

## 5.6 Recommendations for preventing dental diseases

### 5.6.1 *Background*

Oral health is related to diet in many ways, for example, through nutritional influences on cranio-facial development, oral cancer and oral infectious diseases. The purpose of this review, however, is to focus on the nutritional aspects of dental diseases. Dental diseases include dental caries, developmental defects of enamel, dental erosion and periodontal disease. Dental diseases are a costly burden to health care services, accounting for between 5% and 10% of total health care expenditures and exceeding the cost of treating cardiovascular disease, cancer and osteoporosis in industrialized countries (1). In low-income countries, the cost of traditional restorative treatment of dental disease would probably exceed the available resources for health care. Dental health promotion and preventive strategies are clearly more affordable and sustainable.

Although not life-threatening, dental diseases have a detrimental effect on quality of life in childhood through to old age, having an impact on self-esteem, eating ability, nutrition and health. In modern society, a significant role of teeth is to enhance appearance; facial appearance is very important in determining an individual's integration into society, and teeth also play an essential role in speech and communication. Oral diseases are associated with considerable pain, anxiety and impaired social functioning (2, 3). Dental decay may result in tooth loss, which reduces the ability to eat a nutritious diet, the enjoyment of food, the confidence to socialize and the quality of life (4–6).

### 5.6.2 *Trends*

The amount of dental decay is measured using the dmf/DMF index, a count of the number of teeth or surfaces in a person's mouth that are decayed, missing or filled as a result of caries in primary dentition/permanent dentition. An additional dental status indicator is the proportion of the population who are edentulous (have no natural teeth).

In most low-income countries, the prevalence rate of dental caries is relatively low and more than 90% of caries are untreated. Available data (7) show that the mean number of decayed, missing or filled permanent teeth (DMFT) at age 12 years in low-income countries is 1.9, 3.3 in middle-income countries and 2.1 in high-income countries (Table 12).

Data on the level of dental caries in the permanent dentition of 12-year-olds show two distinct trends. First, a fall in the prevalence of dental caries in developed countries, and second an increase in the prevalence of the disease in some developing countries that have increased their consumption of sugars and have not yet been introduced to the presence

of adequate amounts of fluoride. Despite the marked overall decline in dental caries over the past 30 years, the prevalence of dental caries remains unacceptably high in many developed countries. Even in countries with low average DMFT scores, a significant proportion of children have relatively high levels of dental caries. Moreover, there is some indication that the favourable trends in levels of dental caries in permanent teeth have come to a halt (8).

Table 12  
**Trends in levels of dental caries in 12-year-olds (mean DMFT per person aged 12 years)**

Country or area	Year	DMFT	Year	DMFT	Year	DMFT
<b>Industrialized countries</b>						
Australia	1956	9.3	1982	2.1	1998	0.8
Finland	1975	7.5	1982	4.0	1997	1.1
Japan	1975	5.9	1993	3.6	1999	2.4
Norway	1940	12.0	1979	4.5	1999	1.5
Romania	1985	5.0	1991	4.3	1996	3.8
Switzerland	1961–1963	9.6	1980	1.7	1996	0.8
United Kingdom	1983	3.1	1993	1.4	1996–1997	1.1
United States	1946	7.6	1980	2.6	1998	1.4
<b>Developing countries</b>						
Chile	1960	2.8	1978	6.6	1996	4.1
Democratic Republic of the Congo	1971	0.1	1982	0.3	1987	0.4–1.1
French Polynesia	1966	6.5	1986	3.2	1994	3.2
Islamic Republic of Iran	1974	2.4	1976	4.9	1995	2.0
Jordan	1962	0.2	1981	2.7	1995	3.3
Mexico	1975	5.3	1991	2.5–5.1	1997	2.5
Morocco	1970	2.6	1980	4.5	1999	2.5
Philippines	1967	1.4	1981	2.9	1998	4.6
Uganda	1966	0.4	1987	0.5	1993	0.4

DMFT, decayed, missing, filled permanent teeth.  
 Source: reference 7.

