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# Nutrition and Oral Health: A Review

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**ABSTRACT** *Oral health is related to diet in many ways; e.g. influence of nutrition on craniofacial development, infectious diseases of the oral cavity and oral cancer. Oro-dental diseases impact considerably on self-esteem and quality of life and are expensive to treat. Nutrition affects the teeth as well as oral cavity during development and malnutrition may exacerbate periodontal and oral infectious diseases. Diet plays an important role in the prevention of oro-dental diseases including dental caries, dental erosion, developmental defects, oral mucosal diseases and periodontal diseases. However, the most significant effect of nutrition on teeth is the local action of diet in the mouth on the development of dental caries and dental erosion. The objective of this paper is to review the evidence for an association between nutrition, diet and dental diseases and to present dietary recommendations for their prevention.*

**Introduction**

Nutrition can be considered as a core pillar of human development.[1] In the last two centuries, there has been a general improvement in the health of people worldwide attributed largely to changes in nutrition, hygiene and public health. Nutrition can be defined as the study of nutrients in food, how the body uses nutrients, and the relationship between diet, health and disease and how food affects the body. It is the adequate provision of vitamins, minerals, fiber, water and other food components to cells and organisms, to support life.[2] World Health Organization (WHO) defines malnutrition as the cellular imbalance between supply of nutrients and energy and the body's demand for them to ensure growth, maintenance, and specific functions. Malnutrition can either be over-nutrition or under-nutrition.[3] Nutrition is an integral component of oral health. There is a continuous synergy between nutrition and the integrity of the oral cavity in health and disease. Nutrition affects oral health, and oral health affects nutrition. This interdependent relationship sees good nutritional health, promoting good oral health and vice versa.[4]

**Nutrition and Dental Caries**

Dental caries is demineralization of the inorganic part of the tooth with the dissolution of the organic substance due to a multifactorial etiology. The demineralization of the enamel and of the dentine is caused by organic acids that form in the dental plaque because of bacterial activity, through the anaerobic metabolism of sugars found in the diet.[5] The evidence linking dietary sugar to caries comes from a number of different types of study, namely; human intervention studies, human observational studies, animal studies, enamel slab experiments, plaque pH experiments and incubation experiments. Collected evidence from each study type gives an overall picture of the cariogenic potential of sugars and other foods.[6]

Numerous cross-sectional studies attempting to indicate a relationship between sugar consumption and dental caries have been carried out, of which Rugg-Gunn in 1993 has presented a comprehensive summary. The experimental design varies widely between cross sectional studies and often only the correlation coefficient and not the absolute dental caries experience is reported. Even when significant relationships have been found, absolute differences in dental caries experience have sometimes been small. In other studies, large differences in absolute values for decayed, missing and filled deciduous teeth have been found between high and low sugar consumers but numbers have been insufficient for statistical significance.[7]

Granath et al. found a significant relationship between sugar consumption between meals and dental caries, which was independent of fluoride intake or oral hygiene practices.[8] Children with low sugar intake between meals had significantly fewer caries than children with high sugar con-

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sumption between meals.[7] Studies of older children have found the relationships between oral hygiene, water fluoridation, *Streptococcus mutans* and dental caries to be more important than the relationship between sugar intake and dental caries.[5] Human intervention studies in the field of diet and dental caries are rare owing to ethical problems and the difficulty of placing groups of people on strict dietary regimens for long periods of time. The only two studies from which conclusive evidence can be drawn are the Vipeholm study[9] and the Turku study.[10]

Frequency of sugar consumption is undoubtedly an important factor in the etiology of dental caries, but there is also evidence that amount of sugar consumed influences dental caries development independently of frequency, suggesting that both factors are important. Rugg-Gunn and Mikx et al. carried out a study in which five groups of rats received diets of varying sugar concentration and found a significant positive correlation between the sucrose concentration and the incidence of dental caries.[7,11]

Animal studies have compared the relative cariogenicity of different sugars. A theory existed that sucrose was uniquely cariogenic because consumption resulted in dextran formation and increased plaque volume.[5] Studies done on rats have shown sucrose to be more cariogenic than fructose, maltose, lactose or glucose but the rats were superinfected with *Streptococcus mutans* which preferentially take up and utilize sucrose.[5,11] The cariogenicity of uncooked starch is very low but this is seldom consumed by humans. Finely ground and heat treated starch can cause dental caries but to a much lesser extent than sucrose. There is little evidence to show that cooked staple foods such as rice, potatoes, and bread are cariogenic as consumed by humans.[12] Manufactured foods, in which starch is heat treated and hydrolyzed and especially if mixed with sugars, form a potential threat to teeth. The addition of sugar greatly increases the cariogenicity of cooked starches. Less refined starchy foods contain protective factors and it has been suggested that their fibrous nature may aid removal of plaque and food from the mouth, although only from exposed surfaces.[13]

