

The role of diet and nutrition in the etiology and prevention of oral diseases

Paula J. Moynihan¹

Abstract Diet plays an important role in preventing oral diseases including dental caries, dental erosion, developmental defects, oral mucosal diseases and, to a lesser extent, periodontal disease. This paper is intended to provide an overview of the evidence for an association between diet, nutrition and oral diseases and to clarify areas of uncertainty. Undernutrition increases the severity of oral mucosal and periodontal diseases and is a contributing factor to life-threatening noma. Undernutrition is associated with developmental defects of the enamel which increase susceptibility to dental caries. Dental erosion is perceived to be increasing. Evidence suggests that soft drinks, a major source of acids in the diet in developed countries, are a significant causative factor. Convincing evidence from experimental, animal, human observational and human intervention studies shows that sugars are the main dietary factor associated with dental caries. Despite the indisputable role of fluoride in the prevention of caries, it has not eliminated dental caries and many communities are not exposed to optimal quantities of fluoride. Controlling the intake of sugars therefore remains important for caries prevention. Research has consistently shown that when the intake of free sugars is < 15 kg/person/year, the level of dental caries is low. Despite experimental and animal studies suggesting that some starch-containing foods and fruits are cariogenic, this is not supported by epidemiological data, which show that high intakes of starchy staple foods, fruits and vegetables are associated with low levels of dental caries. Following global recommendations that encourage a diet high in starchy staple foods, fruit and vegetables and low in free sugars and fat will protect both oral and general health.

Keywords Oral health; Nutrition; Malnutrition; Diet; Diet, Cariogenic; Dental caries/etiology/prevention and control; Tooth erosion/etiology/prevention and control; Dietary sucrose/adverse effects; Dietary carbohydrates; Fluorides/therapeutic use; Mouth diseases/etiology/prevention and control (*source: MeSH, NLM*).

Mots clés Hygiène buccale; Nutrition; Malnutrition; Régime alimentaire; Régime cariogène; Carie dentaire/étiologie/prévention et contrôle; Erosion dentaire/étiologie/prévention et contrôle; Sucrose alimentaire/effets indésirables; Glucide alimentaire Fluorures/usage thérapeutique; Glucide alimentaire; Bouche, Maladie/étiologie/prévention et contrôle (*source: MeSH, INSERM*).

Palabras clave Salud bucal; Nutrición; Desnutrición; Dieta; Dieta cariogénica; Caries dental/etiología/prevencción y control; Erosión dentaria/etiología/prevencción y control; Sacarosa en la dieta; Carbohidratos en la dieta/efectos adversos; Fluoruros/uso terapéutico; Enfermedades de la boca/etiología/prevencción y control (*fuelle: DeCS, BIREME*).

الكلمات المفتاحية: صحة الفم؛ التغذية؛ سوء التغذية؛ القوت؛ القوت المسيب للتسوس؛ تسوس الأسنان، سبببات تسوس الأسنان، الوقاية من تسوس الأسنان ومكافحته؛ تآكل الأسنان، سبببات تآكل الأسنان، الوقاية من تسوس الأسنان ومكافحته؛ الآثار الضائرة للسكرورز الغذائي، الكربوهيدرات الغذائية؛ الاستخدام العلاجي للفلوريدات؛ سبببات الأمراض الغذائية، الوقاية من الأمراض الغذائية، الوقاية من الأمراض الغذائية ومكافحتها (المصدر: رؤوس الموضوعات الطبية - المكتب الإقليمي لشرق الأوسط).

Bulletin of the World Health Organization 2005;83:694-699.

Voir page 697 le résumé en français. En la página 698 figura un resumen en español.

يمكن الاطلاع على الملخص بالعربية في صفحة 698.

Introduction

Oral health is related to diet in many ways; for example, nutrition influences craniofacial development and oral mucosal and dental diseases including dental caries, enamel defects and periodontal disease. Dental diseases impair quality of life and have a negative impact on self-esteem, eating ability and health, causing pain, anxiety and impaired social functioning (1, 2). Tooth loss reduces the ability to eat a nutritious diet, the enjoyment of food and confidence to socialize (3, 4). This article is intended to provide an overview of the relationships between diet, nutrition and oral diseases.