Many developing countries have low decayed, missing, filled primary teeth (dmft) values but a high prevalence of dental caries in the primary dentition. Data on 5-year-old children in Europe suggest that the trend towards reduced prevalence of dental decay has halted (9–11). In children aged 5–7 years, average dmft values of below 2.0 have been reported for Denmark, England, Finland, Italy, Netherlands and Norway (12). Higher dmft values were reported recently for Belarus (4.7) (13), Hungary (4.5) (14), Romania (4.3) (15) and the Russian Federation (4.7) (16).

Being free from caries at age 12 years does not imply being caries-free for life. The mean DMFT in countries of the European Union after 1988 varied between 13.4 and 20.8 at 35–44 years (17). The WHO guidelines on oral health state that at age 35–44 years a DMFT score of 14 or above is

considered high. In most developing countries, the level of caries in adults of this age group is lower, for example, 2.1 in China (18) and 5.7 in Niger (19). Few data are available on the prevalence and severity of root caries in older adults, but with the increasingly ageing population and greater retention of teeth, the problem of root caries is likely to become a significant public health concern in the future.

The number of edentulous persons has declined over the past 20–30 years in several industrialized countries (3). Despite overall gains however, there is still a large proportion of older adults who are edentulous or partially dentate and as the population continues to age tooth loss will affect a growing number of persons worldwide. Table 13 summarizes the available information on the prevalence of edentulousness in old-age populations throughout the world.

Dental erosion is a relatively new dental problem in many countries throughout the world, and is related to diet. There is anecdotal evidence that prevalence is increasing in industrialized countries, but there are no data over time to indicate patterns of this disease. There are insufficient data available to comment on worldwide trends; in some populations, however, it is thought that approximately 50% of children are affected (20).

### 5.6.3 ***Diet and dental disease***

Nutritional status affects the teeth pre-eruptively, although this influence is much less important than the post-eruptive local effect of diet on the teeth (21). Deficiencies of vitamins D and A and protein–energy malnutrition have been associated with enamel hypoplasia and salivary gland atrophy (which reduces the mouth’s ability to buffer plaque acids), which render the teeth more susceptible to decay. In developing countries, in the absence of dietary sugars, undernutrition is not associated with dental caries. Undernutrition coupled with a high intake of sugars may exacerbate the risk of caries.

There is some evidence to suggest that periodontal disease progresses more rapidly in undernourished populations (22); the important role of nutrition in maintaining an adequate host immune response may explain this observation. Apart from severe vitamin C deficiency, which may result in scurvy-related periodontitis, there is little evidence at present for an association between diet and periodontal disease. Current research is investigating the potential role of the antioxidant nutrients in periodontal disease. Poor oral hygiene is the most important risk factor in the development of periodontal disease (21). Undernutrition exacerbates the severity of oral infections (e.g. acute necrotizing ulcerative gingivitis) and may eventually lead to their evolution into life-threatening diseases such as noma, a dehumanizing oro-facial gangrene (23).

Table 13

**Prevalence of edentulousness in older people throughout the world**

Country or area	Prevalence of edentulousness (%)	Age group (years)
<b>African Region</b>		
Gambia	6	65 +
Madagascar	25	65-74
<b>Region of the Americas</b>		
Canada	58	65 +
United States	26	65-69
<b>South-East Asian Region</b>		
India	19	65-74
Indonesia	24	65 +
Sri Lanka	37	65-74
Thailand	16	65 +
<b>European Region</b>		
Albania	69	65 +
Austria	15	65-74
Bosnia and Herzegovina	78	65 +
Bulgaria	53	65 +
Denmark	27	65-74
Finland	41	65 +
Hungary	27	65-74
Iceland	15	65-74
Italy	19	65-74
Lithuania	14	65-74
Poland	25	65-74
Romania	26	65-74
Slovakia	44	65-74
Slovenia	16	65 +
United Kingdom	46	65 +
<b>Eastern Mediterranean Region</b>		
Egypt	7	65 +
Lebanon	20	64-75
Saudi Arabia	31-46	65 +
<b>Western Pacific Region</b>		
Cambodia	13	65-74
China	11	65-74
Malaysia	57	65 +
Singapore	21	65 +

Source: reference 7.

