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REVIEW ARTICLE

Relationship between Diet and Dental Caries

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ABSTRACT-

An unhealthy diet has been implicated as risk factors for several chronic diseases that are known to be associated with oral diseases. Studies investigating the relationship between oral diseases and diet are limited. Therefore, this study was conducted to describe the relationship between healthy eating habits and oral health status. The dentistry has an important role in the diagnosis of oral diseases correlated with diet. Consistent nutrition guidelines are essential to improve health. A poor diet was significantly associated with increased odds of oral disease. Dietary advice for the prevention of oral diseases has to be a part of routine patient education practices. Inconsistencies in dietary advice may be linked to inadequate training of professionals. Literature suggests that the nutrition training of dentists and oral health training of dietitians and nutritionists is limited.

Key words- Diet, Caries, Micronutrient Deficiency, Oral Cancer.

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INTRODUCTION-

A dynamic relation exists between sugars and oral health. Diet affects the integrity of the teeth, quantity, pH and composition of the saliva and plaque. Sugars and other fermentable carbohydrates after being hydrolyzed by salivary amylase, provide substrate for the actions of oral bacteria which in turn lower plaque and salivary PH. The resultant action the beginning of tooth demineralisation. According to the American Dietetic association Nutrition is an integral component of oral health. Diet and nutrition are major multifactorial environmental factors in the etiology and pathogenesis of craniofacial diseases. There is new evidence showing that excessive sugar consumption increases the risk of caries, even if the correlation between sugar intake and dental health has weakened due to exposure to fluoride. Moreover, there are still patient groups whose fluoride exposure seems to be insufficient to tolerate the generally increased level of sugar intake. Nonnutritive sweeteners offer no energy and can sweeten

with little volume. Both sugar alcohols and nonnutritive sweeteners can replace the sugars and are sometimes referred to as sugar substitutes, sugar replacers, or alternative sweeteners (1). We searched the pertinent literature in Pub Med and MEDLINE databases by using key words such as sugar in dental caries, sugar substitute in dental caries, and various types of sugar in dental caries. Clinical relevance patients with high caries risk and insufficient fluoride exposure need personal advice and recommendations related to the use of sugars, sweets, juices and soft drinks, even today. Patients with increased risk of obesity may also benefit from dietary recommendations conducted by dental professionals. Dental caries, a chronic disease is unique among human and is one of the most common important global oral health problems in the world today. It is the destruction of dental hard a cellular tissue by acidic by products from the bacterial fermentation of dietary carbohydrates especially sucrose. It progresses slowly in most of the people which

results from an ecological imbalance in the equilibrium between tooth minerals and oral biofilms which is characterized by microbial activity, resulting in fluctuations in plaque pH due to bacterial acid production, buffering action from saliva and the surrounding tooth structure. The microbial community of caries is diverse and contains many facultative and obligatory anaerobic bacteria. *S. mutans* is the most primary associated with it. Dental caries can affect the human in various ways i.e. presence of tooth pain, infection or dysfunction of the stomatognathic system can limit the necessary ingestion of energetic foods, affecting the growth in children and adults as well as their learning, communication skills and recreational activities. Moreover, oral and pharyngeal cancers and oral tissue lesions are also significant health concern. Therefore, prevention is more affordable. Dietary modification should be recommended.

FACTORS RESPONSIBLE IN ADDITION TO SUGARS AFFECTS THE CARIES PROCESS

1. The form of food or fluid
2. Duration of exposure
3. Nutrition composition
4. Sequence of eating
5. Salivary flow
6. presence of buffers
7. Oral hygiene

SUGAR RESPONSIBLE FOR CARIES

1. Sucrose (table sugar)-a simple sugar made from cane sugar or beets
2. Fructose-a simple sugar in fruits, plants, honey
3. Maltose-a complex sugar in barley and malt sugar
4. Lactose-a complex sugar in milk
5. Dextrose-a refined simple sugar from corn, sugar cane or beets
6. Glucose-a simple sugar in fruits, vegetable and grains

