Session C
Nutritional Impact in Oral Health Promotion

Paula Moynihan[a]

Key words: sugars intake, nutrition, oral health, caries

Oral Health Prev Dent 2003; 1: Supplement 1: 385-402.

In this paper, an overview of diet, nutrition and oral health associations is presented and recommendations for inclusion of this topic in the dental hygienists curriculum are made. Nutrition and diet impact upon many oral diseases including periodontal disease, oral infections, oral cancer, enamel developmental defects, dental caries and dental erosion. Poor nutritional status compromises the immune status increasing the severity of oral infections, whereas good nutrition may protect against certain oral conditions, e.g. noma. Nutritional affects the teeth pre-eruptively during development but this effect is of much less importance than the local effect of diet in the mouth, e.g. in the causation of dental erosion and dental caries. Dental erosion is perceived to be increasing and is in particular associated with consumption of acidic drinks. Dental caries in many European countries remains unacceptably high, and evidence for an association between sugars and dental caries is indisputable; intakes of free sugars below 15 – 20 kg per year are associated with good dental health. Fluoride increases the safe threshold for sugars intake yet alone, it does not eliminate caries. Dietary advice for dental health must be consistent with advice for general health. Current dietary recommendations for the prevention of chronic diseases promote increased consumption of fruit vegetables and starchy wholegrain foods and reduced consumption of fat (especially saturated) free sugars and salt: these dietary changes would be likely to reduce oral diseases. It is recommended that the dental hygienist has a broad background knowledge of general nutrition, an in-depth knowledge of the associations between nutrition and oral health and good communication skills necessary to impart dietary advice and support behavioral changes.

[a] *School of Dental Sciences, University of Newcastle upon Tyne, Newcastle upon Tyne, UK.*

Reprint requests: *Dr. Paula Moynihan, Senior Lecturer in Nutrition, Department of Oral Biology, The Dental School, University of Newcastle upon Tyne, Newcastle upon Tyne, NE2 4HH, UK. Fax: + 44 191 222 5928. E-mail: p.j.moynihan@ncl.ac.uk*

BACKGROUND

The recently published World Health Organization (WHO) report 'Diet in the Prevention of Chronic Diseases '(WHO, 2003) recommended that "the training of all health professionals (including physicians, dentists, nurses and nutritionists) should include diet, nutrition and physical activity as key determinants of medical and dental health." The aim of this paper is to provide an overview to the scientific background of the current status of evidence for the interrelationships between nutrition and oral health and to summarize how this forms the evidence base for oral health promotion. A brief overview of the role between diet and general health is presented and recommendations for inclusion of nutrition and diet in the curriculum of dental hygienists are summarized.

Oral health encompasses the health of the teeth, the tissues surrounding the teeth (the periodontium) and the oral mucosa – where any of these structures may become diseased. Enamel defects, dental caries, erosion of the teeth, periodontal disease, mouth ulcers and oral cancer are all oral conditions that are influenced by nutrition and diet. Whilst periodontal disease remains the major cause of tooth loss worldwide, dental caries is the main cause of tooth loss in children. Table 1 summarizes the oral conditions that are associated with diet and/or nutrition that will be considered in this review.

Table 1 Oral conditions associated with nutrition and diet

Oral condition	Nutritional/dietary association
Angular Cheilitis	Deficiency of riboflavin (vitamin B2), vitamin B6, folate, vitamin C, iron
Burning mouth syndrome	Deficiencies of Thiamin (vitamin B1), Vitamin B6
Dental caries	Free sugars intake
Dental erosion	Acidic drink and food intake
Enamel hypoplasia	Deficiencies of vitamins A and D and protein
Enamel opacities	Excess fluoride
Glossitis	Deficiency of riboflavin (vitamin B2), vitamin B6, folate, iron
Noma	Protein energy malnutrition
Oral cancer	Low intake of fruits and vegetables
Periodontal disease	Vitamin C deficiency
Recurrent aphthae	
Salivary gland atrophy	Deficiency of riboflavin (vitamin B2), folate, vitamin C
Stomatitis	Deficiencies of Protein and Vitamin A
Tooth loss	Low intake of fiber, vitamin C and fruits and vegetables

Table 2 Nutrient and dietary recommendations for the prevention of chronic diseases: population goals (WHO, 2003)

Nutrient or food group	Goal
Total fat	15 – 30% energy intake
Saturated fat	< 10% energy intake
Carbohydrate	55 – 75% energy intake
Free sugars	< 10% energy intake
Protein	10 – 15% energy intake
Cholesterol	< 300 mg per day
Sodium chloride (salt)	< 5 g per day
Fruits and vegetables	> 400 g per day
Non-starch polysaccharide (dietary fiber)	from foods*

* The recommended intake of fruit and vegetables and consumption of wholegrain foods is likely to provide > 20 g per day of non-starch polysaccharide

Diet and General Health

In addition to dental diseases, many other chronic diseases including cardiovascular diseases, type II diabetes, and cancer, are associated with dietary risk factors. WHO recently published a report of an expert consultation on diet in the prevention of chronic diseases (WHO, 2003), where more in-depth information on the associations between diet and general health may be found. The current nutrient guidelines from this report are summarized in Table 2.

Obesity is a major health problem for all age groups throughout Europe that now affects between 10 and 40% of the adult population and is increasing rapidly in young adults and children (Astrup, 2001; WHO, 2003). Obesity increases the risk of developing Type II Diabetes, cardiovascular disease and some cancers. Eating behaviors that are associated with obesity include snacking and eating frequently, and eating outside the home (WHO, 2003). Regular physical activity is associated with a lower risk of obesity as are diets high in non-starch polysaccharide (NSP), whereas a sedentary lifestyle and

a high intake of energy dense foods rich in fats and free sugars and low in micronutrients are associated with increased risk (Astrup, 2001; WHO 2003). There is some evidence that a high intake of sugared soft drinks and fruit juices contributes to obesity (WHO, 2003).

The prevalence of Type II diabetes is increasing and is associated with overweight and obesity (Astrup, 2001; WHO 2003). Evidence shows that diets high in saturated fatty acids increase the risk of Type II diabetes and replacement of saturated fats with unsaturated improves glucose tolerance. Physical activity and diets high in non-starch polysaccharides (NSP) have a protective effect and diets high in whole grains, fruits and vegetables (all rich in NSP) improve glucose tolerance and may slow the progression of impaired glucose tolerance into the full expression of type II diabetes (WHO, 2003).

Although most attention has focused on reducing intake of saturated fat for prevention of cardiovascular disease, increasing the intake of fish oils, foods high in linoleic acid and potassium (e.g. vegetables) NSP, antioxidants, calcium, folic acid and low to moderate alcohol consumption along with increased physical activity, may contribute to its prevention (Renaud and Lanzmann-Petithory, 2001; WHO, 2003). There is strong evidence that saturated fatty acids, high salt intake, being overweight or obese and a high alcohol intake increase the risk of cardiovascular disease. A high intake of dietary cholesterol and unfiltered boiled coffee may also increase risk (WHO, 2003).

Diet is an important determinant of risk for many cancers accounting for 30% of cancers in Westernized countries (Doll and Peto, 1996). There is strong evidence for an increased risk of cancer associated with overweight and obesity, high consumption of alcohol, aflatoxins and salted fermented fish. There is also evidence to show that consumption of hot drinks and foods and consumption of salt preserved items are associated with cancer. There is good evidence that a high consumption of fruits and vegetables protects against certain cancers.

Based on the present available evidence for an association between diet and chronic diseases the dietary recommendations for industrialized countries are to increase the consumption of fruits, vegetables and starchy staple foods, especially wholegrain varieties and to decrease the intake of fat, in particular saturated fat, and free sugars (Table 2).

The Impact of Oral Disease

Dental diseases impose both financial and social burdens; the treatment of dental caries is expensive for governments, costing between 5% and 10% of total healthcare expenditures exceeding the cost of treating cardiovascular disease, cancer and osteoporosis (Sheiham, 2001). Despite a low mortality rate associated with dental diseases, they have a considerable impact on self-esteem, eating ability, nutrition and health, both in childhood and older age. Dental decay may result in tooth loss, which reduces the ability to eat a varied diet and is in particular associated with a diet low in fruits, and vegetables and NSP (Moynihan et al, 1994; Steele et al, 1998). Tooth loss may therefore impede the achievement of dietary goals and the enjoyment of food.

Periodontal Disease

Apart from severe vitamin C deficiency, which results in scurvy-related periodontitis, there is at present little evidence for a strong association between diet and periodontal disease. The main overriding factor in the etiology of periodontal disease is the presence of plaque, and prevention measures focus on oral hygiene. There is some evidence to suggest that periodontal disease progresses more rapidly in undernourished populations and the important role of nutrition in maintaining an adequate host immune response may explain this observation (Enwonwu, 1995). A high sucrose intake is associated with increased plaque volume due to the production of extra cellular glucans, and there is a strong association between plaque volume and gingivitis. Human intervention studies have shown higher plaque volumes and increased gingivitis with high sucrose diets compared with low sucrose diets (Scheinin et al, 1976; Sidi and Ashley, 1984). However, the maximum reduction in sugar in the diet that is practically possible would not be sufficient to prevent the development of gingivitis (Gaengler et al, 1986).

Nutrition and Oral Infectious Diseases

Malnutrition impairs immune function of the host, thereby intensifying the severity of oral infections and may lead to their evolution into life-threatening diseases (Enwonwu et al, 2002). For example, malnutrition accelerates the progression of necrotizing periodontitis (NP) to life threatening noma. NP is a lesion affecting the interproximal gingival papillae that predominantly affects young children who are immunocompromised due to malnutrition and common tropical infections. If not promptly treated, NP and other oral inflammatory lesions in malnourished children may evolve into noma (cancrum oris). This is a dehumanizing oro-facial gangrene that destroys the soft and hard tissues of the oral and surrounding structures.

Nutrition and Oral Cancer

Oral cancer (cancers of the tongue, gums, floor of mouth and other mucosal surfaces, lips and salivary glands) is the fifth most common cancer in the world and the seventh most common cause of death from cancer (World

Cancer Research Fund, 1997). Tobacco use and alcohol are the main aetiological factors, although there is some evidence for a protective effect of some micronutrients and increasing evidence for a preventive role of fruits and vegetables.