Dental caries occur because of demineralization of enamel and dentine by organic acids formed by bacteria in dental plaque through the anaerobic metabolism of sugars derived from the diet (24). Organic acids increase the solubility of calcium hydroxyapatite in the dental hard tissues and demineralization occurs. Saliva is super-saturated with calcium and phosphate at pH 7 which promotes remineralization. If the oral pH remains high enough for sufficient time then complete remineralization of

enamel may occur. If the acid challenge is too great, however, demineralization dominates and the enamel becomes more porous until finally a carious lesion forms (25). The development of caries requires the presence of sugars and bacteria, but is influenced by the susceptibility of the tooth, the bacterial profile, and the quantity and quality of the saliva.

#### *Dietary sugars and dental caries*

There is a wealth of evidence from many different types of investigation, including human studies, animal experiments and experimental studies in vivo and in vitro to show the role of dietary sugars in the etiology of dental caries (21). Collectively, data from these studies provide an overall picture of the cariogenic potential of carbohydrates. Sugars are undoubtedly the most important dietary factor in the development of dental caries. Here, the term “sugars” refers to all monosaccharides and disaccharides, while the term “sugar” refers only to sucrose. The term “free sugars” refers to all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, fruit juices and syrups. The term “fermentable carbohydrate” refers to free sugars, glucose polymers, oligosaccharides and highly refined starches; it excludes non-starch polysaccharides and raw starches.

Worldwide epidemiological studies have compared sugar consumption and levels of dental caries at the between-country level. Sreebny (26, 27) correlated the dental caries experience (DMFT) of 12-year-olds with data on sugar supplies of 47 countries and found a significant correlation (+0.7); 52% of the variation in the level of caries was explained by the per capita availability of sugar. In countries with a consumption level of sugar < 18 kg per person per year caries experience was consistently < DMFT 3. A later analysis by Woodward & Walker (28) did not find a similar association for developed countries. Sugar availability nevertheless accounted for 28% of the variation in levels of dental caries; 23 out of 26 countries with a per capita sugar availability < 50 g per day had a mean DMFT score for 12-year olds of < 3, whereas only half of the countries with sugar availability above this level had achieved a DMFT score that was < 3.

Miyazaki & Morimoto (29) reported a significant correlation ( $r = +0.91$ ) between sugar availability in Japan and DMFT at age 12 years between 1957 and 1987. Populations that had experienced a reduced sugar availability during the Second World War showed a reduction in dental caries which subsequently increased again when the restriction was lifted (30–32). Although the data pre-date the widespread use of fluoride dentifrice, Weaver (33) observed a reduction in dental caries between 1943 and 1949 in areas of northern England with both high and low concentrations of fluoride in drinking-water.

Isolated communities with a traditional way of life and a consistently low intake of sugars have very low levels of dental caries. As economic levels in such societies rise, the amount of sugar and other fermentable carbohydrates in the diet increases and this is often associated with a marked increase in dental caries. Examples of this trend have been reported among the Inuit in Alaska, USA (34), as well as in populations in Ethiopia (35), Ghana (36), Nigeria (37), Sudan (38), and on the Island of Tristan da Cunha, St Helena (39).

There is evidence to show that many groups of people with high exposure to sugars have levels of caries higher than the population average. Examples include children with chronic diseases requiring long-term sugar-containing medicines (40), and confectionery workers (41–44). Likewise, experience of dental caries has seldom been reported in groups of people who have a habitually low intake of sugars, for example, children of dentists (45, 46) and children in institutions where strict dietary regimens are inflicted (47, 48). A weakness of population studies of this type is that changes in intake of sugars often occur concurrently with changes in the intake of refined starches, making it impossible to attribute changes in dental caries solely to changes in the intake of sugars. An exception to this are the data from studies of children with hereditary fructose intolerance (HFI). Studies have shown that people with HFI have a low intake of sugars and a higher than average intake of starch, but have a low dental caries experience (49).