Influence of nutrition on oral diseases

Periodontal disease

Periodontal disease (gum disease) progresses more rapidly in undernourished populations (5); the role of nutrition in maintaining an adequate immune response may explain this observation. Periodontal disease is associated with an increased production of reactive oxygen species which, if not buffered sufficiently, cause damage to the host cells and tissues (6). Antioxidant nutrients, for example, ascorbic acid (vitamin C), beta-carotene and alpha-tocopherol (vitamin E) are important buffers of reactive oxygen species and are found in many fruits,

¹ School of Dental Sciences, University of Newcastle upon Tyne, Framlington Place, Newcastle upon Tyne, NE2 4BW, England (email: p.j.moynihan@ncl.ac.uk).

Ref. No. 04-020347

(Submitted: 1 March 2005 – Final revised version received: 4 July 2005 – Accepted: 6 July 2005)

Paula J. Moynihan

vegetables, grains and seeds. Current research is investigating the potential protective role of antioxidant nutrients in periodontal disease. However, apart from severe vitamin C deficiency, which may result in scurvy-related periodontitis, there is as yet no strong evidence for an association between diet and periodontal disease.

Diseases of the oral mucosa

The first signs of deficiency of some micronutrients, for example the B-vitamins, are seen in the mouth and include glossitis, cheilitis and angular stomatitis (6). Undernutrition exacerbates the severity of oral infections and is a contributing factor to life-threatening diseases such as noma, a dehumanizing oro-facial gangrene (7).

Oral cancer is the eighth most common cancer in the world; its prevalence is high in developing countries and is increasing in some developed countries, such as Denmark, Germany, Scotland as well as in eastern Europe (8). Diet is a preventable risk factor for oral cancer. Trials of individual nutrients including iron, selenium and vitamins E, A and beta-carotene have produced equivocal results, but several case-control studies have shown a protective role of vitamin C (6). The report *Diet nutrition and the prevention of chronic diseases* (9) stated that there was convincing evidence that scalding hot foods and drinks increased the risk for oral cancer and an association with consumption of char-grilled foods has been found. Although wholegrain foods may be protective, the most conclusive evidence exists for a protective role of fruits and vegetables. A number of epidemiological studies have shown that the risk of oral cancer decreases with increasing fruit and vegetable intake (10). In a large cohort study in the USA, the risk of cancer was decreased by 40–80% in people with a high intake of fruits and vegetables compared with those with a low intake (11). The evidence indicates a stronger effect of fruits, in particular citrus fruits (12). However, prospective dietary intervention trials are lacking, probably due to the methodological complexities of undertaking such studies (10).

Interrelationship between nutritional status and HIV/AIDS

There is a strong relationship between immune status and the oral symptoms of human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS) (13). Poor nutritional status may compound the impaired immune status associated with HIV, possibly contributing to more rapid development of the oral symptoms including ulceration, candidiasis, drug-induced xerostomia and neoplasms (13). The oral manifestations of HIV exacerbate poor nutritional intake as a result of oral pain, dry mouth, dysphagia and, in the case of neoplasms, obstruction. Nutritional intervention together with oral care is essential to prevent the patient becoming severely nutritionally compromised.

Developmental defects of the enamel

Nutritional status affects the teeth pre-eruptively, although this influence is much less important than the post-eruptive local effect of diet (14). Deficiencies of vitamins D and A and protein-energy malnutrition are associated with enamel hypoplasia and salivary gland atrophy, both of which increase susceptibility to dental caries. Excessive fluoride ingestion while enamel is forming (up to the age of 6 years for permanent dentition) may cause dental fluorosis.

Dental erosion

Dental erosion is the irreversible loss of dental hard tissue (enamel and dentine) that is chemically etched away by acids in a process not involving bacteria. Dietary acids include citric, phosphoric, ascorbic, malic, tartaric, oxalic and carbonic acids. These acids are found, for example, in fruits and fruit juices, soft drinks and vinegar. Observational studies in humans have shown an association between dental erosion and the consumption of a number of acidic foods and drinks, including consumption of fruit juice, soft drinks, vinegar, citrus fruits and berries (15). Age-related increases in dental erosion have been shown to be greater in those with the highest intake of soft drinks (16). Animal studies have shown that fruits and soft drinks cause erosion, although fruit juices were 3–10 times more destructive than whole fruit (17). WHO (9) concluded that consumption of soft drinks was a probable cause of dental erosion and that citrus fruits were a possible cause, however, there was insufficient evidence to incriminate other fruits.

