

Full Length Research Paper

Dental caries: The most common disease worldwide and preventive strategies

Dogan Ozdemir

Ishik University, Faculty of Dentistry, Basic Sciences, 44, Arbil, Iraq.
E-mail: ozdemirtalha@gmail.com, Fax: +9647506369587.

Accepted 22 July, 2014

Dental caries is the most prevalent infectious disease worldwide. There are different causes of tooth caries such as the dietary habit and oral hygiene. The morphology of the tooth also plays an important role in the formation of caries. The organic material of the tooth is made up of hydroxyapatite crystals. These crystals are demineralized in the presence of high acidity. The pH of the mouth environment is critical in the mineralization-demineralization process. It is a well-known fact that when carbohydrate is consumed in daily diet with the help of bacteria, the sugar is fermented and lactic acid is formed. Consequently the pH of the environment decreases which will lead the dental caries formation. The purpose of this review is to give a comprehensive look at dental caries in terms of microbiology, diet, oral hygiene and its preventive measures.

Keywords: Dental caries, tooth enamel, oral hygiene, diet and lactic acid.

INTRODUCTION

Tooth decay (dental caries) is a significant health problem worldwide. It affects not only the vast majority of adults but also children, from 60% to 90% of them. In other words, six to nine children in every 10 are affected by tooth decay (Marinho et al. 2013).

Dental caries is an infective transmittable bacterial disease characterized by a multi factorial pathology (Xhemnica et al. 2008). The main players in the etiology of this disease are: cariogenic bacteria, fermentable carbohydrates, a susceptible tooth host and time which is described in the following graph (Richard et al. 2010).

Carbohydrates play a very important role in the development of caries. When the carbohydrates (especially sugar and starch) are consumed in diet highly, the fermentation occurs as per the following sequence; Starch $\xrightarrow{\text{hydrolysis}}$ Glucose $\xrightarrow{\text{glycolysis}}$ Pyruvate $\xrightarrow{\text{fermentation}}$ Lactic acid.

The environment pH of mouth is 7.4 which is slightly basic. When the lactic acid produced the pH decreases which will result as acidity in the mouth and this will demineralize the tooth and will lead to teeth decay. S.

mutans is the common infectious bacteria that leads to biofilm formation then fermentation.

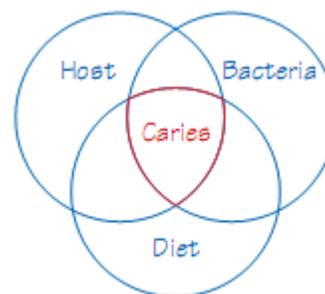


Figure 1. Requirement for caries to occur.

Caries

Tooth decay (dental caries) is a significant health problem worldwide. It affects not only the vast majority of adults but also children, from 60% to 90% of them. In

other words, six to nine children in every 10 are affected by tooth decay. Repairing and replacing decayed teeth is extremely costly in terms of time and money and is a major drain on the resources of healthcare systems (Sofia et al. 2010).

The tooth surface is covered with a biofilm—a slime layer consisting of millions of bacterial cells, salivary polymers, and food debris. Uncontrolled, this biofilm can easily reach a thickness of hundreds of cells on the surfaces of the teeth. The formed biofilm, also called plaque, provides an excellent adhesion site for the colonization and growth of many bacterial species. (Marinho et al. 2013). The localized destruction of the tissues of the tooth by bacterial action, either enamel or cementum is demineralized by microbial acids. The initial caries lesion is sub-surface, due to acid diffusion. The primary lesion that is detectable clinically is known as a white spot and can be reversed by re-mineralization and re-growth of hydroxyapatite crystals, a process enhanced by fluoride. Advanced caries results in cavitations that is irreversible and can progress to the dentin and into the pulp chamber ultimately causing necrosis and periapical abscesses. (Lakshman, 2006).

The disease is the most prevalent of the chronic diseases affecting the human race. The original still-prevailing theory explaining the disease process implicates carbohydrates, oral microorganisms, and acids as the main factors in the caries process. Chemico-parasitic process consisting of two stages, the decalcification of enamel, which results in its total destruction, and the decalcification of enamel (Selwitz et al. 2007)

Dental caries is also a common disease in children. (Sheiham 2006; Petersen 2009). Untreated dental caries can affect body weight, growth and quality of life in preschool children.

(Li, 2002) Caries experience in early childhood has been linked to caries experience in the permanent dentition in several studies (Alm et al. 2007, Skeie et al. 2006 and Powell LV. 1998). The burden of dental caries lasts a lifetime because once the tooth structure is destroyed it will usually require restoration and on-going maintenance throughout life (Tove et al. 2012)

The major factors that involved the etiology of caries are: host factors (tooth, saliva and diet) bacteria.