There is some evidence for an association between deficiency of iron and oral cancer (Rich and Radden, 1984; Chyou et al, 1995; World Cancer Research Fund, 1997) although some results are conflicting making the overall evidence inconclusive. Likewise, despite a few associations between selenium status and oral cancer, results are inconclusive (World Cancer Research Fund, 1997). Antioxidant vitamins A, C, E and the carotenoids, may have a protective role by scavenging potentially mutagenic free radicals from damaged cells. There is convincing evidence for a protective role of vitamin C, a limited number of interventions studies to show a preventive effect of vitamin E, and good evidence that carotenoids prevent leucoplakia (pre-cancer) (World Cancer Research Fund, 1997). A meta-analysis (Macfarlane, 1993) investigating the effects of tobacco, alcohol, fruits and vegetables, macronutrients, vitamin C and fiber on oral cancer found that fiber and vitamin C status were inversely related to disease risk across study populations and the greatest reduction in risk from vitamin C, occurred in heavy smokers.

Vitamin supplementation studies have produced inconsistent results, suggesting the individual vitamins may not be the only bioactive component and could serve as a marker for other 'bioactive' substances found in the same food sources. The convincing evidence that diets high in fruits and vegetables offer protection against oral cancer supports this view. The protective effect of fruits and vegetables remains after adjusting for tobacco use and alcohol consumption. The evidence shows that the strongest protection is offered by fruit (World Cancer Research Fund, 1997). However, well designed dietary intervention studies that prospectively investigate the association between fruit and vegetable intake and risk of oral cancer are required.

The Effects of Diet and Nutrition on Enamel Defects

Nutritional status affects the teeth during the pre-eruptive stage and this subsequently may influence the susceptibility of the teeth to decay. However, the pre-eruptive nutritional influence is much less important that the post-eruptive local effect of diet on caries formation (Rugg-Gunn, 1993).

Developmental defects may be broadly classified into opacities which are areas of opaque enamel largely caused by excess fluoride ingestion, or 'hypoplasia' which manifests as pits, fissures or larger areas of missing enamel. Nutritional deficiency is just one cause of enamel hypoplasia (Pindborg, 1982). Most attention has focused disturbances of calcium and phosphorus metabolism and deficiencies of vitamins A and D and protein as nutritional causes of hypoplasia (Rugg-Gunn, 1993). Malnourished communities have a higher caries incidence than expected if compared with well nourished communities consuming similar levels of sugar, however, this may be due to nutritional influences on the salivary glands as well as enamel formation. Excessive ingestion of fluoride during the development of enamel causes enamel fluorosis (Rugg-Gunn et al, 1998). There is some evidence that malnutrition exacerbates fluorosis (Rugg- Gunn et al, 1998). Damage to the teeth during the development is permanent damage that affects the aesthetics of the teeth and the susceptibility to dental diseases.

Dental Caries

Despite the marked decline in dental caries in developed countries over the past 30 years, dental caries remains unacceptably high and is a major public health problem (Table 3). Available data show that in high income countries the mean DMFT at age 12 years is 2.1 (WHO, 2001).

However, even in countries with low average DMFT scores, dental caries still affects the majority of children. There is some indication that the favorable trends in dental caries levels have come to a halt (Fejerskov and Baelum, 1998).

In the absence of dietary sugars very little dental caries occur and dental caries was not a problem before widespread production and consumption of sugar. The development of caries requires sugars and bacteria to occur, but is influenced by the susceptibility of the tooth, the bacterial profile, quantity and quality of the, saliva, bacterial profile, and the time for which dietary sugars are in contact with oral bacteria.

Dietary Sugars and Dental Caries

There is a wealth of information on the role of sugars in the etiology of dental caries. The evidence comes from many different types of investigation (Table 4) including human, animal and in vitro studies. Together, information from each type of study provides an overall picture of the cariogenic potential of foods containing sugars and other carbohydrates. In this report paper the term 'sugars' refers to all mono and disaccharides while the term 'sugar' only refers to sucrose, the term 'free sugars' refers to all mono and disaccharides added to foods by manufacturer, cook or consumer, plus sugars naturally present in honey, fruit juices and syrups.

Worldwide Ecological Studies

Sugar intake and levels of dental caries can be compared at a between-country level. Sreebny (1982) corre-

lated the dental caries experience of 12-year-olds to sugar supplies data of 47 countries observing a significant correlation (r = + 0.7) and indicating that 52% of the variation in caries levels could be explained by the per-capita availability of sugar (Fig 1). From these data Marthaler calculated that for every 25 g of sugar per day one tooth per child would become decayed, missing or filled (Marthaler, 1990). A later analysis by Woodward and Walker (1994) did not find a significant association between per capita sugar availability and caries levels for developed countries (however, overall, sugars availability still accounted for 28% of the variation in caries levels), possibly because with such high sugar intakes in these countries, changing the level of sugar intake by a few kilograms per year will not influence the caries challenge (Nadanovsky, 1994).

Observations of Populations Following a Change in Diet

As populations move away from their traditional diets and adopt a more Westernized diet, high in free sugars, a marked increase in caries is observed. Examples of this are the North American Eskimos, Greenlanders, Indians and the inhabitants of the Island of Tristan da Cunha (Fisher, 1968). The Islanders of Tristan da Cunha had exceptionally low level of caries in 1937 when they were an isolated, self-sufficient community with a low sugars intake of only 1.8 g/person/day. From the 1940 s they began to trade with outside communities and sugar was introduced into the diet. By 1966, the average daily intake of sugar was 150 g per person, and the prevalence of dental caries had increased over ten-fold (Fig 2). Since refined flour was introduced at the same time as sugar, it is not possible to make a clear distinction between the effects of sugar and starch, however, their traditional diet was high in starch, in the form of cooked potato, which suggests that sugar had the most marked effect.

Observations of Diets before, during, and after the Second World War

Food rationing during the years of the Second World War resulted in people consuming a diet that was low in sugars. Data from Europe and Japan indicate that this was associated with a marked reduction in caries prevalence which subsequently increased again when restrictions were lifted (Sognnaes, 1948; Takeuchi, 1961; Marthaler, 1967). Fig 3 shows data from Japan for sugars intake and dental caries levels before, during, and after the Second World War (Takeuchi 1961). More recently, Miyazaki and Morimoto (1996) reported a significant correlation (r = + 0.91) between sugar availability in Japan and DMFT at age 12 between 1957 and 1987.

Table 3 Mean DMFT of 12-year-old persons in European countries for which post 1997 data are available

Country	Year	DMFT
Austria	1997	1.7
Belarus	2000	2.7
Belgium	1998	1.6
Bosnia & Herzegovina	2001	6.1
Bulgaria	2000	4.4
Croatia	1999	3.5
Czech Republic	1998	3.4
Denmark	2001	0.9
Estonia	1998	2.7
Finland	1997	1.1
France	1998	1.9
Germany	2000	1.2
Greece	2000	2.2
Ireland	1997	1.1
Latvia	2000	3.9
Macedonia	1999	3.03
Netherlands	1998	0.6
Norway	1999	1.5
Poland	2000	3.8
Portugal	2000	2.95
Romania	1998	7.3
Slovakia	1998	4.3
Slovenia	1998	1.8
Sweden	2001	0.9
Switzerland	1996	0.8
United Kingdom	2000-1	0.9
Romania	1996	3.8

Source: WHO Global Oral Health Data Bank (WHO, 2001)

Table 4 The different types of experiment data from which collectively provides evidence for an association between diet and dental health

Experiments in vitro
Plaque pH experiments in human volunteers
Enamel slab experiments in human volunteers
Animal studies
Human observational studies
Human intervention studies

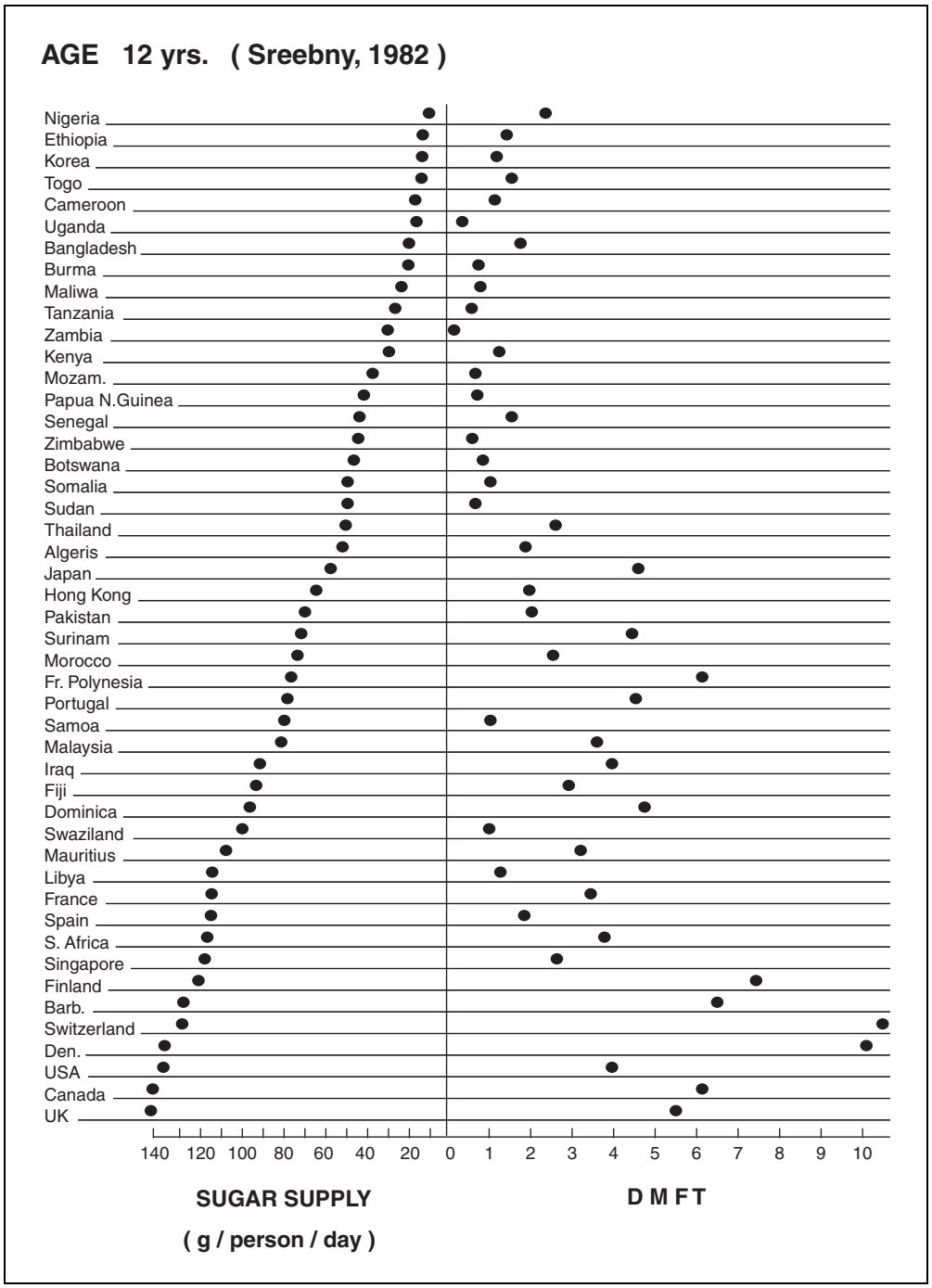

Fig 1 Dental caries experience (DMFT) of 12 year old children plotted against sugar supply data (g/person/day) in 47 countries. Data from Sreebny (1982), reproduced with permission of the editor of Community Dentistry and Oral Epidemiology (Blackwell Publishing).