Dental caries

Despite the marked decline in dental caries in developed countries over the past 30 years, the prevalence remains high and favourable trends are halting (18). In developing countries, dental caries has increased where there has been increased exposure to dietary sugars. Dental caries occurs because of demineralization of enamel and dentine by organic acids formed by bacteria in dental plaque through the anaerobic metabolism of dietary sugars (18). A wealth of evidence from many different types of investigation, including human studies, animal experiments and experimental studies has consistently shown that sugars are the most important factor in caries development.

A study conducted on worldwide epidemiological data in the 1980s found the availability of sugar to account for 52%, and a similar study in the 1990s, for 28% of the variation in levels of dental caries (19, 20). In countries with a level of sugar consumption of less than 18 kg/person/year, caries experience is consistently low (19, 20). The reduced availability of sugar during the Second World War was mirrored by a reduction in dental caries, which subsequently increased when the restriction was lifted (21). Isolated communities with a traditional diet low in sugars have very low levels of dental caries (even when the traditional diet is high in starch) (22). On adopting a more “westernized” diet, i.e. a diet high in sugars, such populations have experienced a marked increase in dental caries. Examples of this trend have been reported among the Inuit in Alaska (23), in Ethiopia (24), Ghana (25), Nigeria (26), Sudan (27) and Tristan da Cunha (22).

There is evidence to show that groups of people with a habitually high intake of sugars also have higher levels of caries, for example, children requiring long-term administration of sugar-containing medicines (28) and confectionery workers (29). Likewise, a low level of dental caries is seen in those who have a habitually low intake of sugars, for example, children on strict dietary regimens (30) and children with hereditary fructose intolerance (31). The latter group has a higher than average intake of starch, which suggests an insignificant role of starch in caries development.

Few intervention studies have investigated the effect of sugars on caries. The Vipeholm study, conducted in an adult

mental institution in Sweden between 1945 and 1953 (32), investigated the effects of consuming sugary foods of different “stickiness” and at various frequencies on the development of caries. It was concluded that sugar had little effect on caries development if ingested with meals no more than four times a day. 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 progression of dental caries halted on withdrawal of sugar from the diet.

The Turku study was a controlled dietary intervention in Finnish adults 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 (33).

Numerous cross-sectional epidemiological studies of the association between intake of sugars with dental caries have produced equivocal results (14, 18). However, cross-sectional studies may not give a true reflection of the role of diet in the development of caries as it is the diet several years earlier that is responsible for current levels of caries. A longitudinal study design that monitors change in levels of caries and relates this to diet is more appropriate. Two comprehensive studies, one in the United Kingdom and one in the USA, have shown small but highly significant correlations between intake of sugars and caries development, with strongly significant differences in caries levels between those with the highest and lowest intakes of sugars (34, 35). Both these studies were conducted in populations with an overall high intake of sugars, but a low variation between individuals; this may account for the weak associations reported. Many earlier studies failed to show a relationship between intake of sugars and development of dental caries because their methodological design was poor, they used unsuitable methods of dietary analysis and lacked sufficient statistical power (36).

Frequency, amount and type of sugar

The importance of frequency of consumption of sugars as opposed to the total amount of sugars consumed is difficult to evaluate, as the two variables are hard to assess separately. However, data from both animal studies (37, 38) and studies in humans (32, 34, 35) indicate that both factors are associated with levels of caries.

There is no clear evidence that, with the exception of lactose (which is less cariogenic (39)), the cariogenicity of different sugars varies: for example, the above-mentioned Turku study (33) found no significant difference in caries development between those consuming sucrose and those consuming fructose. In view of this finding, the term “free sugars” is used to refer to all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus those naturally present in honey, fruit juices and syrups (9). The term free sugars excludes sugars naturally present in whole fruits, vegetables and milk, as evidence suggests that these pose little or no threat to dental or general health (see below) (9, 14, 18). Glucose polymers and non-digestible oligosaccharides are increasingly being used in foods. Limited evidence suggests that glucose polymers are potentially cariogenic, isomaltoligosaccharides and glucooligosaccharides may be less acidogenic than sucrose, but fructooligosaccharides may be as acidogenic as sucrose (39).

Influence of fluoride

Fluoride undoubtedly protects against dental caries (40), reducing caries in children by up to 50%, but it does not eliminate it or remove the cause — sugars. In addition, the people of many parts of the world are not exposed to fluoride. A relationship between intake of sugars and caries still exists in the presence of adequate fluoride (34, 35, 41–44). Exposure to fluoride coupled with a reduction in the intake of sugars has been shown to have an additive effect on caries reduction (41). A recent methodical literature review that investigated the importance of intake of sugars in populations exposed to fluoride concluded that where there is adequate exposure to fluoride, consumption of sugars remains a moderate risk factor for caries in most people (45).