Human intervention studies are rare, and those that have been reported are now decades old and were conducted in the pre-fluoride era before the strong link between sugars intake and dental caries levels was established. It would not be possible to repeat such studies today because of ethical constraints. The Vipeholm study, conducted in an adult mental institution in Sweden between 1945 and 1953 (50), investigated the effects of consuming sugary foods of varying stickiness and at different times throughout the day on the development of caries. It was found that sugar, even when consumed in large amounts, had little effect on caries increment if it was ingested up to a maximum of four times a day at mealtimes only. Increased frequency of consumption of sugar between meals was, however, associated with a marked increase in dental caries. It was also found that the increase in dental caries activity disappears on withdrawal of sugar-rich foods. Despite the complicated nature of the study the conclusions are valid, although they apply to the pre-fluoride era. The Turku study was a controlled dietary intervention study carried out on adults in Finland in the 1970s which showed that almost total substitution of sucrose in the diet with xylitol (a non-cariogenic sweetener) resulted in an 85% reduction in dental caries over a 2-year period (51).

Numerous cross-sectional epidemiological studies have compared sugars intake with dental caries levels in many countries of the world. Those conducted before the early 1990s have been summarized by Rugg-Gunn (21). Nine out of 21 studies that compared amount of sugars consumed with caries increment found significant associations, while the other 12 did not. Moreover, 23 out of 37 studies that investigated the association between frequency of sugars consumption and caries levels found significant relationships, while 14 failed to find any such associations.

A cross-sectional study in the United States of 2514 people aged 9–29 years conducted between 1968 and 1970 found that the dental caries experience of adolescents eating the highest amounts of sugars (upper 15% of the sample) was twice that of those eating the lowest amounts (lower 15% of the sample) (52). Granath et al. (53) showed that intake of sugars was the most important factor associated with caries in the primary dentition of preschool children in Sweden. When the effects of oral hygiene and fluoride were kept constant, the children with a low intake of sugars between meals had up to 86% less caries than those with high intakes of sugars. Other studies have found fluoride exposure and oral hygiene to be more strongly associated with caries than sugars consumption (54, 55). A recent study in the United Kingdom of a representative sample of children aged 4–18 years showed no significant relationship between caries experience and level of intake of free sugars; in the age group 15–18 years, however, the upper band of free sugars consumers were more likely to have decay than the lower band (70% compared with 52%) (20).

Many other cross-sectional studies have shown a relationship between sugars consumption and levels of caries in the primary and/or permanent dentitions in countries or areas throughout the world, including China (56), Denmark (57), Madagascar (58, 59), Saudi Arabia (60), Sweden (61, 62), Thailand (63) and the United Kingdom (64).

When investigating the association between diet and the development of dental caries it is more appropriate to use a longitudinal study design in which sugars consumption habits over time are related to changes in dental caries experience. Such studies have shown a significant relationship between caries development and sugars intake (65–67). In a comprehensive study of over 400 children in England aged 11–12 years, a small but significant relationship was found between intake of total sugars and caries increment over 2 years ( $r = + 0.2$ ) (67). The Michigan Study in the United States investigated the relationship between sugars intake and dental caries increment over 3 years in children initially aged 10–15 years (66). A weak relationship was found between the amount of dietary sugars consumed and dental caries experience.

In a review of longitudinal studies, Marthaler (68) analysed the relationship between dietary sugars and caries activity in countries where the availability of sugars is high and the use of fluoride is extensive. He concluded that in modern societies that make use of prevention, the relationship between sugars consumption and dental caries was still evident (68). He also concluded that many older studies had failed to show a relationship between sugars intake and development of dental caries because they were of poor methodological design, used unsuitable methods of dietary analysis or were of insufficient power (68). Correlations between individuals' sugars consumption and dental caries increments may be weak if the range of sugars intake in the study population is small. That is to say, that if all people within a population are exposed to the disease risk factor, the relationship between the risk factor and the disease will not be apparent (69).

