–6.
39 Rugg-Gunn AJ, Hackett AF, Appleton DR, Jenkins GN, Eastoe JE. Relationship Between Dietary Habits and Caries Increment Assessed over Two Years in 405 English School Children. *Arch. Oral Biol.* 1984; **29**: 983–92.
40 Burt BA, Eklund SA, Morgan KJ, Larkin FE, Guire KE, Brown LO, Weintraub JA. The Effects of Sugars Intake and Frequency of Ingestion on Dental Caries Increment in a Three Year Longitudinal Study. *J. Dent. Res.* 1988; **67**: 1422–9.
41 Beighton D, Adamson A, Rugg-Gunn A. Associations Between Dietary intake, Dental Caries Experience and Salivary Bacterial Levels in 12-Year-Old English Schoolchildren. *Arch. Oral Biol.* 1996; **41**(3): 271–80.
42 Holt RD. Foods and Drinks at Four Daily Time Intervals in a Group of Young Children. *Br. Dent. J.* 1991; **170**: 137–43.
43 Marthaler TM. Changes in the Prevalence of Dental Caries: How Much Can Be Attributed to Changes in Diet? *Caries Res.* 1990; **24**(Suppl. 1): 3–15.
44 Weaver R. Fluorine and Wartime Diet. *Br. Dent. J.* 1950; **88**: 231–9.
45 Fejerskov O, Manji F, Baelum V, Moller IJ. *Dental Fluorosis – a Handbook for Health Workers*. Copenhagen: Munksgaard, 1988.
46 Szpunar SM, Burt BA. Trends in the Prevalence of Dental Fluorosis in the United States. *A Review. J. Public Health Dentistry* 1987; **47**: 71–9.
47 Ismail AL, Brodeur J-M, Kavanaugh M, Boisclair G, Tessier C, Picotte L. Prevalence of Dental Caries and Dental Fluorosis in Students, 11–17 Years of Age in Fluoridated and non-Fluoridated cities in Quebec. *Caries Res.* 1990; **24**: 290–7.
48 Clark DC. Trends in Prevalence of Dental Fluorosis in North America. *Community Dent. Oral Epidemiol.* 1994; **22**: 148–52.
49 Jackson RD, Kelly SA, Katz BP, Hull JR, Stookey GK. Dental Fluorosis and Caries Prevalence in Children Residing in Communities with Different Levels of Fluoride in the Water. *J. Public Health Dent.* 1995; **55**: 79–84.
50 Pendrys DG, Katz RV, Morse DE. Risk Factors for Enamel Fluorosis in a non Fluoridated Population. *Am. J. Epidemiol.* 1996; **143**: 305–15.
51 Holloway PJ, Ellwood RP. The Prevalence, Causes and Cosmetic Importance of Dental Fluorosis in the United Kingdom: A Review. *Community Dent. Health* 1997; **14**: 148–55.
52 Selwitz RH, Nowjack-Raymer RE, Kingman A, Driscoll WS. Dental Caries and Dental Fluorosis Among Children Who Were Lifetime Residents of Communities Having Either Low or Optimal Levels of Fluoride in Drinking Water. *J. Public Health Dentistry* 1998; **58**: 26–35.
53 Wang NJ, Gropen AM, Øgaard B. Risk Factors Associated with Fluorosis in a non-Fluoridated Population in Norway. *Community Dent. Oral Epidemiol.* 1997; **25**: 396–401.
54 Mascarenhas AK, Burt BA. Fluorosis Risk From Early Exposure to Fluoride Toothpaste. *Community Dent. Oral Epidemiol.* 1998; **26**: 241–8.
55 Fejerskov O, Baelum V. Changes in prevalence and incidence of the major oral diseases. In: Guggenheim B, Shapiro S, eds. *Oral Biology at the Turn of the Century*. Basel: Karger, 1998: 1–11.
56 Pollard MA, Toumba KJ, Curzon MEJ. Diet and Dental Caries. Leeds Dental Institute, Mimeo 55 pages. Submission to COMA, 1996.
57 Rose G. *The Strategy of Preventive Medicine*. 2nd Edition. Oxford: Oxford University Press, 1993.
58 Batchelor PA. The Scientific Basis for the Modelling of Caries Preventive Strategies. PhD Thesis. University of London: 1998.