Starch-rich staple foods, fruit and dental caries

Epidemiological evidence shows that starch-rich staple foods pose a low risk to dental health (14). 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 (22, 30, 31, 33, 34, 46, 47). In Norway and Japan the intake of starch increased during the Second World War, yet the occurrence of caries was reduced (18). Animal experiments have shown that raw starch is of low cariogenicity (48), cooked starch is about one-third to one-half as cariogenic as sucrose (49), but mixtures of starch and sucrose are potentially more cariogenic than sugars alone (50). Studies that have monitored changes in acid production in plaque on consuming foods (plaque pH studies) have shown starch-containing foods to reduce plaque pH below the levels associated with enamel demineralization, but to a much lesser extent than sucrose. Plaque pH studies measure acid production rather than caries development and take no account of the protective factors found in some starchy foods or the effect of foods on stimulation of salivary flow. Some studies have used hypersensitive electrodes that give an all-or-nothing response to all carbohydrates (51). The results of such experimental studies should not be interpreted in isolation from epidemiological data.

Epidemiological evidence suggests that intake of fruits is not significant in the development of dental caries (34, 52). However, one study did find an association between high fruit consumption (e.g. three bunches of grapes per day) and the number of missing teeth (53). Some plaque pH studies have found fruit to be acidogenic, although less so than sucrose (54). Animal studies have shown that when fruit is consumed very frequently (e.g. 17 times a day) it may induce caries (55), but less so than sucrose.

Potential impact of reduction of sugars on other dietary components

Despite former claims of an inverse relationship between intake of fat and sugars (based on cross-sectional analysis at one time point), there is increasing evidence from longitudinal studies to show 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, not sugars (56, 57). Increasing intake of wholegrain foods, fruits and vegetables and reducing consumption of free sugars is unlikely to lead to an increase in the consumption of fat.

Safe levels of consumption of free sugars

When consumption of sugars is less than 10 kg/person/year the level of dental caries is low (9, 19, 20, 58). Research has consistently shown that when consumption of sugars exceeds 15 kg/person/year dental caries increases and intensifies (i.e. occurs earlier post-eruptively and progresses more rapidly), although exposure to fluoride may increase the safe level of consumption of sugars to approximately 20 kg/year (59). WHO has recommended that countries with a low intake of free sugars do not increase intake and those with higher intakes (>15–20 kg/year) aim to reduce intake of free sugars to less than 10% of energy intake (which equates to < 15–20 kg/year). It is also recommended that the frequency of intake of free sugars is limited to four times or less per day, because above this frequency the amounts of sugars consumed tends to exceed 15 kg/year and higher levels of caries occur (9, 18, 59).

Dietary factors that protect against dental caries

The caries-preventive action of cheese has been reported in experimental (60), human observational (34), and intervention studies (61). Cow's milk contains calcium, phosphorus and casein, all of which inhibit caries, and plaque pH (62) and animal studies (63) have indicated its caries-preventive nature. Recent epidemiological studies have indicated a positive (64) or neutral (65) effect of consumption of cow's milk on caries. Breastfeeding is associated with low levels of dental caries (9); only a few specific case-studies have linked prolonged ad libitum

and nocturnal breastfeeding to dental caries. Foods that stimulate salivary flow, including wholegrain foods, peanuts, hard cheeses and chewing gum protect against decay.

Conclusions

It is important that there is a recommended maximum level for consumption of free sugars because when consumption of free sugars by a population is less than 15–20 kg/person/year levels of dental caries are low. Population goals enable the health risks of populations to be assessed and progress in achieving health-promotion goals to be monitored.

Many countries that are currently undergoing nutrition transition do not have adequate exposure to fluoride and increasing intake of free sugars by these populations could have a severe impact upon the burden of disease. Promotion of adequate exposure to fluoride is important. To minimize dental erosion, the intake of acidic soft drinks should be limited.

The elimination of malnutrition will help to prevent and control developmental defects of the enamel, oral infectious diseases and periodontal disease and may delay the manifestation of the oral symptoms of HIV.

In line with the dietary goals for the prevention of all major diet-related chronic diseases, a diet that is high in fruits, vegetables and wholegrain starchy foods and low in free sugars and fat is likely to benefit many aspects of oral health including prevention of caries, periodontal conditions, oral infectious diseases and oral cancer. ■

Competing interests: none declared.