*Frequency and amount of sugars consumption.* Several studies, including the above-mentioned Vipeholm study in Sweden, have indicated that caries experience increases markedly when the frequency of sugars intake exceeds four times a day (50, 70–72). 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 frequency of sugars intake in the development of dental caries (73, 74). Some human studies have also shown that the frequency of sugars intake is an important etiological factor for caries development (75). Many studies have related the frequency of intake of sugars or sugars-rich food to caries development but have not simultaneously investigated the relationship between amount of sugars consumed and dental caries, and therefore no conclusion regarding the relative importance of these two variables can be drawn from these studies (76–78).

Animal studies have also shown a relationship between amount of sugars consumed and the development of dental caries (79–82). Several longitudinal studies in humans have indicated that the amount of sugars consumed is more important than the frequency (66, 67, 83, 84), while Jamel et al. (85) found that both the frequency and the amount of sugars intake are important.

The strong correlation between both the amount and frequency of sugars consumption has been demonstrated by several investigators in different countries (67, 86–88). It is therefore highly likely that, in terms of caries development, both variables are potentially important.

*Relative cariogenicity of different sugars and food consistency.* The relative acidogenicity of different monosaccharides and disaccharides has been investigated in plaque pH studies, which have shown that lactose is less acidogenic than other sugars (89). Animal studies have provided no clear

evidence that, with the exception of lactose, the cariogenicity of monosaccharides and disaccharides differs. The above-mentioned study in Turku, Finland, found no difference in caries development between subjects on diets sweetened with sucrose compared with those whose diet had been sweetened with fructose (51). Invert sugar (50% fructose + 50% glucose) is less cariogenic than sucrose (90).

The adhesiveness or stickiness of a food is not necessarily related to either oral retention time or cariogenic potential. For example, consumption of sugars-containing drinks (i.e. non-sticky) is associated with increased risk of dental caries (85, 88)

*Potential impact of sugars reduction on other dietary components.* It is important to consider the potential impact of a reduction in free sugars on other components of the diet. Simple, cross-sectional analysis of dietary data from populations has shown an inverse relationship between the intake of free sugars and the intake of fat (91), suggesting that reducing free sugars might lead to an increase in fat intake. There is, however, a growing body of evidence from studies over time that shows that changes in intake of fat and free sugars are not inversely related, and that reductions in intake of fat are offset by increases in intakes of starch rather than free sugars (92, 93). Cole-Hamilton et al. (94) found that the intake of both fat and added sugars simultaneously decreased as fibre intake increased. Overall dietary goals that promote increased intake of wholegrain staple foods, fruits and vegetables and a reduced consumption of free sugars are thus unlikely to lead to an increased consumption of fat.

*Influence of fluoride.* Fluoride undoubtedly protects against dental caries (95). The inverse relationship between fluoride in drinking-water and dental caries, for instance, is well established. Fluoride reduces caries in children by between 20% and 40%, but does not eliminate dental caries altogether.

Over 800 controlled trials of the effect of fluoride administration on dental caries have been conducted; collectively these studies demonstrate that fluoride is the most effective preventive agent against caries (95). Several studies have that indicated that a relationship between sugars intake and caries still exists in the presence of adequate fluoride exposure (33, 71, 96, 97). In two major longitudinal studies in children, the observed relationships between sugars intake and development of dental caries remained even after controlling for use of fluoride and oral hygiene practices (66, 67). As mentioned earlier, following a review of available longitudinal studies, Marthaler (68) concluded that, even when preventive measures such as use of fluoride are employed, a relationship between sugars intake and caries still exists. He also stated that in industrialized countries where there is adequate exposure to fluoride, no

further reduction in the prevalence and severity of dental caries will be achieved unless the intake of sugars is reduced.