Host factors; Tooth

The structure is important: some areas of the same tooth are much more susceptible to carious attack than others, possibly because of differences in mineral content (especially fluoride). Fissure on enamel and space between teeth.

Saliva

Mechanical washing action of saliva removes food debris and unattached oral microorganisms. It has a high

buffering capacity which tends to neutralize acids produced by plaque bacteria on tooth surfaces, and it is supersaturated with calcium and phosphorus ions, which are important in the remineralization of white-spot lesions. Saliva also acts as a delivery vehicle for fluoride. Saliva contains enzymes such as lactoperoxidase, lysozyme, lactoferrin and immuno-globulin (Ig)A, which can inhibit plaque bacteria. Daniels et al. 1975, Finn et al. 1955 and Frank 1965).

Diet

There is a direct relationship between dental caries and the intake of carbohydrates. The most cariogenic sugar is sucrose. Sucrose is highly soluble and diffuses easily into dental plaque, Sucrose is rapidly fermented to acidic end products, but it is also the only dietary carbohydrate that can be transformed into extracellular polysaccharides (EPS) in the plaque. Thus, it is considered to be the most cariogenic carbohydrate in the human diet acting as a substrate for the production of EPS and acids (Sofia et al. 2010).

Cariogenic streptococci produce glucans and other polysaccharides from excess carbohydrate (often sucrose) in the diet, leading to plaque accumulation; production of acids (principally lactic acid), that generate a low pH environment and enrich for aciduric organisms. Dental caries cannot occur in the absence of dietary fermentable carbohydrates and, therefore, it has been characterized as a “dieto-bacterial” disease. (Richard et al. 2010). Since the original observations of Miller, researchers have recognized fermentable carbohydrates as the “fuel” for the caries process, and in the 1940s, Stephan (Stephan 1940 and Stephan R. 1944), demonstrated the relationship between caries and sugar exposure, leading to the acidification of dental plaque. Moreover, Weiss and Trithart²⁴ reported a direct relationship between caries experience and the frequency of between-meals consumption of sweet snacks, which findings supported those of the earlier Vipeholm study in Sweden (Gustafsson et al. 1954).

Bacteria

Today, *S. mutans* and *streptococcus sobrinus* are considered to be the main aetiological microorganisms in caries disease, with lactobacilli and other microorganisms participating in the disease progression. *S. mutans* and *Streptococcus sobrinus* are the main cariogenic microorganisms. (Tanzer et al. 2001 and Nurelhuda et al. 2010). These acid-producing pathogens inhabiting the mouth cause damage by dissolving tooth structures in the presence of fermentable carbohydrates such as sucrose, fructose, and glucose. (Schafer et al. 2000 and Caufield and Griffen 2000).

Most of the investigations (Van et al. 1982, Berkowitz et al. 1984 and Milnes 1985) have shown that in children

with tooth decay, *S. mutans* has regularly exceeded 30% of the cultivable plaque flora. These bacterial masses are often associated with carious lesions, white spot lesions, and sound tooth surfaces near the lesions.

There is a relationship between colonization by *S. mutans* and subsequent attack on the teeth to generate white spot (early caries) lesions. Epidemiological studies in humans then began to seek correlations between numbers of *S. mutans* present on teeth and the development of dental caries. Numerous studies suggested positive correlations, and showed the idea that *S. mutans* levels were a good indicator of active caries, and may indeed be predictive (Richard et al. 2010).

Virulence factors of *S. mutans* are prevalent plaque adhesin-like cell surface proteins, acid tolerance, acid production, and production of glucosyl transferases, mutacin and intracellular polysaccharides. *S. mutans* ferment many different sugars, and they appear to metabolize sucrose to lactic acid more rapidly than other oral bacteria.

Prevention of tooth decay

Over the years many detailed methods have been described in the prevention of tooth decay. These multifaceted approaches increase the resistance in the dental hard tissues, eliminate or reduce the cariogenic microflora, minimize the availability of microbial diet as sugar especially between meals and changing diet habit of individuals (not to have sugar consumption between meals).

Dental caries is a dynamic dieto-microbial disease involving cycles of demineralization and remineralization. The early stages of this process are reversible by modifying or eliminating etiologic factors (such as plaque biofilm and diet) and increasing protective factors (such as fluoride exposure and salivary flow). This approach manages dental caries by means of prevention and cure, reserving surgical approaches for those whose disease severity and tissue loss leave no other option (Domenick et al. 2009).

Restriction of sugar consumption is considered a major caries-preventive measure, but the use of fluorides, education and oral hygiene are confounding practices and play more important roles. It has been found that the consumption of sweetened baked goods was significantly related to caries (Anderson et al. 2009).