Résumé

Rôles du régime alimentaire et de la nutrition dans l'étiologie et la prévention des affections bucco-dentaires

L'alimentation joue un rôle majeur dans la prévention des affections bucco-dentaires, dont les caries, l'érosion dentaire, les défauts de développement, les maladies des muqueuses buccales et, dans une moindre mesure, la parodontolyse. Le présent article s'efforce de récapituler d'une manière générale les éléments en faveur d'une association entre régime alimentaire, nutrition et affections bucco-dentaires et d'éclaircir les zones d'incertitude. La sous-nutrition aggrave les affections touchant les muqueuses buccales et la parodontolyse. Elle est aussi l'un des facteurs favorisant les stomatites gangréneuses à pronostic parfois mortel. La sous-nutrition peut s'accompagner de défauts de développement de l'émail, propices à la formation des caries dentaires. On constate également une augmentation de l'érosion dentaire. Les données laissent à penser que les boissons sucrées, importante source d'acides dans le régime alimentaire des pays développés, constituent un facteur étiologique majeur de ce phénomène. Des éléments convaincants, issus d'études expérimentales et sur l'animal, ainsi que d'études d'observation et d'intervention chez l'homme, montrent que les sucres sont le principal facteur

alimentaire associé aux caries dentaires. Si le fluor joue un rôle indiscutable dans la prévention des caries, il n'a pas pour autant éliminé ces pathologies et de nombreuses communautés ne sont pas exposées à des quantités optimales de fluor. La maîtrise de la dose de sucres ingérée demeure donc primordiale pour prévenir la formation des caries. Les travaux de recherche montrent de manière cohérente que le taux de caries dentaires reste faible lorsque la dose de sucres libres ingérée est inférieure à 15 kg par personne et par an. Les résultats de certaines études expérimentales et sur l'animal suggérant un effet cariogène de certains aliments et fruits contenant de l'amidon ne sont pas confirmés par les données épidémiologiques, qui font apparaître une association entre une consommation abondante d'aliments, de fruits et de légumes contenant des fibres et de l'amidon et un faible taux de caries dentaires. L'application des recommandations mondiales encourageant un régime alimentaire riche en aliments, en fruits et en légumes contenant de l'amidon et des fibres et pauvre en sucres libres et en graisses protégera la santé générale, comme la santé bucco-dentaire.

Resumen

Papel de la dieta y la nutrición en la etiología y la prevención de las enfermedades bucodentales

El régimen alimentario tiene un importante papel en la prevención de las enfermedades bucodentales, entre ellas la caries dental, los problemas de desarrollo, las enfermedades de la mucosa oral y, en menor grado, las periodontopatías. El objeto de este artículo es revisar a grandes rasgos la evidencia científica sobre la relación existente entre la dieta, la nutrición y las enfermedades bucodentales, y aclarar algunos puntos de incertidumbre. La desnutrición agrava las enfermedades de la mucosa oral y las periodontopatías, y es también un factor que favorece el noma, dolencia potencialmente mortal. La desnutrición se asocia a problemas de desarrollo del esmalte que aumentan la vulnerabilidad a la caries dental. Se considera que los problemas de erosión dental van en aumento. La evidencia disponible indica que las bebidas gaseosas, una importante fuente de ácidos en la dieta en los países desarrollados, son un factor causal relevante. Estudios experimentales, con animales, y estudios observacionales y de intervención en el ser humano han aportado datos que muestran de forma convincente que los azúcares son el principal

factor alimentario asociado a la caries dental. Pese a su papel indiscutible en la prevención de la caries, el fluoruro no ha logrado eliminar la caries dental, y hay muchas comunidades que no consumen cantidades óptimas de fluoruros. El control del consumo de azúcares, por consiguiente, sigue siendo importante para prevenir las caries. Las investigaciones realizadas han demostrado sistemáticamente que cuando la ingesta de azúcares libres es inferior a 15 kg/persona/año el nivel de caries dental es bajo. A pesar de los estudios experimentales y en animales que indican que algunas frutas y alimentos amiláceos son cariogénicos, esa idea no se ve respaldada por los datos epidemiológicos, que muestran que un consumo elevado de alimentos básicos feculentos, frutas y verduras se asocia a niveles bajos de caries dental. El seguimiento de las recomendaciones mundiales a favor de una dieta rica en alimentos básicos feculentos, frutas y verduras, y pobre en azúcares libres y grasas, constituye una medida de protección tanto de la salud bucodental como de la salud general.