A recent systematic review that investigated the importance of sugars intake in caries etiology in populations exposed to fluoride concluded that where there is adequate exposure to fluoride, sugars consumption is a moderate risk factor for caries in most people; moreover sugars consumption is likely to be a more powerful indicator for risk of caries in persons who do not have regular exposure to fluoride. Thus, restricting sugars consumption still has a role to play in the prevention of caries in situations where there is widespread use of fluoride but this role is not as strong as it is without exposure to fluoride (98). Despite the indisputable preventive role of fluoride, there is no strong evidence of a clear relationship between oral cleanliness and levels of dental caries (99–100).

Excess ingestion of fluoride during enamel formation can lead to dental fluorosis. This condition is observed particularly in countries that have high levels of fluoride in water supplies (95).

#### *Starches and dental caries*

Epidemiological studies have shown that starch is of low risk to dental caries. People who consume high-starch/low-sugars diets generally have low levels of caries, whereas people who consume low-starch/high-sugars diets have high levels of caries (39, 48, 49, 51, 67, 101, 102). In Norway and Japan the intake of starch increased during the Second World War, yet the occurrence of caries was reduced.

The heterogeneous nature of starch (i.e. degree of refinement, botanical origin, raw or cooked) is of particular relevance when assessing its potential cariogenicity. Several types of experiment have shown that raw starch is of low cariogenicity (103–105). Cooked starch is about one-third to one-half as cariogenic as sucrose (106, 107). Mixtures of starch and sucrose are, however, potentially more cariogenic than starch alone (108). Plaque pH studies, using an indwelling oral electrode, have shown starch-containing foods reduce plaque pH to 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 of the effect of foods on stimulation of salivary flow.

Glucose polymers and pre-biotics are increasingly being added to foods in industrialized countries. Evidence on the cariogenicity of these carbohydrates is sparse and comes from animal studies, plaque pH studies and studies in vitro which suggest that maltodextrins and glucose syrups are cariogenic (109–111). Plaque pH studies and experiments in vitro suggest that isomalto-oligosaccharides and gluco-oligosaccharides may be less

acidogenic than sucrose (112–114). There is, however, evidence that fructo-oligosaccharides are as acidogenic as sucrose (115, 116).

#### *Fruit and dental caries*

As habitually consumed, there is little evidence to show that fruit is an important factor in the development of dental caries (67, 117–119). A number of plaque pH studies have found fruit to be acidogenic, although less so than sucrose (120–122). Animal studies have shown that when fruit is consumed in very high frequencies (e.g. 17 times a day) it may induce caries (123, 124), but less so than sucrose. In the only epidemiological study in which an association between fruit consumption and DMFT was found (125), fruit intakes were very high (e.g. 8 apples or 3 bunches of grapes per day) and the higher DMFT in fruit farm workers compared with grain farm workers arose solely from differences in the numbers of missing teeth.

#### *Dietary factors which protect against dental caries*

Some dietary components protect against dental caries. The cariostatic nature of cheese has been demonstrated in several experimental studies (126, 127), and in human observational studies (67) and intervention studies (128). Cow's milk contains calcium, phosphorus and casein, all of which are thought to inhibit caries. Several studies have shown that the fall in plaque pH following milk consumption is negligible (129, 130). The cariostatic nature of milk has been demonstrated in animal studies (131, 132). Rugg-Gunn et al. (67) found an inverse relationship between the consumption of milk and caries increment in a study of adolescents in England. Wholegrain foods have protective properties; they require more mastication thereby stimulating increased saliva flow. Other foods that are good gustatory and/or mechanical stimulants to salivary flow include peanuts, hard cheeses and chewing gum. Both organic and inorganic phosphates (found in unrefined plant foods) have been found to be cariostatic in animal studies, but studies in humans have produced inconclusive results (133, 134). Both animal studies and experimental investigations in humans have shown that black tea extract increases plaque fluoride concentration and reduces the cariogenicity of a sugars-rich diet (135, 136).