**Nutrition and Periodontal Disease**

One of the oldest observations on nutrition and periodontal health is James Lind's account of scurvy in the first controlled therapeutic trial conducted in 1747. Before Loe et al. published their work on the bacterial causation of gingivitis in 1965, nutritional and other systemic factors were assumed to be virtually exclusively responsible for periodontal diseases, even though it had become clear that vitamin C deficiency could only explain a small segment of the problem. Periodontitis is a ubiquitous chronic inflammatory disease affecting the supporting structures of the teeth and if not promptly recognized and correctly managed can ultimately lead to tooth loss resulting in reduced masticatory function and subsequent alterations in dietary intake and nutritional status. Periodontal disease evolves more quickly in undernourished populations. The pathology starts in the gingiva and could involve the periodontal ligament up to the alveolar bone. The most important risk factor in the development of periodontal disease is represented by inadequate oral hygiene.[14]

Role of vitamins have been extensively researched in relation to periodontal diseases. Deficiencies of vitamin A, C, E and folate have detrimental effects on periodontal health. The role of folate in the prevention of gingivitis is of current interest and further research into the role of free radicals and dietary antioxidants in relation to periodontal disease is expected. Vitamin A is intimately involved in epithelial maintenance and so deficiency is likely to influence the gingiva. In 1962, Shaw reviewed the evidence from animal studies on the relationship between vitamin A deficiency and periodontal disease and found that gingivitis, gingival hypoplasia, proliferation of crevicular epithelium and resorption of alveolar bone were all associated with deficiency of vitamin A. Evidence exists from early animal studies which has shown that deficiency of nicotinic acid, pantothenic acid, riboflavin and folic acid results in gingival inflammation.[14] An epidemiological study by WHO done in Sri Lanka investigated the relationship between vitamin deficiencies and periodontal diseases, has shown that deficiency of vitamin B complex were associated with lower resistance to bacterial irritants. Deficiency of most B complex vitamins is rare in modern society and the only B vitamin which is of current interest in relation to periodontal disease is folic acid. Owing to its high cellular turnover rate gingival epithelium is especially vulnerable to folic acid deficiency, which reduces the ability of this tissue to function as a barrier against bacterial insults.[15]

Vitamin C has well established functions in the maintenance of periodontal tissues. It has a key role in collagen synthesis which is important for the maintenance of the periodontal ligament, gingiva, alveolar bone and blood vessel walls. The immunological role of vitamin C is also important in determining host resistance to plaque microorganisms. Animal studies have demonstrated that acute vitamin C deficiency results in oedema and haemorrhage in the periodontal ligament, osteoporosis of the alveolar bone, tooth mobility and degeneration of the collagen fibres of the gingivae.[16] Epidemiological studies in humans have failed to demonstrate a relationship between vitamin C and gingival health or disease.[17] This is probably because gingivitis and periodontitis are of multifactorial etiology. Patients with severe scurvy can have healthy gingivae, but a deficiency of vitamin C can exacerbate an existing gingivitis. Another study reported that a daily supplement of 70 mg ascorbic acid for six weeks produced marked changes in the ultrastructure of the epithelium and connective tissue of the periodontium. Desmosomal junctions between epithelial cells became longer and the contact surface between cells increased.[18] Dachi et al. found that vitamin C supplementation was unable to reduce gingival sulcus depth in healthy dental students, but it is unlikely that these subjects were deficient.[19] Vitamin C and other antioxidant nutrients may be important in protection of the gingiva from oxidative damage. It has also been suggested that free radicals play a role in collagen destruction in periodontitis by Asman et al.[20]

There exists a relationship between calcium intake and periodontal diseases. This may be due to calcium's role in building density in the alveolar bone that supports the teeth. Calcium is necessary for healthy bones, teeth, muscle contractions and other functions. The ratio of calcium to phosphorus in the diet is also important since secondary hyperparathyroidism causes marked loss of alveolar bone. The composition of the diet can affect the amount and consistency of plaque. Sugar consumption can increase the volume of plaque - the major etiological agent in periodontal disease. Reduction in sugar consumption is not, however, a pragmatic approach to plaque control, because the maximum practical reduction in dietary sugar is not capable of preventing gingivitis. Likewise consumption of fibrous foods is not a substitute for tooth brushing.[20]