59 Scheutz F, Poulsen S. Determining Causation in Epidemiology. *Community Dent. Oral Epidemiol.* 1999; **27**: 161–70.
60 Rothman KJ, Greenland S. *Modern Epidemiology*. Philadelphia, PA: Lippincott-Raven Publishers, 1998.
61 König KG, Navia JM. Nutritional role of Sugars in Oral Health. *Am. J. Clin. Nutrition* 1995; **62**(Supp. l): 275S–83S.
62 Burt BA, Ismail AI. Diet, Nutrition, and Food Cariogenicity. *J. Dent. Res.* 1986; **65**(Sp Iss): 1475–84.
63 Department of Health. *Dietary Reference Values for Food Energy and Nutrients for the United Kingdom. Committee on Medical Aspects of Food Policy*. COMA. Report No 41. London: H.M. Stationery Office, 1991.
64 Gustafsson BE, Quensel CE, Sweander Lanke L, Lundqvist C, Grahnen H, Bonow BE, Krasse B. The Effect of Different Levels of Carbohydrate Intake on Caries Activity in 436 Individuals Observed for Five Years. The Vipeholm Dental Caries Study. *Acta Odontologika Scandinavika* 1954; **11**: 232–364.
65 Rodrigues CS. Dietary Guidelines, Sugar Intake and Caries Increment: A Study in Brazilian Nursery School Children. PhD Thesis. University of London: 1997.
66 Garn SM, Cole PE, Solomon MA, Schaefer AE. Relationships Between Sugar-Foods and the DMFT in 1968–1970. *Ecology of Food and Nutrition* 1980; **9**: 135–8.
67 Sreebny LM. Sugar Availability, Sugar Consumption and Dental Caries. *Community Dent. Oral Epidemiol.* 1982; **10**: 1–7.
68 Downer MC. Caries Experience and Sucrose Availability: An Analysis of the Relationship in the United Kingdom Over Fifty Years. *Community Dent. Health* 1999; **16**: 18–21.
69 Hackett AF, Rugg-Gunn AJ, Appleton DR, Allinson M, Eastoe JE. Sugars-Eating Habits of 405 11- to 14-Year-Old English Children. *Br. J. Nutrition* 1984; **51**: 347–56.
70 Árnadóttir IB, Rozier RG, Saemundsson SR, Sigurjons H, Holbrook WP. Approximal Caries and Sugar Consumption in Icelandic Teenagers. *Community Dent. Oral Epidemiol.* 1998; **26**: 115–21.
71 Burt BA, Szpunar SM. The Michigan Study: the Relationship Between Sugars Intake and Dental Caries Over Three Years. *Int. Dent. J.* 1994; **44**: 230–40.
72 Miyazaki H, Morimoto M. Changes in Caries Prevalence in Japan. *European J. Oral Science* 1996; **104**: 452–458.
73 Downer MC. The Changing Pattern of Dental Disease Over 50 Years. *Br. Dent. J.* 1998; **185**: 36–41.
74 Woodward M, Walker ARP. Sugar and Dental Caries: The Evidence From 90 Countries. *Br. Dent. J.* 1994; **176**: 297–302.
75 Nadanovsky P. *Letter. Br. Dent. J.* 1994; **177**(8): 280.
76 Ruxton CHS, Garceau FJS, Cottrell RC. Guidelines for Sugar Consumption in Europe: Is a Quantitative Approach Justified? *European J. Clin. Nutrition* 1999; **53**: 503–13.
77 WHO. *Diet, Nutrition and Prevention of Chronic Diseases*. Geneva: World Health Organization, 1990.
78 Ismail AI, Burt BA, Eklund SA. The Cariogenicity of Soft Drinks in the United States. *J. the Am. Dent. Association* 1984; **109**: 241–5.
79 Cleaton-Jones P, Richardson BD, Sreebny LM, Fatti P, Walker AR. The Relationship Between the Intake Frequency and the Total Consumption of Sucrose Among Four South African Ethnic Groups. *J. Dentistry for Children* 1987; **54**: 251–4.
80 Rodrigues CS, Watt RG, Sheiham A. The Effects of Dietary Guidelines on Sugar Intake and Dental Caries in 3 Year Olds Attending Nurseries. *Health Promotion Int.* 1999; **14**: 329–35.