#### *Breastfeeding and dental caries*

In line with the positive health effects of breastfeeding, epidemiological studies have associated breastfeeding with low levels of dental caries (137, 138). A few specific case studies have linked prolonged ad libitum and nocturnal breastfeeding to early childhood caries. Breastfeeding has the advantage that it does not necessitate the use of a feeder bottle, which has been associated with early childhood caries. A breastfed infant will

also receive milk of a controlled composition to which additional free sugars have not been added. There are no benefits to dental health of feeding using a formula feed.

#### *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. Extrinsic dietary acids include citric acid, phosphoric acid, ascorbic acid, malic acid, tartaric acid and carbonic acid found, for example, in fruits and fruit juices, soft drinks and vinegar. Erosion in severe cases leads to total tooth destruction (139). Human observational studies have shown an association between dental erosion and the consumption of a number of acidic foods and drinks, including frequent consumption of fruit juice, soft drinks (including sports drinks), pickles (containing vinegar), citrus fruits and berries (140–144). Age-related increases in dental erosion have been shown to be greater in those with the highest intake of soft drinks (20). Experimental clinical studies have shown that consumption of, or rinsing with, acidic beverages significantly lowers the pH of the oral fluids (121). Enamel is softened within one hour of exposure to cola but this may be reversed by exposure to milk or cheese (145, 146). Animal studies have shown that fruit and soft drinks cause erosion (124, 147), although fruit juices are significantly more destructive than whole fruits (148, 149).

#### 5.6.4 **Strength of evidence**

The strength of the evidence linking dietary sugars to the risk of dental caries is in the multiplicity of the studies rather than the power of any individual study. Strong evidence is provided by the intervention studies (50, 51) but the weakness of these studies is that they were conducted in the pre-fluoride era. More recent studies also show an association between sugars intake and dental caries albeit not as strong as in the pre-fluoride era. However, in many developing countries people are not yet exposed to the benefits of fluoride.

Cross-sectional studies should be interpreted with caution because dental caries develop 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. Longitudinal studies (66, 67) that have monitored a change in caries experience and related this to dietary factors provide stronger evidence. Such studies have been conducted on populations with an overall high sugars intake but a low interindividual variation; this may account for the weak associations that have been reported.

The studies that overcome the problem of low variation in consumption of sugars are studies that have monitored dental caries following a marked

change in diet, for example, those conducted on populations during the Second World War and studies of populations before and after the introduction of sugars into the diet. Such studies have shown clearly that changes in dental caries mirror changes in economic growth and increased consumption of free sugars. Sometimes changes in sugars consumption were accompanied by an increase in other refined carbohydrates. There are, however, examples where sugars consumption decreased and starch consumption increased yet levels of dental caries declined.

Strong evidence of the relationship between sugar availability and dental caries levels comes from worldwide ecological studies (26, 28). The limitations of these studies are that they use data on sugar availability and not actual intake, they do not measure frequency of sugars intake, and they assume that level of intake is equal throughout the population. Also, the values are for sucrose, yet many countries obtain a considerable amount of their total sugars from other sugars. These studies have only considered DMFT of 12-year-olds, not always from a representative sample of the population.

Caution needs to be applied when extrapolating the results of animal studies to humans because of differences in tooth morphology, plaque bacterial ecology, salivary flow and composition, and the form in which the diet is provided (usually powdered form in animal experiments). Nonetheless, animal studies have enabled the effect on caries of defined types, frequencies and amounts of carbohydrates to be studied.

Plaque pH studies measure plaque acid production, but the acidogenicity of a foodstuff cannot be taken as a direct measurement of its cariogenic potential. Plaque pH studies take no account of protective factors in foods, salivary flow and the effects of other components of the diet. Many of the plaque pH studies that show falls in pH below the critical value of 5.5 with fruits and cooked starchy foods have been conducted using the indwelling electrode technique. This electrode is recognized as being hypersensitive and non-discriminating, tending to give an “all or nothing” response to all carbohydrates (150).