ملخص

دور القوت (النظام الغذائي) والتغذية في حدوث أمراض الفم والوقاية منها

بتسوس الأسنان. وبالرغم من الدور المؤكد للفلوريدات في الوقاية من التسوس، إلا أنها لم تؤد إلى التخلص منه، كما أن العديد من المجتمعات لا تتعرض للكيميائيات المثلى من الفلوريدات. ومن ثم يظل التحكم في مدخول السكريات عاملاً مهماً في الوقاية من التسوس. وقد بينت السبوح أن انخفاض مدخول السكريات الحرة عن 15 كغم للشخص في العام، يؤدي إلى انخفاض مستوى تسوس الأسنان. وبالرغم من أن الدراسات الحيوانية والتجريبية تشير إلى أن بعض الأغذية والفاكهة المحتوية على النشا تسبب التسوس، إلا أن هذا الرأي لا تدعمه معطيات وبائية (إبيدميولوجية) تبين أن المدخول المرتفع من الأغذية النشوية الرئيسية والفاكهة والخضروات يرتبط بمستويات منخفضة من تسوس الأسنان. ومن شأن الالتزام بالتوصيات العالمية التي تشجع تناول القوت المرتفع في الأغذية النشوية الرئيسية والفاكهة والخضروات، والمنخفض في السكريات الحرة والدهون، أن يحمي كلاً من الصحة العامة وصحة الفم.

الملخص: يؤدي القوت (النظام الغذائي) دوراً مهماً في الوقاية من أمراض الفم، مثل تسوس الأسنان، وتآكل الأسنان، والعيوب النمائية، وأمراض الغشاء المخاطي للفم، وبدرجة أقل مرض دواعم الأسنان. والغرض من هذه الورقة استعراض البيانات على الارتباط بين القوت (النظام الغذائي) والتغذية وبين أمراض الفم، وتوضيح مجالات عدم اليقين في هذه العلاقة. فنقص التغذية يزيد من وخامة أمراض الغشاء المخاطي للفم ودواعم الأسنان، كما يسهم في حدوث آكلة الفم المهتدة للحياة. ويرتبط نقص التغذية بالعيوب النمائية لبناء الأسنان، مما يزيد من الاستعداد لتسوس الأسنان. والملاحظ أيضاً أن تآكل الأسنان يتجه إلى التزايد. وتنم البيانات عن أن المشروبات غير الكحولية، التي تمثل مصدراً رئيسياً للأحماض في القوت الغذائي في البلدان المتقدمة، هي عامل رئيسي مسبب لتآكل الأسنان. كما تشير البيانات القاطعة الناتجة عن دراسات المراقبة التجريبية والحيوانية والبشرية، ودراسات المداخلات البشرية، إلى أن السكريات هي العامل الغذائي الرئيسي المرتبط

References

1. Kelly M, Steele J, Nuttall N. *Adult dental health survey. Oral health in the United Kingdom 1998*. London: The Stationery Office; 2000.
2. Chen M, Andersen RM, Barmes DE, Leclercq M-H, Lyttle SC. *Comparing oral health systems. A Second International Collaborative Study*. Geneva: World Health Organization; 1997.
3. Josphura KJ, Willett WE, Douglass CW. The impact of edentulousness on food and nutrient intake. *Journal of the American Dental Association* 1966;129:1261-9.
4. 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.
5. Enwonwu CO. Interface of malnutrition and periodontal diseases. *American Journal of Clinical Nutrition* 1995;61 Suppl:430S-436S.
6. Moynihan PJ, Lingström P. Oral consequences of compromised nutritional well-being. In: Touger-Decker R, Sirois D, Mobley C, editors. *Nutrition and oral medicine*. New Jersey: Humana Press; 2005.
7. Enwonwu CO, Phillips RS, Falkler WA. Nutrition and oral infectious diseases: state of the science. *Compendium of Continuing Education in Dentistry* 2002;23:431-6.
8. Petersen PE. The World Oral Health Report 2003: continuous improvement of oral health in the 21st century — approach of the WHO Global Oral Health Programme. *Community Dentistry and Oral Epidemiology* 2003;31 Suppl1:3-24.
9. *Diet, nutrition and the prevention of chronic diseases*. Geneva: World Health Organization; 2003. WHO Technical Report Series, No. 916.
10. Morse DE. Oral and pharyngeal cancer. In: Touger-Decker R, Sirois DA, Mobley CC, editors. *Nutrition and oral medicine*. New Jersey: Humana Press; 2004:205-21.
11. McLaughlin JK, Grindley G, Block G, Winn DM, Preston-Martin S, Schoenberg JB, et al. Dietary factors in oral and pharyngeal cancer. *Journal of the National Cancer Institute* 1988;80:1237-43.
12. Franceschi S, Fevero A, Conti E, Talamini R, Volpe R, Negri E, et al. Food groups, oils and butter and cancer of the oral cavity and pharynx. *British Journal of Cancer* 1999;80:614-20.
13. Patel A, Glick M. Human immunodeficiency virus. In: Touger-Decker R, Sirois DA, Mobley CC, editors. *Nutrition and oral medicine*. New Jersey: Humana Press; 2004.