81 Rodrigues CS, Sheiham A. The Relationships Between Dietary Guidelines, Sugar Intake and Caries in Primary

Teeth in Low Income Brazilian 3 Year Olds: A Longitudinal Study. *Int. J. Paedodontics* 2000; In press.
82 Szpunar SM, Eklund SA, Burt BA. Sugar Consumption and Caries Risk in Schoolchildren with Low Caries Experience. *Community Dent. Oral Epidemiol.* 1995; **23**: 142–6.
83 Grindefjord M, Dahllof G, Nilsson B, Modeer T. Prediction of Dental Caries Development in 1-Year-Old Children. Department of Orthodontics and Pediatric Dentistry, School of Dentistry, Karolinska Institutet, Stockholm, Sweden. *Caries Res.* 1995; **29**: 343–8.
84 Grindefjord M, Dahllof G, Nilsson B, Modeer T. Stepwise Prediction of Dental Caries in Children up to 3.5 Years of Age. *Caries Res.* 1996; **30**: 256–66.
85 Holbrook WP, Árnadóttir IB, Takazoe I, Birkhed D, Frostell G. Longitudinal Study of Caries, Cariogenic Bacteria and Diet in Children Just Before and After Starting School. *European J. Oral Science* 1995; **103**(1): 42–5.
86 Holbrook WP, Kristinsson MJ, Gunnarsdóttir S, Briem B. Caries Prevalence, Streptococus Mutans and Sugar Intake Among 4-Year-Old Urban Children in Iceland. *Community Dent. Oral Epidemiol.* 1989; **17**: 292–5.
87 Bjarnason S, Finnbogason SY, Noren JG. Sugar Consumption and Caries Experience in 12- and 13- Year-Old Icelandic Children. *Acta Odontologika Scandinavika* 1989; **47**: 315–21.
88 Sundin B, Granath L, Birkhed D. Variation of Posterior Approximal Caries Incidence with Consumption of Sweets with Regard to Other Caries-Related Factors in 15–18-Year-Olds. *Community Dent. Oral Epidemiol.* 1992; **20**: 76–80.
89 Sundin B, Granath L. Sweets and Other Sugary Products Tend to Be the Primary Etiologic Factors in Dental Caries. *Scandinavian J. Dent. Res.* 1992; **100**: 137–9.
90 Kalsbeek H, Verrips GH. Consumption of Sweet Snacks and Caries Experience of Primary School Children. *Caries Res.* 1994; **28**: 477–83.
91 Jamel HA, Sheiham A, Watt RG, Cowell CR. Sweet preference, Consumption of Sweet Tea and Dental Caries: Studies in Urban and Rural Iraqi Populations. *Int. Dent. J.* 1996; **47**: 213–7.
92 Jones C, Woods K, Whittle G, Worthington H, Taylor G. Sugar, Drinks, Deprivation and Dental Caries in 14-Year-Old Children in the North West of England in 1995. *Community Dent. Health* 1999; **16**: 68–71.
93 Kleemola-Kujala E, Rasanen L. Relationship of Oral hygiene and Sugar Consumption to Risk of Caries in Children. *Community Dent. Oral Epidemiol.* 1982; **10**(5): 224–33.
94 Larsson B, Johansson I, Ericson T. Prevalence of Caries in Adolescents in Relation to Diet. *Community Dent. Oral Epidemiol.* 1992; **20**: 133–7.
95 Bergendal B, Hamp SE. Dietary Pattern and Dental Caries in 19-Year-Old Adolescents Subjected to Preventive Measures Focused on Oral hygiene and/or Fluorides. *Swedish Dent. J.* 1985; **9**: 1–7.
96 Scheinin A, Mäkinen KK. Turku Sugar Studies 1-XX! *Acta Odontologika Scandinavika* 1975; **33**(Supp 70): 1–351.
97 Scheie AA, Fejerskov OB. Xylitol in Caries Prevention – What is the Evidence for Clin. efficacy? *Oral Diseases* 1998; **4**: 268–78.
98 Frostell G, Birkhed D, Edwardsson S, Goldberg P, Petersson L-G, Priwe C, Winholt A-S. Effect of Partial Substitution of Invert Sugar for Sucrose in Combination with Duraphat Treatment on Caries Development in Preschool Children: The Malmo Study. *Caries Res.* 1991; **25**: 304–10.