High and Low Sugar Consumers

Evidence for a link between sugars intake and caries also comes from observations of groups of people that habitually consume either high or low levels of sugars. Low dental caries experience has been reported in groups of people with a habitually low sugars intake, e.g. children in institutions where with strict dietary regimens are inflicted (Harris, 1963) and children with hereditary fructose intolerance (Newbrun et al, 1980).

Children living in the Hopewood House children's home in Australia followed a strict lacto vegetarian diet that was low in sugars and refined flour. Their oral hygiene was virtually absent and fluoride exposure was low. Dental caries levels were monitored annually between 1947 and 1962 and were much lower than children attending

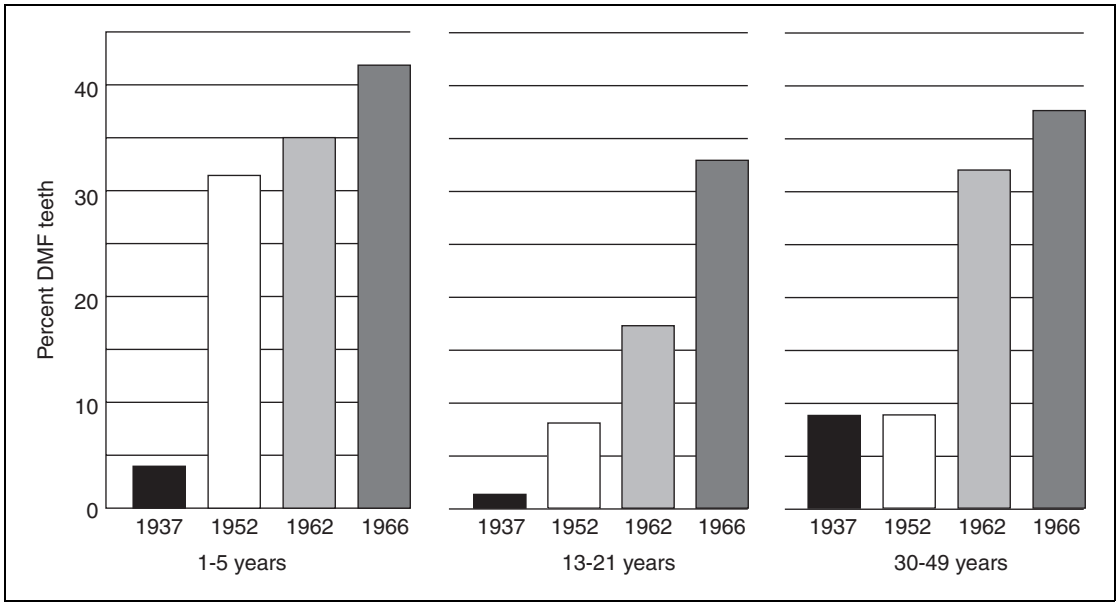

Fig 2 Severity of dental caries (per cent DMFT/dmft) in three age groups of islanders of Tristan da Cunha, St Helena, at four examinations between 1937 and 1966. Reproduced from Rugg-Gunn 1993, by kind permission of Oxford University Press.

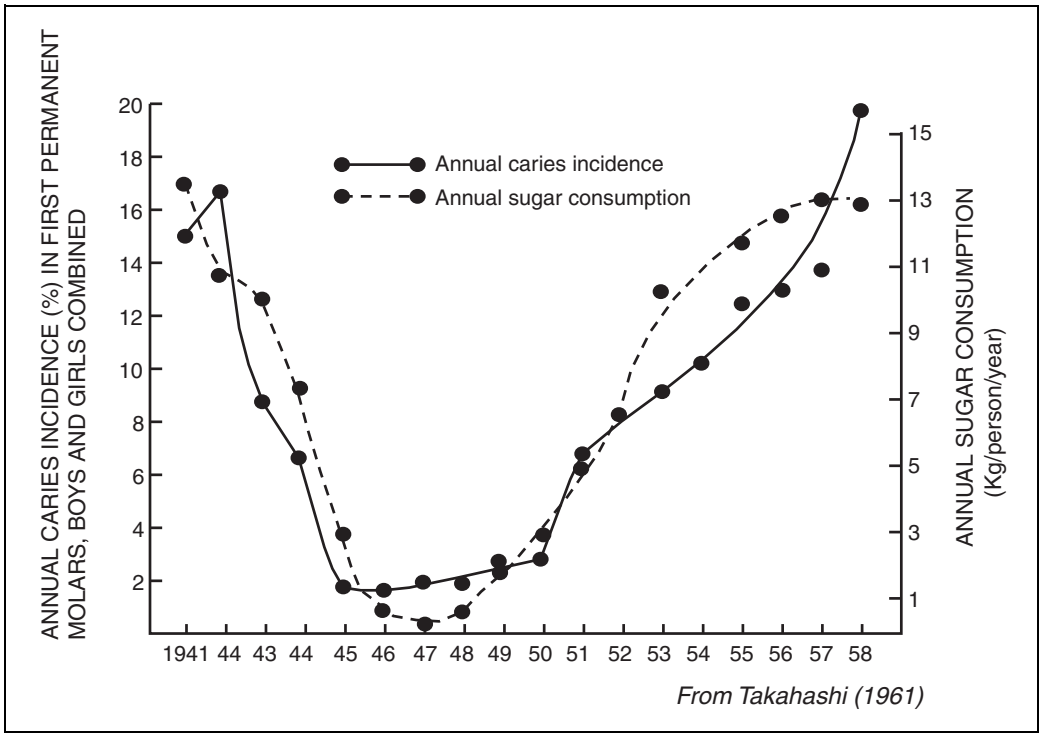

Fig 3 The relationship between the annual caries incidence in first permanent molar teeth in over 7000 Japanese children and annual sugar consumption in Japan. Data from Takahashi (1961). Reproduced by kind permission of Professor J J Murray

state schools in New South Wales. Forty-six per cent of 12-year-olds in Hopewood House were caries-free compared with only 1% of the children from state schools. However, after 12 years of age when the children left the home the rate of caries increased to levels observed in children from the state schools (Harris, 1963).

A weakness of the data from observations of populations is that changes in intake of sugars are often asso-

ciated with changes in the intake of refined flour, so attributing changes in dental caries solely to changes in the intake of sugars is not possible. An exception to this is children with Hereditary Fructose Intolerance (HFI), a congenital deficiency of fructose-1-phosphate aldolase in which fructose (and therefore sucrose) is excluded from the diet. People with HFI have a low intake of sugars but, as glucose is tolerated do not have a restriction on intake of starch. Studies have shown that subjects with HFI have a low intake of sugars and a higher than average intake of starch yet a low caries experience (Newbrun et al, 1980; Petersen, 1983).

People with high intakes of sugars such as confectionery industry workers (Anaise, 1978; Petersen, 1983) and children with chronic diseases requiring long-term sugar-containing medicines (Roberts and Roberts, 1979) have been shown to have high caries levels. Earlier observations were from populations not exposed to the benefits of fluoride, however, relatively more recently, Danish chocolate factory workers were found to have significantly higher dental disease than ship-yard workers (Petersen, 1983). Similar findings were reported by Masalin in a study of sweet biscuit factory workers in Finland (Masalin et al, 1990).

Cross-sectional Studies

Dental caries develops over time and therefore simultaneous measurements of disease levels and diet may not give a true reflection of the role of diet in the development of the disease. It is the diet several years earlier that may be responsible for current caries levels. Cross-sectional studies should therefore be interpreted with caution. However, the diets of young children may not have changed significantly since the eruption of the teeth. A UK survey of children aged 1.5 to 4.5 years showed an association between high intake of confectionery and soft drinks and dental caries (Hinds and Gregory, 1995). However, Marques and Messer (992) failed to show an association between sugars intake and dental caries in the primary dentition.

Rugg-Gunn (1993) summarized numerous cross-sectional studies that compare sugars intake with dental caries in the permanent dentition. Nine out of 21 studies that investigated a relationship between amount of sugars consumed and dental caries found a significant relationship and the remaining 12 did not. Twenty-three out of the 37 studies investigating the relationship between frequency of sugars consumption and caries levels found a significant relationship and 14 did not. More recently, the UK National Diet and Nutrition Survey of young people aged 4 – 18 years, found no relationship between amount of free sugars consumed and levels of dental caries. However, in the 15 to 18 year old group, those in the upper band of intake of free sugars were more likely to have decay than those in the lower band (70% compared with 52%) (Walker et al, 2000).

Longitudinal Studies

When investigating the association between diet and the development of dental caries it is best to relate sugars consumption to changes in dental caries over time in a longitudinal design. Due to the cost and time implications of this type of study, longitudinal studies are relatively rare.

Stecksen-Blicks and Gustafsson (1996) measured caries increment over one year in 8 and 13-year-old children and related it to diet at one time point. Despite the short period of observation, a significant relationship between caries development and intake of sugars was found for both the primary and permanent dentition. In a comprehensive study of caries and diet of over 400 English adolescents aged 11 to 12 years (Rugg-Gunn et al, 1984) a small but significant relationship was found between intake of total sugars and caries increment over two years ($r = + 0.2$). In this study, those with the highest levels of sugars intake developed significantly more caries than those with lower sugars intakes. Individuals who developed no caries during the experimental period consumed less confectionery and fewer sugared hot drinks than average.