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Another study found that supplementing the diet with sucrose increased plaque volume and this was not observed when the diet was supplemented with glucose. In studies of human experimental gingivitis, higher bleeding scores have been shown to occur on high carbohydrate rich diet compared to low carbohydrate rich diet and frequent sugar intake increased gingival inflammation. However, the maximum reduction in sugar in the diet within the limits of practicality is not capable of preventing gingivitis.[21] According to one retrospective cohort study which was conducted to examine whether an exposure to Early Childhood Protein-Energy Malnutrition (ECPEM) was related to a worsened periodontal status in the permanent dentition during adolescence, revealed that ECPEM was related to a poorer periodontal status. Because ECPEM is likely to affect the developing immune system, a person's ability to respond to the colonization with the periodontal pathogens may be adversely affected permanently.[22]

**Malnutrition and Oral Health**

Malnutrition is a multifactorial disease that can have an early onset during the intrauterine life or childhood or it can occur during an individual's lifetime as a result of poor nutrition. Malnutrition appears to have multiple effects on the oral tissues and the subsequent oral disease development. It affects the development of the oral cavity and the progression of the oral diseases through altered tissue homeostasis, a reduced resistance to the microbial biofilm and a reduced tissue repair capacity.[4]

Teeth are affected during their formation by nutrition. Deficiency of nutrients can result in defective enamel formation (enamel hypoplasia) which has aesthetic disadvantages and which may increase the susceptibility to dental caries. Malnutrition can also increase the risk of dental caries by affecting the salivary glands so that the flow rate is reduced and the composition of saliva changed. In poorly nourished communities where sugar is available, malnutrition may increase caries risk by causing defective enamel development and salivary gland atrophy. Defective enamel appears to stem from hypocalcaemia associated with malnutrition and is also caused by vitamin D deficiency. Deficiency of vitamin D and vitamin A and Protein Energy Malnutrition (PEM) have been associated with the enamel hypoplasia. PEM and vitamin A deficiency are also associated with salivary gland atrophy, which subsequently reduces the defense of the oral cavity against infection and its ability to buffer the plaque acids.[6]

It was found that malnutrition was an etiological factor in dental hypoplasia which resulted in increased susceptibility to caries. Despite a general consensus that malnutrition increased defective enamel formation, the mechanism for this was uncertain.[23] It was not until 1981 that Nikiforuk & Fraser showed hypoplasia to be associated with hypocalcaemia, which is a common occurrence in malnutrition due to chronic diarrhea.[24] While dental caries occurs in affluent communities with a good nutritional status, it is rare in many communities in which malnutrition is widespread. However, when developing countries are exposed to sugar in the diet, the level of dental caries is greater than expected from experience in developed countries. This observation has led to the suggestion that malnutrition enhances the cariogenic effect of sugar.[6]

**Nutrition and Dental Erosion**

Dental erosion is progressive irreversible loss of dental tissues which are chemically eroded by extrinsic and intrinsic acids through a process that does not involve bacteria. Poor salivary flow or saliva deficiency is thought to make some individuals more susceptible to acid challenges. Low salivary flow rate or inadequate buffering capacities are factors that exacerbate dental erosion. Intrinsic acids are from vomiting and regurgitation of acid from stomach to oral cavity. The extrinsic acids are from the diet i.e. citric acid, phosphoric acid, ascorbic acid, malic acid, tartaric acid and carbonic acids found in fruits and fruit juices, carbonated soft drinks and still, some herbal teas, dry wines and vinegar containing foods. The critical pH of enamel is 5.5 and therefore, any drink or food with a lower pH may cause dental erosion.[6]

The evidence for an etiological role of diet in the development of dental erosion comes from clinical trials, human observational studies, experimental clinical studies, animal studies, case reports and experiments in vitro. Thomas showed that students who consumed either grapefruit juice, orange juice or cola daily for 6 weeks had signs of dental erosion on the labial surface of incisors and that this was greatest with grapefruit juice.[5] In a case–control study done by Jarvinen et al., it was found that the dietary practices associated with dental erosion were consumption of citrus fruits twice or more per day, consumption of soft drinks once per day and consumption of vinegar or sports drinks more than once a week. Other risk factors included are eating disorders which are largely due to effect of intrinsic acids on vomiting, gastro-esophageal reflux disease and a low salivary flow rate.[25] The United Kingdom National Diet and Nutrition Survey on young people aged 4–18 years showed that the prevalence of dental erosion increased with age in high, moderate and low amount of soft drink consumption. The age-related increase was greatest in the highest amounts of soft drink consumption.[4] Experimental clinical studies have shown that consumption of, or rinsing with acidic beverages significantly lowers the pH of the oral fluids and this is most marked with grapefruit juice. Miller made the important observation that fruit juices were 3–10 times more destructive than whole fruit in rats. However, due to differences in drinking technique and salivary flow and composition, there are difficulties in extrapolating the findings of these studies to humans.[26]