Sucrose for years was billed as the arch criminal of dental caries because it was considered to be so much more cariogenic than other sugars (3). However, later research has suggested that the differences between sucrose and the various monosaccharide in terms of cariogenic potential are less than originally believed (4,5). This is a difficult issue to study in humans because of the variability of the human diet, so views are based principally on extrapolations from animal studies and laboratory research. One study in Sweden involving a small number of preschool children found that those consuming invert sugar (a mixture of glucose and fructose) in place of sucrose had a lower caries increment in 2 years, although the differences did not reach statistical significance [6]. However, one could speculate whether reduced consumption of sucrose in the developed countries has been a factor in the sharp reduction in a proximal and smooth surface caries relative to the overall caries decline. This speculation is based on the fact that the

production of extracellular polysaccharides in plaque depends on sucrose and that smooth surface caries will only develop with plaque that adheres by means of extracellular polysaccharides [7].

Sugars can be readily metabolized by many bacteria involved in dental biofilm formation, generating acid byproducts that can lead to demineralization of the tooth structure. Lactose (milk sugar) has been shown to be less acidogenic than other sugars and less cariogenic, based on animal studies (8). Sucrose has been given special consideration as a cariogenic substrate owing to its unique ability to support the synthesis of extracellular (water-soluble and water-insoluble) glucans by *mutans streptococci*, enhancing its accumulation in the plaque. Some animal studies on rats super infected with *Streptococcus mutans* have reported increased carcinogenicity of sucrose compared to other sugars; however, this effect appears to be bacterial strain-specific and not consistent across different animal models. More recent clinical studies have indicated that the caries-associated virulence of *glutans* may have more to do with an alteration in plaque ecology by increasing the porosity of plaque, permitting deeper penetration of dietary sugars and greater acid production adjacent to the tooth surface (9).

DIETARY FACTORS RESPONSIBLE FOR CARIES-

AMOUNT, FORM, FREQUENCY, PATTERN OF SUGAR INTAKE

It is generally accepted that the prevalence of caries is related to the form in which sugar is ingested and the frequency of its consumption. By "form" we meant the physical consistency of the sugar-containing foods. Distinctions are made between liquid and adhesive (sticky) foods as well as foods which vary in adhesiveness between the extremes. The term "frequency" refers to the number of times per day that sugary foods are eaten. It is clear that both form and frequency affect the length of time that teeth are exposed to sugar (10). However, the relative importance of frequency versus the total amount of sugar consumption is difficult to evaluate.

The relationship of the physical consistency of food to caries is not entirely clear. Several studies have incriminated the stickiness of foods as prime factor in the initiation of caries (11,12)]. Others have shown that semisolid and even liquid sugar-containing foods can be very cariogenic (13). Ericsson (14) reported that the frequent intake of lozenges can cause rampant decay, so can liquids as is evident in the case of nursing bottle caries (15,16) and in experimental human studies investigations by von der Fehr et al. (17) and Geddes et al. (18). It is likely that the length of time that the teeth are exposed to sugar-containing foods rather than simply the form of the food is a critical factor in the promotion of caries (10). Many studies point to the frequency of eating sugars to be of greater etiological importance for caries than the total

consumption of sugars (19,20). The primary evidence comes from the Vipeholm study (11). A positive correlation between the frequency of consumption of confectionery and sugar-containing gum and the DMF rate was also found in a study conducted on 14-year-old Caucasian, Hawaiian, and Japanese schoolchildren in Hawaii (21). A range in intake from zero to five or more sweets per day was followed by a corresponding increase in DMF scores. Against the general perception that frequency of intake is more important than the amount of sugars eaten, two longitudinal studies reported the amount of sugars intake to be more important than frequency (8,22,23). However, there is undoubtedly a strong correlation between the two variables (8) with an increase in one factor often leading to an increase in the other.