The Michigan Study was carried out in the USA between 1982 and 1985 and investigated the relationship between sugars intake and dental caries increment in children initially aged 10 to 15 years (Burt et al, 1988). This study also found a significant relationship between the amount of dietary sugars and dental caries. Children who consumed a higher proportion of their total dietary energy as sugars had a higher caries increment. The amount of sugars eaten in between meals was also related to caries. Intake of sugars was generally high for all subjects in this study with only 20 out of 499 children consuming less than 75 g/day. In the Michigan Study, the reason for the low relative risks of caries development in the high sugars consumers was that small variances were found both for caries increment and intake of sugars (Burt and Szpunar, 1994).

Marthaler (1990) conducted an extensive review to address whether the relationship between dietary sugars and caries activity is vanishing in developed countries with high sugars availability and wide exposure to fluoride. He concluded that in modern societies that make use of prevention a relationship between sugars consumption and caries activity still exists. Marthaler pointed out that many older studies failed to show a relationship between sugars intake and development of dental caries because many of these were of poor methodological design, used unsuitable methods of dietary analysis and were of insufficient power (Marthaler, 1990). Correlations between individuals' sugars consumption and dental caries increments may be weak due to the limited range of sugars intake in the study population, i.e. variation in sugars intake within populations is too low to show an effect on caries occurrence.

Intervention Studies

Human intervention studies, in which diet is altered and caries is monitored over a period of time, give the most accurate assessment of the effects of diet on caries. However, such studies are rare and those that have been reported are from decades ago – often before the use of fluoride was widespread. Nonetheless, two such studies provide strong evidence for a link between the intake of dietary sugars and the development of dental caries. The first was the Vipeholm study (Gustafsson et al, 1954) which was conducted in an adult mental institution in Sweden in the late 1940 s before the strong association between sugars and caries was established. The aim of the study was to investigate the effects of consuming sugary foods of varying stickiness and at different frequencies (mealtimes only vs meal times and in between meals) on caries development. It was found that sugar had little effect on caries increment if it was ingested up to a maximum of 4 times a day and at mealtimes only. However, consumption of sugar in-between meals was associated with a marked increase in dental caries. It was also found that caries activity disappeared on withdrawal of sugar from the diet. It would not be possible to repeat such a study today, as it would be unethical to prescribe high sugars diets knowing of the association between sugars and dental caries.

The second intervention study, the Turku Sugars Study (Scheinin et al, 1976), was carried out in Finland in the 1970 s on adult volunteers who consumed a diet in which all of the sucrose was replaced with either xylitol or fructose. A third group of subjects consumed a normal sucrose-containing diet and acted as a control. Three groups of subjects (n = 125) aged 12 to 53 years, with 65% being in their twenties, consumed a diet sweetened with sucrose, fructose or xylitol for a period of 25 months. Changes in the levels of dental caries were measured over the study period. The results showed an 85% reduction in dental caries in the xylitol group who had removed sugar from their diet. The findings are summarized in Table 5. Overall, the conclusions of the Turku Study are that substitution of sugar with xylitol virtually abolished caries activity.

Table 5 Findings of the Turku Study (Scheinin et al, 1976)

Group	DMFS (inc. pre-cavitation)	DMFS (cavities only)
Sucrose	7.2	3.33
Fructose	3.8	3.57
Xylitol	0	1.47

Frequency versus Amount of Sugars

The importance of frequency versus the total amount of sugars is difficult to evaluate as the two variables are hard to distinguish from each other. Data from animal studies have indicated the importance of both frequency (Konig et al, 1968; Firestone et al, 1984) and amount (Mikx et al, 1975; Hefti and Schmid, 1979) of sugars intake in the development of dental caries.

Some human studies have shown that the frequency of sugars intake is an important factor for caries development including the aforementioned Vipeholm study that showed caries increment was relatively low when intake of sugars was consumed up to four times a day at mealtimes only (Gustafsson et al, 1954). More recent studies have also shown caries development to be lower when intake of sugars does not exceed 4 times a day. In a study of 5-year-old Icelandic children, those reporting 4 or more intakes of sugars per day or 3 or more between-meal snacks per day, had markedly increased caries scores compared with children who consumed sugars less frequently (Holbrook et al, 1989; Holbrook et al, 1995). In a longitudinal study of English pre-school children, DMFT was higher (DMFT 1.69) in children who had four or more sweetened snacks and drinks a day compared with children who only had them once a day (DMFT 1.01) (Holt, 1991). The studies above suggest that if free sugars intake is limited to a maximum of four times a day, caries levels will be reduced.

Several longitudinal studies show amount of sugars intake to be more important that frequency (Rugg-Gunn et al, 1984; Burt et al, 1988; Szpunar et al, 1995). Kleemola-Kujala and Rasanen (1982) found that dental caries increased with increasing amount of sugars consumption only when oral hygiene was poor.

There is a strong correlation between the amount and frequency of sugars consumption. In an analysis of dietary data from over 400 11 – 12-year-old children, Rugg-Gunn found the correlation between frequency of intake of sugar-rich foods and total weight consumed to be + 0.77 (Fig 4) (Rugg-Gunn et al, 1984). Other investigators have also reported a strong association between frequency and amount of sugars consumption hence supporting the view that both the frequency and the amount of sugar intake are important.(Cleaton-Jones et al, 1984; Ismail et al, 1984; Jamel et al, 1996).

Type and Form of Sugars

There is no evidence to show that, with the exception of lactose, the cariogenicity of mono and disaccharide differs. For example the aforementioned Turku study showed no difference between the cariogenicity of sucrose compared with fructose (Scheinin et al, 1976). Therefore, all free sugars pose a threat to teeth.

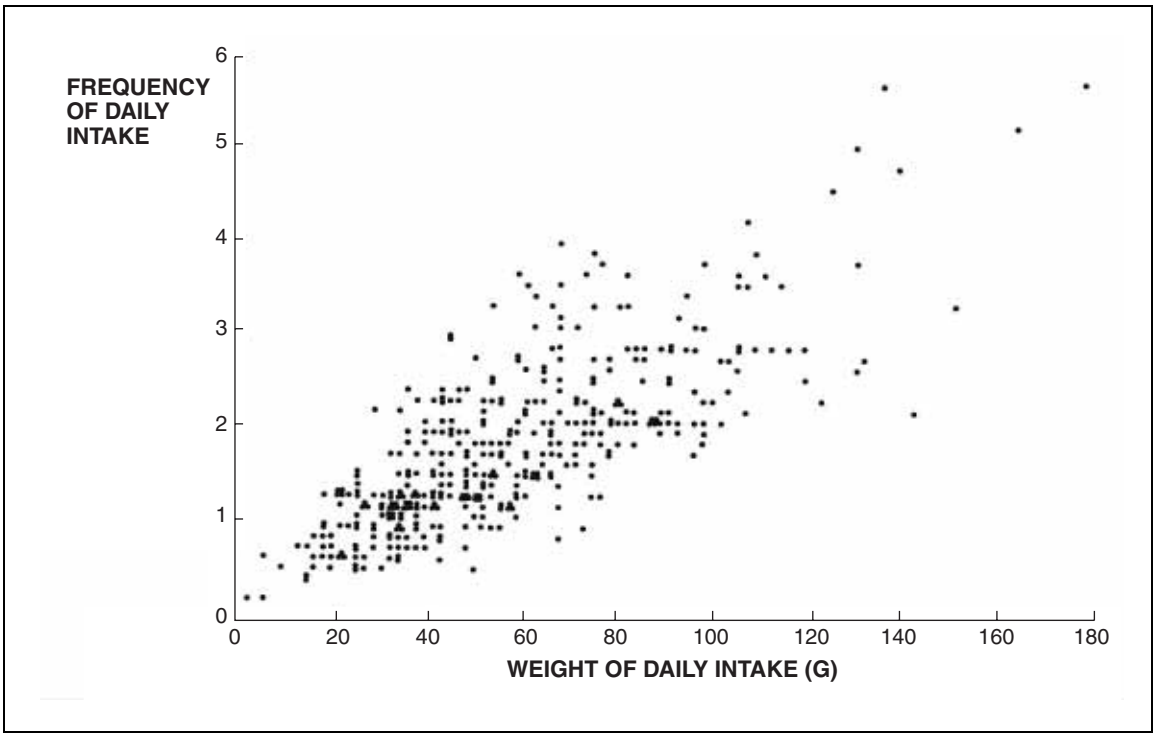

Fig 4 Graph to illustrate the relationship between frequency of intake against amount of intake of confectionery by 405 12 – 14 year old English children. Data from Rugg-Gunn (1984). Reproduced with kind permission of Professor Andrew Rugg-Gunn.

The adhesiveness or 'stickiness' of a food is not necessarily related to either oral retention time or cariogenic potential, as there is evidence to show that the amount and frequency of consumption of high sugars drinks (with low stickiness/oral retention) are associated with increased risk of dental caries (Ismail et al, 1984; Jamel et al, 1996).

Time of Day

Research has indicated that the worst time to consume free sugars is close to bedtime. This is due to the low salivary flow rate during sleep. Associations between consumption of free sugars at bedtime and dental caries have been reported in children (Hinds and Gregory, 1995; Walker et al, 2000). Recent data has shown children that consume sugary foods and drinks close to bed time have been shown to have four times the number of decayed teeth compared with children that did not have sugary items within an hour of bedtime (Pitts et al, 2003).

The Influence of Fluoride on the Sugars-caries Relationship

The inverse relationship between fluoride in drinking-water and dental caries is well established and widespread use of fluoride largely accounts for the decline in dental caries that has been observed in many countries over the last 30 years. However, fluoride alone does not eliminate dental caries.