99 Birkhed D, Bär A. Sorbitol and Dental Caries. *World Review of Nutrition & Diet* 1991; **65**: 1–37.
100 Frostell G, Blomlof L, Blomqvist T, Dahl GM, Edward S, Fjellstrom A, Henrikson CO, Larje O, Nord CE, Nordenvall KJ. Substitution of Sucrose by Lycasin in Candy the Roslagen Study. *Acta Odontologika Scandinavika* 1974; **32**: 235–54.
101 Rugg-Gunn AJ. *Starchy Foods and Fresh Fruits: Their Relative Importance as a Source of Caries in Britain. Occasional Paper No. 3*. London: Health Education Council, December 1975.
102 Rugg-Gunn AJ, Nunn JH. *Nutrition, Diet and Oral Health*. Oxford University Press, 1999: 43–5.
103 Edgar WM. Prediction of the Cariogenicity of Various Foods. *Int. Dent. J.* 1985; **35**(3): 190–4.
104 Edgar WM. Extrinsic and Intrinsic Sugars: A Review of Recent UK Recommendations on Diet and Caries. *Caries Res.* 1993; **27**(Suppl 1): 64–7.
105 Koulourides T, Bodden R, Keller S, Manson-Hing L, Lastra J, Housch T. Cariogenicity of Nine Sugars Tested with an Intraoral Device in Man. *Caries Res.* 1976; **10**: 427–41.
106 Bowen WH. Dental caries in primates. In: Tanzer JM, ed. *Animal Models in Cariology*. London: IRL, 1980: 131–5.
107 Firestone AR, Schmid R, Muhlemann HR. Cariogenic Effects of Cooked Wheat Alone or with Sucrose and Frequency-controlled Feedings in Rats. *Arch. Oral Biol.* 1982; **27**: 759–63.
108 Mundorff SA, Featherstone JDB, Eisenberg AD, Cowles E, Curzon MEJ, Espeland MA, Shields CP. Cariogenic Potential of Foods. II. Relationship of Food Composition, Plaque Microbial Counts, and Salivary Parameters to Caries in the Rat Model. *Caries Res.* 1994; **28**: 106–15.
109 Cohen B, Bowen WH. A Bacteriological Study of Dental Caries in Experimental Monkeys. *Br. Dent. J.* 1966; **121**: 269–76.
110 Cohen B. Letter to editor. *Br. Dent. J.* 1983; **155**: 329.
111 Lehner T, Challacombe SJ, Caldwell J. An Experimental Model for Immunological Studies of Dental Caries in the Rhesus Monkey. *Arch. Oral Biol.* 1975; **20**: 299–304.
112 Pollard MA. Potential Cariogenicity of Starches and Fruits as Assessed by Plaque Sampling Method and Intraoral Cariogenicity. *Caries Res.* 1996; **29**: 68–74.
113 Harris R. The Biology of the Children of Hopewood House, Bowral, 4. Observations on Dental Caries Experience Extending Over Five Years (1957–61). *J. Dent. Res.* 1963; **42**: 1387–99.
114 Newbrun E, Hoover C, Mettraux G, Graf H. Comparison of Dietary Habits and Dental Health of Subjects with Hereditary Fructose Intolerance and Control Subjects. *J. the Am. Dent. Association* 1980; **101**: 619–26.
115 Rugg-Gunn AJ, Hackett AF, Appleton DR. Relative Cariogenicity of Starch and Sugars in a 2-Year Longitudinal Study of 405 English Schoolchildren. *Caries Res.* 1987; **21**: 464–73.
116 Toverud G. The Influence of War and Post-war Conditions on the Teeth of Norwegian Schoolchildren. *Millbank Memorial Fund Quarterly* 1957; **35**: 127–96, 373-459.
117 Takeuchi M. Epidemiological Study on Dental Caries in Japanese Children Before, During and After World War II. *Int. Dent. J.* 1961; **11**: 443–57.
118 Künzel W, Fischer T. Rise and Fall of Caries Prevalence in German Towns with Different F Concentrations in Drinking Water. *Caries Res.* 1997; **31**: 166–73.
119 Zeisenitz SC, Edmondson EMS. Dental Health. Relationship Between Dental caries, Diet, Eating Patterns and Fluoride. Paper submitted to the EU Working Group 1 on Nutrition and Diet or Healthy Lifestyles in Europe. Rome, October 1999.