Most of the reports on diet and dental erosion have been single case reports and have shown that extensive dental erosion has been associated with sucking lemon wedges, drinking cola continuously or holding cola in the mouth, addition of baby fruit juices to comforters or reservoir feeders, or mega doses of chewable vitamin C. In-vitro experiments supplement the clinical evidence for an association between diet and dental erosion but vary wide in methodology. However, in general such studies have shown that beverages with a high acidity or a pH 4 can lead to dental erosion. Fruit juices have also been shown to be more erosive than pulped fruits. Citric, malic and tartaric acids are particularly erosive and carbonic acid is the least erosive.[6]

To summarize, dental erosion appears to be an increasing problem in industrialized countries and is related to extrinsic and intrinsic acids. Factors such as salivary flow, fluoride, calcium and phosphate may protect against dental erosion although there is no consensus as to how effective these factors are in prevention. Overall, there is a need for more comprehensive population-based studies on the prevalence of dental erosion using a standard index of measurement. The longitudinal patterns of the dental erosion in populations need to be monitored and related to

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changes in dietary factors.[4]

**Nutrition and Oral Cancer**

The association between nutrition and oral cancer is extremely serious. Oral cancer is a pathology that is diagnosed in three hundred thousand new cases in the world every year and presents the greatest incidence in people who smoke, chew tobacco, and consume alcohol. The use of tobacco can alter the distribution of nutrients such as antioxidants, which develops protective action towards the cells. The study on the incidence of oral cancer has underlined the possibility that diet and nutrition can represent an important etiological factor for oral carcinogenesis. Vitamins A, E, C, and Beta Carotene have antioxidant properties. They neutralize metabolic products, interfere with the activation of pro-carcinogens, inhibit chromosomal aberrations and potentially inhibit the growth of potentially malignant lesions.[4]

The mechanism that connects smoke to this disease has not been discovered but some progress has been made. Smoke modifies the distribution of protective substances such as folic acid and some antioxidants. A rebalancing of nutrients obtained through diet can modify the altered distribution caused by the consumption of tobacco. In an unbalanced diet, there is a depletion of antioxidant nutrients. Fruit and vegetables have, vice versa, important antioxidant properties. Many micronutrients, vitamins in particular are used in chemoprevention programs formulated by the US National Cancer Institute.[6] The National Cancer Institute and the American Cancer Society have established some prudential dietary recommendations for the choice of food; maintain a desirable body weight, eat a varied diet, include a new variety of fruits and vegetables in the daily diet, consume a greater quantity of foods rich in fibre, decrease the total intake of fats (30% less than the total calories), limit the consumption of alcohol, limit the consumption of salted foods or foods preserved with nitrates.[5]

In a recent research it is shown that lycopene can exert protective effects against 4-nitroquinoline-1-oxide induced tongue carcinogenesis through reduction in cell proliferation and enhanced cellular adhesion, suggesting a new mechanism for the anti-invasive effect of lycopene.[27] In a recent report which has shown that diets rich in animal origin and animal fats are positively, and those rich in fruit and vegetables and vegetable fats inversely related to oral and pharyngeal cancer risk.[28]

Malnutrition also interferes negatively with humoral and cellular immune competence and with tissue and reparative functions. In addition, the alteration of the liver function can change the way drugs are metabolized. Therefore, malnutrition can interfere with oncological therapy and increase the severity of the collateral effects[4].

**Conclusion**

Nutrition is a major modifiable determinant of chronic disease, with scientific evidence increasingly supporting the view that alterations in diet have strong effects, both positive and negative, on health throughout life. Most importantly, dietary adjustments may not only influence present health, but may determine whether or not an individual will develop such diseases as cancer, cardiovascular disease and diabetes much later in life. However, these concepts have not led to a change in policies which are practiced. In many developing countries, food policies remain focused only on under-nutrition and are not addressing the prevention of chronic diseases. There is an increasing need to prevent and control the public health problems of chronic diseases by promoting appropriate diets and healthy lifestyles.

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