More recent studies on the relationship between sugars and caries are confounded by the presence of fluoride but show that a relationship between sugars intake and caries still exists. Longitudinal studies have shown the relationship between sugars intake and caries increment remains even after controlling for use of fluoride and oral hygiene practices (Rugg-Gunn et al, 1984; Burt et al, 1988). A relationship between frequency of sugars-rich snacks and caries levels in young children has been found to exist even after controlling for use of fluoride (Holt, 1991). Weaver (1950) reported on the decline in caries levels during the Second World War sugars restrictions in 12-year-old children from areas with naturally high and low water fluoride concentration in North East England. Caries levels were lower in the high fluoride area in 1943, but by 1949 fell a further 54% indicating that exposure to fluoride did not totally override the effect of sugars in the diet. Kunzel and Fischer (Kunzel and Fischer, 1997) also showed that the beneficial effects of fluoride vary according to levels of sugars consumed. Marthaler (1990) concluded that, even when there is exposure to fluoride, a relationship between sugars intake and caries exists and on reviewing the literature on declines in caries and associated factors stated

Table 6 Systematic review of sugars intake and dental caries: Distribution of 36 papers showing strong, moderate and weak relationships between sugars intake and dental caries by type of study design (Burt and Pai, 2001)

	Strong	Moderate	Weak	Totals
Cohort studies	1	7	4	12
Case-control studies	0	1	0	1
Cross-sectional studies	0	11	12	23
Totals	1	19	16	36

that "within modern societies which are aware and make use of prevention, the relation between sugars consumption and caries activity still exists." He also concluded that "recent studies have demonstrated that sugar – sucrose as well as other hexoses – continues to be the main threat for dental health of: 1) whole populations some developed and many developing countries; 2) for the individual in both developed and developing countries; and 3) in spite of the progress made in using fluorides and improved oral hygiene." It is likely that, in industrialized countries where there is adequate exposure to fluoride, a further reduction in the prevalence and severity of dental caries will not be achieved without a reduction in free sugars intake.

Burt and Pai (2001) conducted a systematic review that aimed to answer the question "in the modern age of extensive fluoride exposure, do individuals with a high level of sugars intake experience greater caries severity relative to those with a lower level of intake?" The review included papers published between 1980 and 2000 that met the pre-determined inclusion criteria and a threshold for scientific merit – a total of 36, were included in the final analysis (Table 6). No paper failed to find a relationship between sugars intake and caries. The conclusions were that: 1) where there is good exposure to fluoride, sugars consumption is a moderate risk factor for caries in most people; 2) sugars consumption is likely to be a more powerful indicator for risk of caries in persons who do not have regular exposure to fluoride; and 3) with widespread use of fluoride, sugars consumption still has a role to play in the prevention of caries but this role is not as strong as it is without exposure to fluoride.

What is the Safe Threshold for Free Sugars Consumption?

It has been repeatedly observed that in countries/populations with an intake of free sugars below 15 – 20 kg kg/person per year (equivalent to 40 – 55 g/person/day) experience of caries is consistently low (Sreebny, 1982; Woodward and Walker, 1994; Sheiham, 2001; World Health Organization, 2003). As intake exceeds 15 kg per person per year dental caries intensifies. Table 7 summarizes the evidence for limiting the amount of free sugars consumption.

Potential Impact of Free Sugars Reduction on Other Dietary Components

Dietary advice for dental health must not conflict with dietary advice for general health and therefore, it is important to consider the potential impact of a reduction in free sugars on other components of the diet. There are some reports from cross-sectional analysis of dietary data of populations that show an inverse relationship between the intake of free sugars and the intake of fat (Gibney, 1995), suggesting that reducing free sugars might lead to an increase in fat intake. There is, however, an increasing evidence base from studies that have monitored dietary changes over time that shows that changes in intake of fat and free sugars are not inversely related, and that reductions in intake of dietary fat have occurred and have been offset by an increase in intakes of starch and not free sugars (Fletcher et al, 2001; Alexy et al, 2002).

Starches and Dental Caries

Dietary starch is heterogeneous in nature, may be consumed cooked or raw and may vary in degree of refinement. These factors need to be considered when assessing the cariogenicity of foods that contain starch. Current dietary guidelines encourage the consumption of starch-rich staple foods, e.g. bread, potatoes, pasta and rice, and fruit and vegetables that naturally contain starch. It is therefore important that the cariogenic effects of these types of starch are understood, to ensure that dietary recommendations for general health are not contraindicated in terms of dental health.

Table 7 Summary of the evidence for low levels of intake of free sugars

kg/person/year	Author/year	Caries levels	Source of data
0	Turku study (1976) Scheinin et al, (1976)	56% less caries when xylitol replaced sucrose	Intervention study, Finland
0.2 – 15	Takahashi (1961)	Annual caries increment was positively related to sugars when annual sugar intakes ranged from 0.2 – 15 kg. (r = + 0.8)	Data from Japan
10 – 15	Sheiham (1983)	Caries low	WHO database
< 10 > 15	Takeuchi (1962)	Seldom caries in first two post eruptive years in first molars Caries occurred in first post eruptive year and intensified	Data from Japan 1941 – 1958
> 12.3	Rodriguez (1997)	Caries in deciduous dentition 29% more likely	Preschool children in Brazil
< 10 < 18.25	Woodward and Walker (1994)	78% of countries had DMFT < 2 and 30% with > 10 kg per year had DMFT < 2.0 DMFT < 3 in 23/26 countries	WHO database
< 10	Knowles (1946)	> 50% of 3 – 7 year-olds were caries free	War-time data children in Jersey/ England
< 15	Miyazaki and Morimoto (1996)	DMFT < 3.0	Data from Japan 1945 – 1987
10.4	Schulerud (1950)	Good dental health in 6 – 12-year-old children	War-time data from Norway
< 20	Buttner (1979)	Caries very low	Data from 18 countries in 1959
< 18.25	Sreebny (1982)	DMFT < 3.0	WHO database

Experiments in vitro and animal experiments have shown that raw starch is of low cariogenicity (Grenby, 1963; Grenby, 1970; Brudevold et al, 1985), cooked starch is about one-third to one-half as cariogenic as sucrose (Koulourides et al, 1976; Bowen et al, 1980; Firestone et al, 1982), however, mixtures of starch and sucrose are as cariogenic as sucrose alone (Firestone et al, 1982). Plaque pH studies, using an indwelling oral electrode, have shown starch-containing foods to reduce plaque pH below 5.5, but starches are less acidogenic than sucrose. Plaque pH studies measure acid production from a substrate rather than caries development, and take no account of the protective factors found in some starch-containing foods or the effect of foods on stimulation of salivary flow. There is no evidence from human epidemiological studies that consumption of staple starchy foods is associated with dental caries. For example, before the introduction of sugar, the inhabitants of the Island of Tristan da Cunha had a low prevalence of caries, despite having a diet that was high in cooked starch in the form of cooked potato (Fisher, 1968). People with HFI tend to have an above average intake of starch but a low level of caries (Newbrun et al, 1980). In the Turku study, restricting the intake of sucrose alone, not restricting the intake of starch, resulted in a low caries increment (Scheinin et al, 1976). Most studies of populations with low intakes of sugars and high intakes of starch have reported low caries levels (Alfonsky, 1951; Russell et al, 1960). In Norway and Japan the intake of starch increased during the Second World War yet dental caries declined. Overall, dietary recommendations to reduce the intake of free sugars and increase the intake of starchy staple foods are unlikely to cause an increase in dental caries.

Other Carbohydrates

Glucose polymers (maltodextrins and glucose syrups) are produced from starch and are used in products such as soft drinks, infant food and drinks, soya infant formula, sports drinks, desserts, confectionery and energy supplements. In medical practice, glucose polymers are commonly added to soup, drinks, mashed potato, porridge and puddings to increase the energy content.

In theory, glucose polymers have the potential to cause dental caries as oral bacteria can utilize some of the shorter-chain glucose polymers and salivary amylase may also hydrolyse glucose polymers, to form shorter chains that are readily metabolized. Information on the cariogenic potential of glucose polymers is limited and no clinical trials have been conducted. Grenby (1972) observed no difference in caries development between rats fed diets containing either glucose syrups or sucrose, although glucose syrups were significantly less cariogenic than sucrose when given to rats in a solution. Plaque pH studies in human volunteers have shown that maltodextrins depress plaque pH to a lesser extent than sucrose (Moynihan et al, 1996a).

Glucose syrups are present in place of lactose in soya infant formula, raising concern about its cariogenic potential. Soya infant formula contains free sugars and lower concentrations of calcium and phosphate than standard infant milk. Plaque pH studies have shown no significant difference in acidogenic potential between soya infant formula and standard infant milk (Moynihan et al, 1996b). However, infants with intolerance to lactose or cows' milk protein may remain on this formula for a number of years, whereas other young children can consume cows' milk (which has a lower sugars content and is higher in protective factors) from 12 months. Early weaning onto a cup and good oral hygiene are of particular importance to infants fed soya infant formula.

Fruit and Dental Caries

When consumed as part of a mixed human diet there is little evidence showing that fruit is an important factor in the development of dental caries (Martinsson, 1972; Clancy et al, 1977; Rugg-Gunn et al, 1984). A number of plaque pH studies have found fruit to be acidogenic, but less so than sucrose (Ludwig and Bibby, 1957; Imfeld, 1983; Hussein et al, 1996). Animal studies have shown that when fruit is consumed in very high frequencies (e.g. 17 times a day) it may cause caries (Imfeld et al, 1991; Stephan, 1966) but the extent of this is much less than that observed with sucrose. Recent research has shown that apple polyphenols have an antibacterial nature and that when they are extracted and added to animal feeds they reduce caries development (Cordeiro et al, 2000). Clinical trials of the effects of apple consumption on dental caries have produced equivocal results (Slack and Martin, 1958; Averill and Averill, 1968). Only one epidemiological study has reported an association between fruit consumption and dental caries (Grobler and Blignaut, 1989). In this study of fruit farm workers, fruit intakes were very high (e.g. 8 apples or 3 bunches of grapes per day) and DMFT of the workers was higher compared with grain farm workers. However the difference in DMFT arose solely from differences in the numbers of missing teeth the cause of which was not ascertained.

Dietary Factors which Protect against Dental Caries

Some dietary components protect against dental caries. Dairy products without added sugars have long been considered to be safe for teeth and milk was one of the first foods that was discovered to be cariostatic (Sperling et al, 1955). Cows' milk contains calcium, phosphorus and casein, all of which inhibit caries. Several studies have shown that the fall in plaque pH following cows' milk consumption is negligible (Rugg-Gunn et al, 1985; Frostell, 1970). Evidence from animal experiments shows cows' milk is anti-cariogenic; cows' milk reduces the cariogenicity of sucrose-containing foods (Bibby et al, 1980; Reynolds and Johnson, 1981; Thompson et al, 1984). Human observational studies have shown an inverse relationship between the consumption of cows' milk and caries increment in children.