120 Afronsky D. Some Observations on Dental Caries in Central China. *J. Dent. Res.* 1951; **30**: 53–61.
121 Russell AL, Littleton NW, Leatherwood EC, Sydow GE, Green JC. Dental Surveys in Relation to Nutrition. *Public Health Reports* 1960; **75**: 717–23.
122 Fisher FJ. A Field Survey of Dental Caries, Periodontal Disease and Enamel Defects in Tristan da Cunha. Part 2, Methods and Results. *Br. Dent. J.* 1968; **125**: 447–53.
123 Anaise JZ. Prevalence of Dental Caries among Workers in

the Sweet industry in Israel. *Community Dent. Oral Epidemiol.* 1980; **8**: 142–5.
124 Katayama T, Nagagawa E, Honda O, Tani H, Okado S, Suzuki S. Incidence and Distribution of Strep. Mutans in Plaque From Confectionery Workers. *J. Dent. Res.* 1979; **58**(Sp. issue. D): 2251 (Abstr. 11).
125 Petersen PE. Dental Health Among Workers at a Danish Chocolate Factory. *Community Dent. Oral Epidemiol.* 1983; **11**: 337–41.
126 Sreebny LM. Cereal Availability and Dental Caries. *Community Dent. Oral Epidemiol.* 1983; **10**: 1–7.
127 Moynihan PJ. Update on the Nomenclature of Carbohydrates and Their Dental Effects. *J. Dentistry* 1998; **26**: 209–18.
128 Clancy KL, Bibby BG, Goldberg HJV, Ripa LW, Barenie J. Snack Food Intake of Adolescents and Caries Development. *J. Dent. Res.* 1977; **56**: 568–73.
129 Grobler SR, Blignaut JB. The Effect of a High Consumption of Apples or Grapes on Dental Caries and Periodontal Disease in Humans. *Clin. Preventive Dentistry* 1989; **11**: 8–12.
130 Petersson HG, Bratthall D. The Caries Decline: A Review of Reviews. *European J. Oral Science* 1996; **104**: 436–43.
131 Sutcliffe P. Oral Cleanliness and Dental Caries. In: Murray JJ, ed. *The Prevention of Oral Disease*. Oxford: Oxford University Press, 1996: 68–77.
132 Bellini H, Arneberg P, von der Fehr F. Oral Hygiene and Caries. A Review. *Acta Odontologica Scandinavica* 1981; **39**: 257–65.
133 Health Education Authority. *The Scientific Basis of Dental Health Education. A Policy Document*. Fourth Edition. 1996; London: Health Education Authority,.
134 Sullivan HR, Harris R. The biology of Children of Hopewood House, Bowral, NSW. 2. Observations on Oral conditions. *Australian Dent. J.* 1958; **23**: 311–7.
135 Sheiham A. What explains the caries decline? In: Guggenheim B, Shapiro S, eds. *Oral Biology at the Turn of the Century*. Basel: Karger, 1998: 32–42.
136 Nadanovsky P, Sheiham A. The Relative Contribution of Dental Services to the Changes and Geographical Variations in Caries Status of 5 and 12 Year-Old Children in England and Wales in the 1980s. *Community Dent. Health* 1994; **11**: 215–23.
137 Nadanovsky P, Sheiham A. The Relative Contribution of Dental Services to the Changes in Caries Levels of 12 Year-Old Children in 18 Industrialized Countries in the 1970s and early 1980s. *Community Dent. Oral Epidemiol.* 1995; **23**: 231–9.
138 König KG. Changes in Prevalence of Dental Caries, How Much Can Be Attributed to Changes in Diet? *Caries Res.* 1990; **24**(Suppl): S16–8.
139 Poulsen S. Dental Caries in Danish Children and Adolescents 1988–94. *Community Dent. Oral Epidemiol.* 1996; **24**: 282–5.
140 Frencken JE, Kalsbeck H, Verrips GH. Has the Decline in Dental Caries Been Halted? *Int. Dent. J.* 1990; **40**: 225–30.
141 Bowen WH. Biological Mechanisms of Early Childhood Caries. *Community Dent. Oral Epidemiol.* 1998; **26**(1 Suppl): 28–31.