Although a high intake frequency increases the overall length of time that the teeth are exposed to sugars, it does not give complete information on the total time of exposure. The total cariogenic load is also determined by the form of the food product; that is, the physical consistency of the sugar-containing foods affects their retention time in the mouth. Distinctions can be made between liquids that are cleared rapidly and adhesive (sticky) foods that vary widely in retentiveness. Particularly high retention rates have been found for products such as sweet biscuits, crackers, and potato chips (crisps) (24). Other aspects of intake pattern are also believed to be of importance. The sequence of eating a cariogenic food product during a meal or snack can alter its cariogenic properties. Both cheese and peanuts can reduce the acid production after a previous intake of sucrose-containing foods. Conversely, starches can increase the cariogenic properties of sugars if they are consumed at the same time. The stickiness of starch enhances the retention time of sugars, resulting in a prolonged pH fall, as occurs in breakfast cereals with added sugars. Another important issue that is difficult to account for in determining the relationship between the dietary intake and caries is that many food products contain hidden sugars. Examples of such sugar-containing products may vary from one country to another. It is not obvious to most people that sugars may be a major constituent in products such as marmalade, breakfast cereals, flavored crisps (chips), caviar, ketchup, and, in many countries, bread. Thus, just focussing on confectioneries may have little impact on reducing caries activity if an individual is exposed to many other sugary products per day (8)

DEVELOPMENT OF DIET ON TOOTH DEVELOPMENT STAGES

Teeth in a preeruptive phase are influenced by the nutritional state. A lack of vitamins D and A and protein-energy malnutrition have been associated to hypoplasia of the enamel and atrophy of the salivary glands, conditions that determine a greater susceptibility to caries. Some hypoplasia and pits on the surface of the enamel correlate

to a lack of vitamin A, a lack of vitamin D is associated to the more diffused hypoplastic forms. The structural damage can testify to the period in which the lack of nutrition occurred (15).

EFFECT OF NUTITION ON PERIODONTAL DISEASES

Periodontal disease evolves more quickly in undernourished populations: "...the pathology starts in the gum and could interest 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 (Figure 5). Data supplied by the National Health and Nutrition Examination Survey 2001/02 underlined that a low level of folic acid is associated to periodontal disease. The serum level of folates is an important index of periodontal disease and can represent an objective that should be pursued in the promotion of periodontal health (19). Malnutrition and bad oral hygiene represent two important factors that predispose for necrotizing gingivitis. Prevention programs against disease must therefore include a correct evaluation of the immune system and the promotion of nutritional programs. The aim of nutritional support in inflammatory diseases is to provide the right energy and nourishment to respond to the increased demand for protein synthesis in the acute phase, inflammatory mediators, antioxidant defence mechanisms, as well as for the promotion of tissue repairment. Some nutrients have a very important role in the resolution of the inflammatory process. These observations confirm the relationship between diet and periodontal disease (20). In a recent interview, the president of the American Society of Periodontology, Michael P. Rethman(20), underlined the importance of diet for a healthy smile. In particular, the correlation between the intake of calcium and periodontal disease can be due to the role that calcium has in the density of the alveolar bone that supports teeth. Also the intake of vitamin C is fundamental for maintenance and for the activation of reparative mechanisms thanks to its antioxidant properties (20)

EFFECT OF DIET ON ORAL CANCER

The association between diet and oral cancer is extremely serious. It 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 develop a protective action toward the cells: smokers present levels of carotenoids and vitamin E in the blood that are superior to those in the oral mucous and, in addition, have a different distribution in comparison to the norm; the levels of folates in the blood and in the cells of the oral tissues of smokers are inferior to those of nonsmokers; the inside of the cheeks of smokers presents numerous micronuclei (modifications typical of pre- and

neoplastic lesions) (25,26). The study of the incidence of this illness has underlined the possibility that diet can represent an important etiological factor for oral carcinogenesis. Vitamins A, E, C, and Beta Carotene have antioxidant properties.(i)They neutralize metabolic products.(ii)They interfere with the activation of procarcinogens.(iii)They inhibit chromosomal aberration.(iv)They potentially inhibit the growth of malignant lesions (leukoplakia)