Human milk has a higher lactose content (~ 7%) and lower concentrations of calcium and phosphate than cows' milk, yet epidemiological studies have associated breastfeeding with low levels of dental caries (Holt et al, 1982; Silver, 1987). There have been a few specific case studies of early childhood caries in infants that received prolonged ad libitum and nocturnal breastfeeding. An additional advantage of breastfeeding is that it does not necessitate the use of a feeder bottle to which additional free sugars may be added.

The cariostatic nature of cheese has been demonstrated in several experimental studies (Rugg-Gunn et al, 1975; Moynihan et al, 1999), human observational studies (Rugg-Gunn et al, 1984), and intervention studies (Gedalia et al, 1994). Hard cheeses are good gustatory stimulants and consumption raises plaque calcium concentrations, aiding remineralization (Rugg-Gunn et al, 1975; Moynihan et al, 1999). In a controlled clinical trial of children, Gedalia and colleagues (Gedalia et al, 1994), demonstrated that eating a 5 g piece of hard cheese daily, following breakfast, for a period of two years, resulted in the development of significantly fewer caries.

Consumption of wholegrain foods may protect against decay. Such foods require more mastication thereby stimulating increased saliva flow. Peanuts and chewing gum are also salivary stimulants. Foods of plant origin contain organic phosphates, inorganic phosphates, polyphenols

Table 8 Non-sugar sweeteners permitted for food use

Bulk sweeteners	Intense sweeteners
☐ Sorbitol	☐ Saccharin
☐ Mannitol	☐ Aspartame (NutraSweet, Canderel)
☐ Xylitol	☐ Acesulfame K (Sunett)
☐ Maltitol	☐ Thaumatin
☐ Lactitol	
☐ Isomalt (Palatinit)	
☐ Hydrogenated glucose syrup (Lycasin)	

Table 9 Dietary acids

Acid	Example of food source
Citric	Citrus fruit/juices
Malic	Apples/apple juice
Phosphoric	Soft drinks, e.g. cola
Ascorbic (vitamin C)	Vitamin C tablets
Tartaric	Grapes, soft drinks
Carbonic	Sparkling drinks
Oxalic	Rhubarb

and phytate which may have some protective effects against caries. Both organic and inorganic phosphates have been found to be effective in animal studies, but studies in humans have produced inconclusive results (Nizel and Harris, 1964; Craig, 1975).

Due to recognition that sugars cause caries there has been a growth in the development of non-cariogenic sweeteners and their use in drinks, confectionery and chewing gums – foods otherwise high in free sugars that tend to be consumed between meals. Non-sugar sweeteners include 'bulk sweeteners' (mostly sugar alcohols) that add sweetness, volume and calories to products, and 'intense sweeteners' that only add sweetness to a product and contain negligible calories (Table 8). Numerous studies including plaque pH studies, animal studies and clinical trials have shown that non-sugars bulk sweeteners are safe for teeth. Intense sweeteners are not chemically related to sugars and are therefore non-cariogenic. In 1989 a UK Department of Health report concluded that non-sugars sweeteners are non-cariogenic, or virtually so, and that substitution of sugars with non-sugars sweeteners could substantially reduce caries development. It also suggested that these products would be most beneficial if they are used to replace the sugars in the foods that are ingested most frequently (Department of Health, 1989).

A successful use of non-cariogenic sweeteners has been chewing gum. There has been much interest in the cariostatic nature of xylitol, a sugar alcohol that is non-fermentable by most oral microorganisms (Edwardsson et al, 1977) and which is bacteriostatic. Several trials of xylitol containing chewing gums have shown a positive effect in reducing dental caries (Scheinen et al, 1975; Isokangas et al, 1988, Kandelman and Gagnon, 1990; Mäkinen et al, 1995).

Dental Erosion

Dental erosion is the progressive irreversible loss of dental hard tissue that is chemically etched away from the tooth surface by extrinsic and/or intrinsic acids by a process that does not involve bacteria. There is a perception that the prevalence of dental erosion is increasing especially in westernized countries. Dietary acids (Table 9) are a major cause of erosion and the large increase in the consumption of soft drinks is thought to be largely responsible. Carbonic acid is, however, the least erosive dietary acid and it is not just the effervescent nature of the drink that is the aetiological factor. Human observational studies have shown an association between dental erosion and the consumption of acidic drinks and foods, including fruit juice, soft drinks (including sports drinks), foods containing vinegar, and some fruits (Stabholz et al, 1983; Linkosalo and Markkanen, 1985; Jarvinen et al, 1991; Millward et al, 1994). In the UK where soft drink consumption has increased 800 fold in the last 50 years, over half of teenagers have erosion to their permanent teeth and age-related increases in dental erosion have been shown to be greater in those with the highest intake of soft drinks (Walker et al, 2000). Animal studies have shown that fruit and soft drinks cause erosion (Holloway et al, 1958; Stephan, 1966), although fruit juices are significantly more destructive than whole fruit (Miller, 1950; Grenby et al, 1990). Many countries are not yet largely affected by dental erosion and there are a number of approaches for preventing it. For example, some makers of soft drinks are trying to reduce the erosive nature of their drinks. In vitro studies have shown drinks to which calcium is added have reduced erosive and acidogenic potential (Hughes et al, 1999).

The Effects of Tooth Loss on Diet, Nutrition and Health

In older people dental impairment may result in a poor diet low in fruit and vegetables, fiber and vitamin C (Moynihan et al, 1994; Steele et al, 1998). This may predispose to increased risk of diet related diseases such as cardiovascular disease and cancer. Tooth loss is sometimes perceived as a diminishing problem yet the majority of those aged 65 years and over has no teeth (WHO, 2001) and many have fewer than the 21 that are thought to be necessary for adequate chewing function. Additionally the number of people aged over 65 years is increasing in many countries; for example in Europe it is estimated that the number of people aged 65 years and above will increase by 82% over the next 25 years. So tooth loss and its associated problems for diet, health and quality of life are unlikely to be eliminated in the foreseeable future. Effective means of dietary intervention in those with compromised dental function need to be identified and implemented.

Summary of Scientific Evidence

In summary, the scientific evidence relating to diet and oral diseases supports the following in oral health promotion:

At present there is no strong evidence for a role of dietary intervention in the prevention and/or treatment of periodontal disease. However, a healthy diet, rich in fruits and vegetables and micronutrients will help maintain an effective immune system necessary for the host defense against inflammation and infection. Prevention of malnutrition in impoverished societies is of paramount importance in the prevention of oral infectious diseases including noma.

Deficiencies of vitamin D and malnutrition may cause enamel developmental defects and salivary gland defects which may increase susceptibility to decay. However, in developed countries, such deficiencies are rare and oral health promotion should focus on the local effect of diet in the mouth which is of relatively much greater importance.

A wealth of evidence shows an association between free sugars consumption and dental caries and indicates that limiting the consumption of free sugars remains an important part of caries prevention. Oral health promotion should include means to reduce the amount and frequency of consumption of free sugars. Intake of free sugars should not exceed 10% of energy intake (~ 55 g per day) and the number of intakes of foods containing free sugars should be limited to four times per day.

Highly refined starches may also cause dental caries and if mixed with free sugars they may be as cariogenic as sugar alone. However, there is no evidence that starch-rich staple foods such as bread, rice and other cereals cause dental caries. Oral health promotion should aim to reduce the consumption of foods high in refined starch and sugars and promote increased consumption of staple starchy wholegrain foods.

Fruit is high in essential micronutrients and fiber and has been shown to be protective against oral cancer. There is no strong evidence for an association between fruit consumption and dental caries; fruit is a good stimulant of salivary flow and contains factors that protect against dental decay. High levels of consumption coupled with unusual dietary practices (e.g. sucking lemons) may be associated with dental erosion. However, normal fruit consumption as part of a mixed diet is unlikely harm dental health.

There is good evidence for an association between consumption of acidic drinks and dental erosion. Oral health promotion should encourage drinks that are safe for teeth, including milk and water and encourage a reduction in frequency and amount of soft drink consumption.

Tooth loss impedes the ability to consume a healthy diet adequate in fruits, vegetables and fiber. Prosthetic rehabilitation alone may not provide sufficient drive to change diet and such patients would potentially benefit from dietary advice.

Dietary Advice in Dental Practice

The dental health professional needs to understand the nutrition goals that are set for populations but also needs to be able to translate them into terms of foods consumed to relate them to dietary advice at the level of the individual patient in the dental practice. Based on the scientific evidence summarized above, the following are recommendations for dietary advice dental practice setting.

It is generally recommended that sugars-rich foods should be limited to mealtimes and avoided in-between meals. Intakes of sugary foods should be limited to a maximum of four times a day. Providing sugars-free alternatives to sugars-rich products is a practical way to reducing free-sugars intake. Table 8 lists the non-sugars sweeteners permitted for food use.

It is important that free sugars are avoided near bedtime because during sleep salivary flow is curtailed making the teeth vulnerable to decay. Consuming free sugars close to bedtime leaves insufficient time for the oral pH to rise before the salivary flow is reduced. As a result, the teeth are exposed to acids for an extended period of time.

To minimize the risk of dental erosion the consumption of acidic drinks (still, carbonated, sugared and sugar-free) should be limiting to mealtimes only. Some manufacturers are now developing and marketing non-erosive soft drinks (Hughes et al, 1999); these may be effective if used to substitute standard varieties of soft drinks.

Table 10 Snacks that are safer for teeth

- ☐ Milk
- ☐ Cheese
- ☐ Peanuts
- ☐ Sugars-free chewing gum
- ☐ Fibrous foods (e.g. raw vegetables)
- ☐ Xylitol sweeteners, gum and mints
- ☐ Tea (unsweetened)
- ☐ Bread (sandwiches, toast, crumpets and pita bread)
- ☐ Pasta, rice and starchy staple foods
- ☐ Unsweetened or artificially sweetened yogurt
- ☐ Low-sugars breakfast cereals, (e.g. shredded wheat)
- ☐ Sugars-free confectionery
- ☐ Fresh fruit (whole and not juices)
- ☐ Water
- ☐ Sugars-free drinks

Source: Moynihan (2002)

It is important that oral health promotion must not be considered in isolation to general health. The dietary guidelines for the prevention of chronic diseases encourage increased consumption of fruits, vegetables and starchy staple foods and reduced consumption of fats (in particular saturated fats), free sugars and salt. Effective implementation of these dietary guidelines would be likely to result in a reduction in dental caries and dental erosion and offer protection against oral infections and oral cancer.