142 Persson LA, Holm AK, Arvidsson S, Samuelson G. Infant Feeding and Dental Caries: a Longitudinal Study of Swedish Children. *Swedish Dent. J.* 1985; **9**: 201–6.
143 Rugg-Gunn AJ, Edgar WM. Sugar and Dental Caries: A Review of Evidence. *Community Dent. Health* 1984; **1**: 85–92.
144 Moore LM, Nielsen CR, Mistretta CM. Sucrose Taste Thresholds: Age Related Differences. *J. Gerontology* 1982; **37**: 64–9.
145 Steele JG, Sheiham A, Marcenes W, Walls AWG. *National Diet and Nutrition Survey: People Aged 65 Years and Over*. Volume 2: Report of the Oral Health Survey. London: The Stationery Office, 1998.
146 Newbrun E. Sucrose in the Dynamics of the Carious Process. *Int. Dent. J.* 1982; **32**: 13–23.
147 Sheiham A. Sugars and Dental Decay. *The Lancet* 1983; **I**: 282–4.
148 Sheiham A. Sucrose and Dental Caries. *Nutrition and Health* 1987; **5**: 25–9.
149 Sreebny LM. The Sugar-Caries Axis. *Int. Dent. J.* 1982; **32**: 1–12.
150 Wilska A. Sokerikaries-Vuosistamme Levinnen tauti (Sugar Caries – The Most Prevalent Disease of our Century). *Duodecim* 1947; **63**: 449–510.
151 Takeuchi M. Epidemiological Study on Relation Between Dental Caries Incidence and Sugar Consumption. *Bulletin of the Tokyo Dental College* 1960; **1**: 58–70.
152 Takeuchi M. On the Rpidemiological Principles in Dental Caries Attack. *Bulletin of the Tokyo Dental College* 1962; **3**: 96–111.
153 Takahashi K. Statistical Study on Caries Incidence in the First Molar in Relation to the Amount of Sugar Consumption. *Bulletin of the Tokyo Dental College* 1961; **2**: 44–57.
154 Shimamura S. A Cohort Survey on Caries Attacks in Permanent Teeth During a Period of Approximately 20 kg of Annual Sugar Consumption Per Person in Japan. *J. Dent. Health* 1974; **24**: 46–52.
155 Schulerud A. *Dental Caries and Nutrition During Wartime in Norway*. Oslo: Fabritius and Sonners Trykkeri, 1950.
156 Knowles EM. The Effects of Enemy Occupation on the Dental Condition of Children in the Channel Islands. *Monthly Bull. Min. of Health* 1946: 161–72.
157 Buttner W. Zuckeraufnahme und karies. In: Cremer HD, ed. Grundfragen der Ernährungswissenschaft. Freiburg im Breisgau, Rombach, 1971: 175–191. Cited by Marthaler, TM. In: Guggenheim B, ed. Health and Sugar Substitutes. Proceedings of ERGOB Conference on Sugar Substitutes. Basel: Karger, 1979; 27–34.
158 Künzel W. Beziehungen der Kariesverbreiting zum Fluoridgehalt des Trinkwassrs sowie dem jährlichen Zuckerkonsum. *Zahn-Mund-und Kieferheilkunde Bild* 1982; **70**: 584–90.
159 U.S. Department of Health and Human Services, Public Health Service. The Surgeon General's Report on Nutrition and Health Washington, D.C: Government Printing Office, 1988: 368.
160 Cannon G. *Food and Health: The Experts Agree*. London: Consumers Association, 1992.
161 Freire MDCM, Cannon G, Sheiham A. Sugar and Health – An analysis of the recommendations on Sugars and Health in One Hundred and Fifteen Authoritative Scientific Reports on Food, Nutrition and Public Health Published Throughout the World in Thirty Years Between 1961–1991. Department Monograph Series, No. 1. Department of Epidemiology and Public Health, University College London, 1992.
162 Marinho V. A Systematic Review of Topical Fluorides in Caries Prevention. A Meta-analysis Being Conducted Under the Supervision of the Cochrane Collaboration. (unpublished), 2000.
163 Bär A. Caries Prevention with Xylitol. *World Review of Nutrition & Diet* 1988; **55**: 183–209.
164 Silverstein SJ, Knapp JF, Kirkos L, Edwards H. Dental Caries Prevalence in Children With a Diet Free of Refined Sugar. *Am. J. Public Health* 1983; **73**: 1196–9.