14. Rugg-Gunn AJ. *Nutrition and dental health*. Oxford: Oxford Medical Publications; 1993.
15. Jarvinen VK, Rytomaa I, Heinonen OP. Risk factors in dental erosion. *Journal of Dental Research* 1991;70:942-7.
16. 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.
17. Holloway PJ, Mellanby M, Stewart RJC. Fruit drinks and tooth erosion. *British Dental Journal* 1958;104:305-9.
18. Moynihan PJ, Petersen PE. Diet, nutrition and the prevention of dental diseases. *Public Health Nutrition* 2004;7:201-26.
19. Sreebny LM. Sugar availability, sugar consumption and dental caries. *Community Dentistry and Oral Epidemiology* 1982;10:1-7.
20. Woodward M, Walker ARP. Sugar and dental caries: the evidence from 90 countries. *British Dental Journal* 1994;176:297-302.
21. Takeuchi M. Epidemiological study on Japanese children before, during and after World War II. *International Dental Journal* 1961;11:443-57.
22. Fisher FJ. A field study of dental caries, periodontal disease and enamel defects in Tristan da Cunha. *British Dental Journal* 1968;125:447-53.
23. Bang G, Kristoffersen T. Dental caries and diet in an Alaskan Eskimo population. *Scandinavian Journal of Dental Research* 1972;80:440-4.
24. Olsson B. Dental health situation in privileged children in Addis Ababa Ethiopia. *Community Dentistry and Oral Epidemiology* 1979;7:37-41.
25. MacGregor AB. Increasing caries incidence and changing diet in Ghana. *International Dental Journal* 1963;13:516-22.
26. Sheiham A. The prevalence of dental caries in Nigerian populations. *British Dental Journal* 1967;123:144-8.
27. Emslie DR. A dental health survey in the Republic of Sudan. *British Dental Journal* 1966;120:167-78.
28. Roberts IF, Roberts GJ. Relation between medicines sweetened with sucrose and dental disease. *British Medical Journal* 1979;2:14-16.
29. Petersen PE. Dental health among workers at a Danish chocolate factory. *Community Dentistry and Oral Epidemiology* 1983;11:337-41.
30. Harris R. Biology of the children of Hopewood House, Bowral, Australia, 4. Observations on dental caries experience extending over 5 years (1957-61). *Journal of Dental Research* 1963;42:1387-99.
31. Newbrun E, Hoover C, Mettraux G, Graf H. Comparison of dietary habits and dental health of subjects with hereditary fructose intolerance and control subjects. *Journal of the American Dental Association* 1980;101:619-26.
32. Gustafsson BE, Quensel CE, Lanke LS, Lundquist C, Grahnen H, Bonow EE, et al. The Vipeholm dental caries study. The effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for 5 years. *Acta Odontologica Scandinavica* 1954;11:232-364.
33. Scheinin A, Makinen 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 Odontologica Scandinavica* 1976;34:179-98.
34. 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. *Archives of Oral Biology* 1984;29:983-92.
35. Burt BA, Eklund SA, Morgan KJ, Lankin FE, Guire KE, Brown LO, et al. The effects of sugars intake and frequency of ingestion on dental caries increment in a three-year longitudinal study. *Journal of Dental Research* 1988;67:1422-9.
36. Marthaler T. Changes in the prevalence of dental caries: How much can be attributed to changes in diet? *Caries Research* 1990;24:3-15.
37. Konig KP, Schmid P, Schmid R. An apparatus for frequency-controlled feeding of small rodents and its use in dental caries experiments. *Archives of Oral Biology* 1968;13:13-26.
38. Hefti A, Schmid R. Effect on caries incidence in rats of increasing dietary sucrose levels. *Caries Research* 1979;13:298-300.
39. Moynihan PJ. Update on the nomenclature of carbohydrates and their dental effects. *Journal of Dentistry* 1998;26:209-18.
40. *Fluorides and oral health*. Geneva: World Health Organization; 1994. WHO Technical Report Series, No. 846.