To safeguard dental health, foods that are beneficial for the teeth should also be encouraged; these include milk, hard cheese (especially lower fat varieties) and fibrous foods. Chewing sugars-free chewing gum after sugary snacks could also be useful as it is a good mechanical and gustatory stimulus to salivary flow. Some suggestions for snacks and drinks that are safer for teeth are presented in Table 10 (Moynihan, 2002).

It is essential that good dietary practices for dental health are instilled early in childhood. It is recommended that infants be weaned onto a cup as soon as possible – from the age of 6 months and no later than their first birthday. Free sugars should not be added to bottle feeds and sugary foods should only be given at mealtimes.

For older patients presenting for dental prosthesis, dietary advice to increase the consumption of fruits, vegetables and starchy staple foods high in NSP is recommended. Practical advice on ways to include these foods in the diet should consider the potential compromised chewing ability of the patient.

Recommendations for Areas to Include in the Training of Dental Hygienists

Dental hygienists should be equipped with the necessary knowledge and communication skills to enable them to provide advice on healthier eating for dental and general health to individuals or groups of people from a range of age groups, in the dental practice or community setting. Based on the scientific evidence, the knowledge base to achieve this should include the following subject areas:

- Knowledge of the main classes of nutrients, their sources, functions and health effects
- Knowledge of the current national guidelines for diet and health
- Knowledge of the effects of nutrients of the development of the teeth and the supporting tissues
- An understanding of the evidence relating diet to dental caries
- An understanding of the evidence relating diet to dental erosion
- An understanding of the effects of tooth loss on diet and health
- A knowledge of the main dietary goals for dental health.

In addition to the knowledge base, dental hygienists need to learn good communication skills so that they are able to support patients to instigate dietary change as opposed to simply providing information. To achieve this, the hygienist should be able to assess the patient's current dietary practices, make positive suggestions for food choices, be able to assist in patient-centered goal setting and be able to monitor the effectiveness of dietary advice provided. In order to assess patients' diets and make recommendations for change, dental hygienists will require some basic skills in dietary assessment.

REFERENCES

1. Alexy U, Sichert-Hellert W, Kersting M. Fifteen year time trends in energy and macronutrient intake in German children and adolescents: results of the DONALD study. Br J Nutr 2002;87:595-604.
2. Alfonsky D. Some observations on dental caries in central China. J Dent Res 1951;30:53-61.
3. Anaise JZ. Prevalence of dental caries among workers in the sweets industry in Israel. Commun Dent Oral Epidemiol 1978;8:142-145.
4. Astrup A. Healthy lifestyles in Europe: prevention of obesity and type II diabetes by diet and physical activity. Public Health Nutr 2001; 4(2B):449-516.
5. Averill HM, Averill JE. The effect of daily apple consumption on dental caries experience, oral hygiene status and upper respiratory infections. NY State Dent J 1968;34:403-409.
6. Bibby BG, Huang CT, Zero D, Mundorff SA, Little M. Protective effect of milk against in vitro caries. J Dent Res 1980;59:1565-1570.
7. Bowen WH, Amsbaugh SM, Monnell-Torens S, Brunelle S, Kuzmiak-Jones J, Cole MF. A method to assess cariogenic potential of foodstuffs. J Am Dent Ass 1980;100:677-681.
8. Brudevold F, Goulet D, Terani A, Attarzadeh F, van Houte J. Intraoral demineralization and maltose clearance from wheat starch. Caries Res 1985;19:136-144.

9. Burt B, Pai S. Sugar consumption and caries risk: a systematic review. J Dent Ed 2001;65:1017-1023.
10. Burt B, Szpunar SM. The Michigan Study: Relationship between sugars intake and dental caries over three years. Int Dent J 1994;44: 230-240.
11. 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;67:1422-1429.
12. Buttner. Zuckeraufnahme und Karies [sugar and caries]. Basel: Karger 1971.
13. Chyou P, Nomura AM, Stemmermann GN. Diet, alcohol, smoking and cancer or the upper aerodigestive tract: a prospective study among Hawaii Japanese men. Int J Cancer 1995;60:616-621.
14. Clancy KL, Bibby BG, Goldberg HJV, Ripa LW, Barenie J. Snack food intake of adolescents and caries development. J Dent Res 1977; 56:568-573.
15. Cleaton-Jones P, Richardson BD, Winter GB, Sinwell R, Rantsho HM, Jodaikin A. Dental caries and sucrose intake in five South African pre-school groups. Commun Dent Oral Epidemiol 1984;12: 381-385.
16. Cordeiro JGO, Matsudaira F, Ozaki F, Yanagida A, Kitamura C. Caries-protective effect of apple polyphenols on rampant caries of hamster. J Dent Res 2000;79:594.
17. Craig GC. The use of a calcium sucrose phosphates-calcium orthophosphate complex as a cariostatic agent. Br Dent J 1975;138: 25-28.
18. Department of Health. Dietary sugars and human disease. Report on Health and Social Subjects No 37; London: HMSO 1989.
19. Doll R, Peto R. Epidemiology of cancer. In: Wetherall DJ, Lednghan J GG, Warrell DA (eds). Oxford textbook of medicine. Oxford: Oxford University Press 1996;197-221.
20. Edwardsson S, Birkhed D, Mejáre B. Acid production from Lycasin, maltitol, sorbitol and xylitol by oral streptococci and lactobacilli. Acta Odont Scand 1977;35:257-263.
21. Enwonwu CO. Interface of malnutrition and periodontal diseases. Am J Clin Nutr 1995;61(suppl):430S-436S.
22. Enwonwu CO, Phillips RS, Falkler WA. Nutrition and oral infectious diseases: state of the science. Compend Cont Ed Dent 2002;23: 431-436.
23. Fejerskov O, Baelum V. Changes in prevalence and incidence of the major oral diseases. Zurich: Karger 1998.
24. Firestone AR, Imfeld T, Muhlemann HR. Effect of the length and number of intervals between meals on caries in rats. Caries Res 1984; 18:128-133.
25. Firestone AR, Schmid R, Muhlemann HR. Cariogenic effects of cooked wheat starch alone or with sucrose and frequency-controlled feeding in rats. Arch Oral Biol 1982;27:759-763.
26. Fisher FJ. A field study of dental caries, periodontal disease and enamel defects in Tristan da Cunha. Br Dent J 1968;125:447-453.
27. Fletcher E, Adamson A, Rugg-Gunn A. Twenty years of change in the dietary intake and BMI of Northumbrian adolescents. Proc Nutr Soc 2001;80:210A.
28. Frostell G. Effects of milk, fruit juices and sweetened beverages on the pH of dental plaques. Acta Odont Scand 1970;28:609-622.
29. Gaengler P, Pfister W, Sproessig M, Mirgorod M. The effects of carbohydrate reduced diet on development of gingivitis. Clin Prev Dent 1986;8:17-23.
30. Gedalia I, Ben-Mosheh S, Biton J, Kogan D. Dental caries protection with hard cheese consumption. Am J Dent 1994;7:331-332.
31. Gibney M. Consumption of sugars. Workshop on the evaluation of the nutritional and health aspects of sugars. Am J Clin Nutr 1995; 62:178S-194S.
32. Grenby TH. The effects of some carbohydrates on experimental dental caries in the rat. Arch Oral Biol 1963;8:27-30.
33. Grenby TH. The effects of starch and sugar diets on dental caries. Br Dent J 1970;128:575-578.
34. Grenby TH. The effect of glucose syrup on dental caries in the rat. Caries Res 1972;6.
35. Grenby TH, Mistry HM, Desai T. Potential dental effects of infants' fruit drinks studied in vitro. Br J Nutr 1990;64:273-283.
36. Grobler SR, Blignaut JB. The effect of a high consumption of apples or grapes on dental caries and periodontal disease in humans. Clin Prev Dent 1989;11:8-12.
37. Gustafsson BE, Quensel CE, Lanke LS, Lundquiat C, Grahnen H, Bonow BE, Krasse B. The Vipeholm dental caries study. The effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for 5 years. Acta Odont Scand 1954;11: 232-364.
38. Harris R. Biology of the children of Hopewood House, Bowral, Australia, 4. Observations on dental caries experience extending over 5 years (1957-61). J Dent Res 1963;42:1387-1399.
39. Hefti A, Schmid R. Effect on caries incidence in rats of increasing dietary sucrose levels. Caries Res 1979;13.
40. Hinds K, Gregory J. National Diet and Nutrition Survey: Children Aged 1.5 – 4.5 years. Volume 2: Report of the Dental Survey. London: HM Stationery Office 1995.
41. Holbrook WP, Arnadottir 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; 103:42-45.
42. Holbrook WP, Kristinsson MJ, Gunnarsdottir S, Briem B. Caries prevalence, Streptococcus mutans and sugar intake among 4-year-old urban children in Iceland. Commun Dent Oral Epidemiol 1989;17: 292-295.
43. Holloway PJ, Mellanby M, Stewart RJC. Fruit drinks and tooth erosion. Br Dent J 1958;104:305-309.
44. Holt RD. Foods and drinks at four daily time intervals in a group of young children. Br Dent J 1991;170:137-143.
45. Holt RD, Joels D, Winter GB. Caries in preschool children; the Camden study. Br Dent J 1982;153:107-109.
46. Hughes JA, West NX, Parker DM, Newcombe RG, Addy M. Development and evaluation of a low erosive blackcurrant juice drink. Final drink and concentrate, formulae comparison in situ and overview of the concept. J Dent 1999;27:345-350.
47. Hussein I, Pollard MA, Curzon MEJ. A comparison of the effects of some extrinsic and intrinsic sugars on dental plaque pH. Int J Paed Dent 1996;6:81-86.
48. Imfeld TN. Identification of low caries risk dietary components. Monographs in Oral Science 1983;11.
49. Imfeld TN, Schmid R, Lutz F, Guggenheim B. Cariogenicity of Milchschmitte (Ferrero-GmbH) and apple in programme-fed rats. Caries Res 1991;25:352-358.
50. Ismail AI, Burt BA, Eklund SA. The cariogenicity of soft drinks in the United States. J Am Dent Assoc 1984;109:241-245.
51. Isokangas P, Alanen P, Tiekso J, Mäkinen KK. Xylitol chewing gum in caries prevention: a field study in children. J Am Dent Assoc 1988;117:315-320.
52. 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;47:213-217.
53. Jarvinen VK, Rytomaa I, Heinonen OP. Risk factors in dental erosion. J Dent Res 1991;70:942-947.
54. Kandelman D, Gagnon G. A 24-month clinical study of the incidence and progression of dental caries in relation to consumption of chewing gum containing xylitol in school preventive programs. J Dent Res 1990;69:1771-1775.
55. Kleemola-Kujala E, Rasanen L. Relationship of oral hygiene and sugar consumption to risk of caries in children. Commun Dent Oral Epidemiol 1982;10:224-233.
56. Knowles EM. The effects of enemy occupation on the dental condition of children in the Channel Islands. Monthly Bull Min of Health (August) 1946;161-172.
57. Konig KP, Schmid P, Schmid R. An apparatus for frequency-controlled feeding of small rodents and its use in dental caries experiments. Arch Oral Biol 1968;13:13-26.
58. 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;101:427-441.
59. Kunzel W, Fischer T. Rise and fall of caries prevalence in German towns with different F concentrations in drinking water. Caries Res 1997;31:166-173.
60. Linkosalo E, Markkanen H. Dental erosions in relation to lacto-vegetarian diet. Scandinavian J Dent Res 1985;93:436-441.
61. Ludwig TG, Bibby BG. Acid production from different carbohydrate foods in plaque and saliva. J Dent Res 1957;3656-60.
62. Macfarlane GJ (1993) The Epidemiology of Oral Cancer. PhD, Bristol.