Research has consistently shown that when annual sugar consumption exceeds 15 kg per person per year (or 40 g per person per day) dental caries increase with increasing sugar intake. When sugar consumption is below 10 kg per person per year (around 27 g per person per day), levels of dental caries are very low (26, 28, 29, 51, 151–158). Exposure to fluoride (i.e. where the proportion of fluoride in drinking-water is 0.7–1.0 ppm, or where over 90% of toothpastes available contain fluoride) increases the safe level of sugars consumption.

Tables 14–17 summarize the evidence relating to diet, nutrition and dental diseases.

Table 14

**Summary of strength of evidence linking diet to dental caries**

Evidence	Decreased risk	No relationship	Increased risk
<b>Convincing</b>	Fluoride exposure (local and systematic)	Starch intake (cooked and raw starch foods, such as rice, potatoes and bread; excludes cakes, biscuits and snacks with added sugars)	Amount of free sugars Frequency of free sugars
<b>Probable</b>	Hard cheese Sugars-free chewing gum	Whole fresh fruit	
<b>Possible</b>	Xylitol Milk Dietary fibre		Undernutrition
<b>Insufficient</b>	Whole fresh fruit		Dried fruits

Table 15

**Summary of strength of evidence linking diet to dental erosion**

Evidence	Decreased risk	No relationship	Increased risk
<b>Convincing</b>			
<b>Probable</b>			Soft drinks and fruit juices
<b>Possible</b>	Hard cheese Fluoride		
<b>Insufficient</b>			Whole fresh fruit

Table 16

**Summary of strength of evidence linking diet to enamel developmental defects**

Evidence	Decreased risk	No relationship	Increased risk
<b>Convincing</b>	Vitamin D		Excess fluoride
<b>Probable</b>			Hypocalcaemia

Table 17

**Summary of strength of evidence linking diet to periodontal disease**

Evidence	Decreased risk	No relationship	Increased risk
<b>Convincing</b>	Good oral hygiene		Deficiency of vitamin C
<b>Probable</b>			
<b>Possible</b>			Undernutrition
<b>Insufficient</b>	Antioxidant nutrients	Vitamin E supplementation	Sucrose

### 5.6.5 *Disease-specific recommendations*

It is important to set a recommended maximum level for the consumption of free sugars; a low free sugars consumption by a population will translate into a low level of dental caries. Population goals enable the oral health risks of populations to be assessed and health promotion goals monitored.

The best available evidence indicates that the level of dental caries is low in countries where the consumption of free sugars is below 15–20 kg per person per year. This is equivalent to a daily intake of 40–55 g per person and the values equate to 6–10% of energy intake. It is of particular importance that countries which currently have low consumption of free sugars (< 15–20 kg per person per year) do not increase consumption levels. For countries with high consumption levels it is recommended that national health authorities and decision-makers formulate country-specific and community-specific goals for reduction in the amount of free sugars, aiming towards the recommended maximum of no more than 10% of energy intake.

In addition to population targets given in terms of the amount of free sugars, targets for the frequency of free sugars consumption are also important. The frequency of consumption of foods and/or drinks containing free sugars should be limited to a maximum of four times per day.

Many countries that are currently undergoing nutrition transition do not have adequate exposure to fluoride. There should be promotion of adequate fluoride exposure via appropriate vehicles, for example, affordable toothpaste, water, salt and milk. It is the responsibility of national health authorities to ensure implementation of feasible fluoride programmes for their country. Research into the outcome of alternative community fluoride programmes should be encouraged.

In order to minimize the occurrence of dental erosion, the amount and frequency of intake of soft drinks and juices should be limited. Elimination of undernutrition prevents enamel hypoplasia and the other potential effects of undernutrition on oral health (e.g. salivary gland atrophy, periodontal disease, oral infectious diseases).

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