EFFECT OF DIET ON ORAL CANDIDOSIS

A significant correlation has been evinced with a lack of iron (Figure 10). This causes alterations in the epithelium with consequent atrophy and alteration in cellular turnover, an alteration in the iron-dependent enzymatic system depression in cell-mediated immunity, phagocytosis, and in the production of antibodies. The correlation between candidiasis and the lack of folic acid, vitamins A, B1, B2, vitamins C, K, zinc, and a diet rich in carbohydrates is also significant(25)

MICRONUTRIENT DEFICIENCIES AND MUCOSAL DISORDERS

Various types of nutritional deficiencies can produce oral mucosal diseases. Changes such as swelling of the tongue, papillary atrophy, and surface ulceration are possible in case of micronutrient deficiencies (iron, folate, vitamin B12)(26) (27)

EFFECT OF DIET ON DENTAL EROSION

“Dental erosion is the progressive irreversible loss of dental tissue that is chemically corroded by extrinsic and intrinsic acids through a process that does not involve bacteria.”

Extrinsic Acids: Derived from Diet: Citric, phosphoric, ascorbic, malic, tartaric, and carbonic acids that are found in fruit, in fruit juices, in drinks, and in vinegar.

Intrinsic Acids: They are derived from serious gastroesophageal reflux

REFERENCES:

1. Belcastro G, Rastelli E, Mariotti V, Consiglio C, Facchini F, Bonfiglioli B. Continuity or discontinuity of the life-style in central Italy during the Roman imperial age-early middle ages transition: Diet, health, and behavior. *American Journal of Physical Anthropology: The Official Publication of the American Association of Physical Anthropologists*. 2007 Mar;132(3):381-94.
2. Dion N, Cotart JL, Rabilloud M. Correction of nutrition test errors for more accurate quantification of the link between dental health and malnutrition. *Nutrition*. 2007 Apr 1;23(4):301-7.
3. Singh A, Bharathi MP, Sequeira P, Acharya S, Bhat M. Oral health status and practices of 5 and 12 year old Indian tribal children. *Journal of Clinical Pediatric Dentistry*. 2011 Apr 1;35(3):325-30.
4. Dye BA, Nowjack-Raymer R, Barker LK, Nunn JH, Steele JG, Tan S, Lewis BG, Beltran-Aguilar ED. Overview and quality assurance for the oral health component of the National Health and Nutrition Examination Survey (NHANES), 2003-04. *Journal of public health dentistry*. 2008 Sep;68(4):218-26.
5. Scardina GA, Messina P. Nutrition and oral health. *Recenti progressi in medicina*. 2008 Feb;99(2):106-11.
6. Bang G, Kristoffersen T. Dental caries and diet in an Alaskan Eskimo population. *European Journal of Oral Sciences*. 1972 Oct;80(5):440-4.
7. Olsson B. Dental health situation in privileged children in Addis Ababa, Ethiopia. *Community Dentistry and Oral Epidemiology*. 1979 Feb;7(1):37-41.
8. Scheinin A, Mäkinen KK. Turku sugar studies: An overview. *Acta Odontologica Scandinavica*. 1976 Jan 1;34(6):405-8.
9. Gustafsson BE. Vipeholm (Sweden) dental caries study: survey of the literature on carbohydrates and dental caries. *Acta Odontol Scand*. 1954;11:206-31.
10. Lingstrom P, Van Houte J, Kashket YS. Food starches and dental caries. *Critical Reviews in Oral Biology & Medicine*. 2000 Jul;11(3):366-80.
11. Moynihan PJ, Ferrier S, Jenkins GN. Dental caries: The cariostatic potential of cheese: cooked cheese-containing meals increase plaque calcium concentration. *British Dental Journal*. 1999 Dec;187(12):664.
12. Gordon N. Oral health care for children attending a malnutrition clinic in South Africa. *International journal of dental hygiene*. 2007 Aug;5(3):180-6.
13. Lupi-Pegurier L, Muller-Bolla M, Fontas E, Ortonne JP. Reduced salivary flow induced by systemic isotretinoin may lead to dental decay. A prospective clinical study. *Dermatology*. 2007;214(3):221-6.
14. Faggella A, Guadagni MG, Cocchi S, Tagariello T, Piana G. Dental features in patients with Turner syndrome. *European Journal of Paediatric Dentistry*. 2006 Apr;7(4):165.
15. Kargul B, Caglar E, Lussi A. Erosive and buffering capacities of yogurt. *Quintessence international*. 2007 May 1;38(5).
16. Kitchens M, Owens B. Effect of carbonated beverages, coffee, sports and high energy drinks, and bottled water on the in vitro erosion characteristics of dental enamel. *Journal of Clinical Pediatric Dentistry*. 2007 Apr 1;31(3):153-9.
17. Huew R, Waterhouse P, Moynihan P, Kometa S, Maguire A. Dental caries and its association with diet and dental erosion in Libyan schoolchildren. *International Journal of Paediatric Dentistry*. 2012 Jan;22(1):68-76.
18. Yu YH, Kuo HK, Lai YL. The association between serum folate levels and periodontal disease in older adults: data from the National Health and Nutrition Examination Survey 2001/02. *Journal of the American Geriatrics Society*. 2007 Jan;55(1):108-13.
19. Al-Zahrani MS. Increased intake of dairy products is related to lower periodontitis prevalence. *Journal of Periodontology*. 2006 Feb 1;77(2):289-94.
20. Verkleij-Hagoort A, Blik J, Sayed-Tabatabaei F, Ursem N, Steegers E, Steegers-Theunissen R. Hyperhomocysteinemia and MTHFR polymorphisms in association with orofacial clefts and congenital heart defects: a meta-analysis. *American Journal of Medical Genetics Part A*. 2007 May 1;143(9):952-60.
21. G. A. Scardina and P. Messina, “Good Oral Health and Diet,” *Journal of Biomedicine and Biotechnology*, vol. 2012,