63. Mäkinen K K, Bennett C A, Hujoel P P, Isokangas P J, Isotupa K P, Pape H R jr. Xylitol chewing gums and caries rates, a 40-month cohort study. J Dent Res 1995;74:1904-1913.
64. Marques APF, Messer LB. Nutrient intake and dental caries in the primary dentition. Ped Dent 1992;14:314-321.
65. Marthaler T. Changes in the prevalence of dental caries: How much can be attributed to changes in diet? Caries Res 1990;24:3-15.
66. Marthaler TM. Epidemiological and clinical dental findings in relation to intake of carbohydrates. Caries Res 1967;1:222-238.
67. Martinsson T. Socio-economic investigation of school children with high and low caries frequency. Odont Rev 1972;23:93-114.
68. Masalin K, Murtamaa H, Meurman JH. Oral health of workers in the modern Finnish confectionery industry. Comm Dent Oral Epidemiol 1990;18:126-130.
69. Mikx FHM, van der Hoevel JS, Plasschaert AJM, Konig KG. Effect of Actinomyces viscosus on the establishment and symbiosis of Streptococcus mutans and Streptococcus sanguis on SPF rats on different sucrose diets. Caries Res 1975;9:1-20.
70. Miller CD. Erosion of molar teeth by acid beverages. J Nutr 1950;41: 63-71.
71. Millward A, Shaw L, Smith AJ, Rippin JW, Harrington E. The distribution and severity of tooth wear and the relationship between erosion and dietary constituents in a group of children. Int J Paed Dent 1994;4:152-157.
72. Miyazaki H, Morimoto M. Changes in caries prevalence in Japan. European J Oral Science 1996;104:452-458.
73. Moynihan PJ. Dietary Advice in Dental Practice. Br Dent J 2002;193: 563-568.
74. Moynihan PJ, Gould MEL, Huntley N, Thorman S. Effect of glucose polymers in water, milk and a milk substitute on plaque pH in vitro. Int J Paed Dent 1996a;6:19-24.
75. Moynihan PJ, Ferrier S, Jenkins GN. The cariostatic potential of cheese: cooked cheese-containing meals increase plaque calcium concentration. Br Dent J 1999;187:664-667.
76. Moynihan PJ, Snow S, Jepson NJA, Butler TJ. Intake of non-starch polysaccharide (dietary fiber) in edentulous and dentate persons: an observational study. Br Dent J 1994;177:243-247.
77. Moynihan PJ, Wright WG, Walton AG. A comparison of the relative acidogenic potential of infant milk and soya infant formula: a plaque pH study. Int J Paed Dent 1996b;6:177-181.
78. Nadanovsky P. Letter. Br Dent J 1994;177:280.
79. 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 Am Dent Assoc 1980;101:619-626.
80. Nizel AE, Harris RS. The effects of phosphates on experimental dental caries: A literature review. Br Dent J 1964;43:1123-1136.
81. Petersen PE. Dental health among workers at a Danish chocolate factory. Commun Dent Oral Epidemiol 1983;11;337-341.
82. Pindborg JJ. Etiology of developmental enamel defects not related to fluorosis. Int Dent J 1982;32:123-134.
83. Pitts NB, Boyles JJ, Nugent ZJ, Thomas N and Pine CM. The dental caries experience of 5-year old children in England and Wales. Surveys coordinated by the British Association for the Study of Community Dentistry in 2001/2002. Commun Dent Health 2003;20: 49-54.
84. Renaud S, Lanzmann-Petithory D. Coronary heart disease: dietary links and pathogenesis. Public Health Nutr 2001;4(2B):459-476.
85. Reynolds EC, Johnson IH. Effect of milk on caries incidence and bacterial composition of dental plaque in the rat. Arch Oral Biol 1981;26:445-451.
86. Rich AM, Radden BG. Squamous cell carcinoma of the oral mucosa: a review of 244 cases in Australia. J Oral Pathol 1984;13:459-471.
87. Roberts IF, Roberts GJ. Relation between medicines sweetened with sucrose and dental disease. Br Med J 1979;2:14-16.
88. 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 Paed Dent 2000;10: 47-55.
89. Rugg-Gunn AJ. Nutrition and Dental Health. Oxford: Oxford Medical Publications 1993.
90. Rugg-Gunn AJ, Al-Mohammadi SM, Butler TJ. Malnutrition and developmental defects of enamel in 2- to 6- year-old Saudi boys. Caries Res 1998;32:181-192.
91. Rugg-Gunn AJ, Edgar WM, Geddes DAM, Jenkins GN. The effect of different meal patterns upon plaque pH in human subjects. Br Dent J 1979;139:351-356.
92. 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 adolescent schoolchildren. Arch Oral Biol 1984;29:983-992.
93. Rugg-Gunn AJ, Roberts GJ, Wright WG. The effect of human milk on plaque in situ and enamel dissolution in vitro compared with bovine milk, lactose and sucrose. Caries Res 1985;19:327-334.
94. Russell AL, Littleton NW, Leatherwood EC, Syndow GE, Green JC. Dental surveys in relation to nutrition. Publ Health Rep 1960;75: 717-723.
95. Scheinin A, Mäkinen KK, Tammisalo E, Rekola M. Turku sugar studies XVIII. Incidence of dental caries in relation to 1 year consumption of xylitol chewing gum. Acta Odont Scand 1975;33(suppl 70): 307-316.
96. Scheinin A, Mäkinen KK, Ylitalo K. Turku sugar studies V. Final report on the effect of sucrose, fructose and xylitol diets on the caries incidence in man. Acta Odont Scand 1976;34:179-198.
97. Schulerud A. Dental caries and nutrition during wartime in Norway. Oslo: FaBrius and Sonners Trykkeri 1950.
98. Sheiham A. Sugars and dental caries. The Lancet 1983;1:282-284.
99. Sheiham A. Dietary effects on dental diseases. Public Health Nutr 2001;4:569-591.
100. Sidi AD, Ashley PF. Influence of frequent sugar intake on experimental gingivitis. J Periodontol 1984;55:419-423.
101. Silver DH. A longitudinal study of infant feeding practice, diet and caries related to social class in children aged 3 and 8 – 10 years. Br Dent J 1987;163:296-300.
102. Slack GL, Martin WJ. Apples and dental health. Br Dent J 1958;105: 366-371.
103. Sognnaes RF. Analysis of wartime reduction of dental caries in European children. Am J Dis Child 1948;75:792-821.
104. Sperling G, Lovelace F, Barnes LL, Smith CAH, Saxton JA, McKay. Effect of long-time feeding of whole milk diets to white rats. J Nutr 1955;55:399-414.
105. Sreebny LM. Sugar availability, sugar consumption and dental caries. Commun Dent Oral Epidemiol 1982;10:1-7.
106. Stabholz A, Raisten J, Markitziu A. Tooth enamel dissolution from erosion or etching and subsequent caries development. J Periodontol 1983;7:100-108.
107. Stecksen-Blicks C, Gustafsson L. Impact of oral hygiene and use of fluorides on caries increment in children during one year. Commun Dent Oral Epidemiol 1996;14:185-189.
108. 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.
109. Stephan RM. Effects of different types of foods on dental health in experimental animals. J Dent Res 1966;45:1551-1561.
110. Szpunar SM, Eklund SA, Burt BA. Sugar consumption and caries risk in schoolchildren with low caries experience. Commun Dent Oral Epidemiol 1995;23:142-146.
111. 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.
112. Takeuchi M. Epidemiological study on Japanese children before, during and after World War II. Int Dent J 1961;11:443-457.
113. Thompson ME, Dever JG, Pearce EIF. Intra-oral testing of flavoured sweetened milk. NZ Dent J 1984;1 80:44-46.
114. Walker A, Gregory J, Bradnock G, Nunn J, White D. National Diet and Nutrition Survey: young people aged 4 to 18 years. Volume 2: Report of the oral health survey. London: The Stationery Office 2000.
115. Weaver R. Fluorine and war-time diet. Br Dent J 1950;88:231-239.
116. Woodward M, Walker ARP. Sugar and dental caries: The evidence from 90 countries. Br Dent J 1994;176:297-302.
117. World Cancer Research Fund. Food Nutrition and the Prevention of Cancer: a global perspective. World Cancer Research Fund, American Institute for Cancer Research: Washington DC 1997.
118. World Health Organization. Diet in the prevention of chronic diseases. Technical Report Series 916. World Health Organization, Food and Agricultural Organization: Geneva 2003.
119. World Health Organization. Global Oral Health Data Bank. World Health Organization 2001.