41. Weaver R. Fluorine and war-time diet. *British Dental Journal* 1950;88:231-9.
42. Holt RD. Foods and drinks at four daily time intervals in a group of young children. *British Dental Journal* 1991;170:137-43.
43. Kunzel W, Fischer T. Rise and fall of caries prevalence in German towns with different F concentrations in drinking water. *Caries Research* 1997;31:166-73.
44. Beighton D, Adamson A, Rugg-Gunn A. Associations between dietary intake, dental caries experience and salivary bacterial levels in 12-year-old English schoolchildren. *Archives of Oral Biology* 1996;41:271-80.
45. Burt B, Pai S. Sugar consumption and caries risk: a systematic review. *Journal of Dental Education* 2001;65:1017-23.
46. Alfonsky D. Some observations on dental caries in central China. *Journal of Dental Research* 1951;30:53-61.
47. Rugg-Gunn AJ, Hackett AF, Appleton DR. Relative cariogenicity of starch and sugars in a two-year longitudinal study of 405 English school children. *Caries Research* 1987;21:464-73.
48. Grenby TH. The effects of some carbohydrates on experimental dental caries in the rat. *Archives of Oral Biology* 1963;8:27-30.
49. Bowen WH, Amsbaugh SM, Monnell-Torens S, Brunelle S, Kuzmiak-Jones J, Cole MF. A method to assess cariogenic potential of foodstuffs. *Journal of the American Dental Association* 1980;100:677-81.
50. Firestone AR, Schmid R, Muhlemann HR. Cariogenic effects of cooked wheat starch alone or with sucrose and frequency-controlled feeding in rats. *Archives of Oral Biology* 1982;27:759-63.
51. Edgar WM. Prediction of the cariogenicity of various foods. *International Dental Journal* 1985;35:190-4.
52. Clancy KL, Bibby BG, Goldberg HJV, Ripa LW, Barenie J. Snack food intake of adolescents and caries development. *Journal of Dental Research* 1977;56:568-73.
53. Grobler SR, Blignaut JB. The effect of a high consumption of apples or grapes on dental caries and periodontal disease in humans. *Clinical Preventive Dentistry* 1989;11:8-12.
54. Hussein I, Pollard MA, Curzon MEJ. A comparison of the effects of some extrinsic and intrinsic sugars on dental plaque pH. *International Journal of Paediatric Dentistry* 1996;6:81-6.
55. Imfeld TN, Schmid R, Lutz F, Guggenheim B. Cariogenicity of Milchschnitte (Ferrero GmbH) and apple in programme-fed rats. *Caries Research* 1991;25:352-8.
56. Fletcher ES, Rugg-Gunn AJ, Matthews JNS, Hackett AF, Moynihan PJ, Mathers JC, et al. Changes over 20 years in macronutrient intake and body mass index in 11-12 year old adolescents living in Northumberland. *British Journal of Nutrition* 2004;92:321-33.
57. 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. *British Journal of Nutrition* 2002;87:595-604.
58. Miyazaki H, Morimoto M. Changes in caries prevalence in Japan. *European Journal of Oral Sciences* 1996;104:452-8.
59. Sheiham A. Sugars and dental caries. *Lancet* 1983;1:282-4.
60. Rugg-Gunn AJ, Edgar WM, Geddes DAM, Jenkins GN. The effect of different meal patterns upon plaque pH in human subjects. *British Dental Journal* 1975;139:351-6.
61. Gedalia I, Ben-Mosheh S, Biton J, Kogan D. Dental caries protection with hard cheese consumption. *American Journal of Dentistry* 1994;7:331-2.
62. 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 Research* 1985;19:327-34.
63. Bowen WH, Pearson SK, Wuyckhuys BC, Tabak LA. Influence of milk, lactose-reduced milk, and lactose on caries in desalivated rats. *Caries Research* 1991;25:283-6.
64. Levy S, Warren JJ, Broffitt B, Harris SL, Kanellis MJ. Fluoride, beverages and dental caries in the primary dentition. *Caries Research* 2003;37:157-65.
65. Marshall T, Levy SM, Broffitt B, Warren JJ. Dental caries and beverage consumption in young children. *Pediatrics* 2003;112:184-91.