- Article ID 720692, 8 pages, 2012. <https://doi.org/10.1155/2012/720692>.
22. Heba Ashi, Guglielmo Campus, Hélène Bertéus Forslund, Waleed Hafiz, Neveen Ahmed, and Peter Lingström, "The Influence of Sweet Taste Perception on Dietary Intake in Relation to Dental Caries and BMI in Saudi Arabian Schoolchildren," *International Journal of Dentistry*, vol. 2017, Article ID 4262053, 8 pages, 2017. <https://doi.org/10.1155/2017/4262053>.
 23. Taghavi N, Yazdi I. Type of food and risk of oral cancer.
 24. El-Rouby DH. Histological and immunohistochemical evaluation of the chemopreventive role of lycopene in tongue carcinogenesis induced by 4-nitroquinoline-1-oxide. *Archives of oral biology*. 2011 Jul 1;56(7):664-71.
 25. Paillaud E, Merlier I, Dupeyron C, Scherman E, Poupon J, Bories PN. Oral candidiasis and nutritional deficiencies in elderly hospitalised patients. *British Journal of Nutrition*. 2004 Nov;92(5):861-7.
 26. Nagao T, Warnakulasuriya S, Ikeda N, Fukano H, Yamamoto S, Yano M, Miyazaki H, Ito Y. Serum antioxidant micronutrient levels in oral lichen planus. *Journal of oral pathology & medicine*. 2001 May;30(5):264-7.
 27. Thongprasom K, Youngnak P, Aneksuk V. Folate and vitamin B12 levels in patients with oral lichen planus, stomatitis or glossitis. *The Southeast Asian journal of tropical medicine and public health*. 2001 Sep;32(3):643-7.