Dairy products have properties that protect teeth against caries, Harper et al. 1987) and eating cheese after exposure to sugar rapidly neutralizes plaque acidity. (Schachtele and Jensen 1984). Probably cheese contain calcium phosphate and increase salivary flow.

A wide range of sugar substitutes have low or no cariogenic potential. (Zero 2008). For example, Sucralose is a high-intensity non-cariogenic sweetener (Bowen et al. 1990) and Xylitol has been reported to have anti-cariogenic properties.

(Burt, 2006). Chewing sugar-containing gum increases caries risk, (Burt, 2006) but chewing sugar-free gum after meals can reduce caries risk (Glass, 1981). Some food additives may have protective properties that reduce cariogenicity; for instance, berries can reduce bacterial adherence and glucosyltransferase activity of *S. mutans*, (Stokey 2008) and tea extracts inhibit salivary amylase activity (Koo et al. 2006).

Having the information and skills to make appropriate decisions about a child's diet, hygiene, and fluoride use are essential in order to establish healthy behaviors early in a child's life. Children with caries have daily consumption of sugars from liquids (Weinstein et al. 1992).

Caries-promoting sugars such as sucrose, glucose and fructose, contained in fruit juices, (Newbrun 1982, Grenby et al. 1990 and Persson et al. 1985) are readily metabolized by *S. mutans* and lactobacilli to organic acids that demineralize enamel and dentin. The use of nursing bottles and "sippy cups" enhances the frequency of exposure. This type of feeding behavior during sleep intensifies the risk of caries, as oral clearance and salivary flow rate are decreased during sleep (Van 1981). Parents should be advised to change daily diet habit to reduce or destroy cause of tooth decay.

Children's teeth should be brushed last thing at night, before bedtime and on at least on other occasion. Children should have their teeth brushed with fluoride toothpaste. Eating directly after brushing should be avoided, to prevent fluoride from being washed-out of the mouth prematurely. Fluoride is a mineral that prevents tooth decay. Fluoride is added to the water supply in many areas. It can also be applied directly to teeth in the form of fluoride varnish. This is applied to first (baby) and permanent teeth (depending on the age of the child) usually by a dental professional from two to four times a year. Because it stays on the surface of the tooth for relatively long periods of time it releases fluoride in an efficient and effective way (Marinho et al. 2013).

Flossing can remove plaque from approximal tooth surfaces and may have a role in reducing caries. A combination of brushing with fluoride toothpaste and flossing is more efficient, especially if regular flossing is carried out by an adult. Flossing on its own cannot be recommended for the prevention of dental caries in pre-school children without the associated application of fluoride to the dentition (NCQ).

CONCLUSION

In conclusion, tooth decay is one of the significant and costly diseases in the world. Thus, strategies to reduce the risk for dental caries are vital. The strategies usually involve decreasing the growth or activity of bacteria

especially *S. mutans*. We should change daily diet. Parents should advise children to avoid eating between meals especially carbohydrate containing food. The correct method and frequency of brushing should be followed—in the morning and before going to bed and preferably after every major meal. The use of various interdental cleaning aids such as dental floss, interdental brush (Klock and Krasse 1978) should be inculcated. The prevention strategy lies within dietary habit. Therefore, children at below certain age must be regularly advised and followed.

REFERENCE

- Alm A, Wendt LK, Koch G, Birkhed D. 2007. Prevalence of approximal caries in posterior teeth in 15-year-old Swedish teenagers in relation to their caries experience at 3 years of age. *Caries Res*; 41: 392-8.
- Anderson C. A., M. E. J. Curzon, C. Van Loveren, C. Tatsi and M. S. Duggal, 2009. Obesity Department of Paediatric Dentistry, Child Dental Health, Leeds Dental Institute, reviews; 10 (Suppl. 1), 41–54
- Berkowitz RJ, Turner J, Hughes C. 1984. Microbial characteristics of the human dental caries associated with prolonged bottle-feeding. *Arch Oral Biol*;29:949-51.
- Bowen W, Birkhed D. 1986. Dental caries: dietary and microbiology factors. In: Granath L, McHugh WD, eds. *Systematized Prevention of Oral Disease: Theory and Practice*. Boca Raton, Fla.: CRC Press;19–41.
- Bowen WH, Young DA, Pearson SK. 1990. The effects of sucralose on coronal and root-surface caries. *J Dent Res*;69(8):1485–1487.
- Burt BA. 2006 The use of sorbitol- and xylitol-sweetened chewing gum in caries control (published correction appears in *JADA*;137(2):190–196.
- Burt BA. 2006. The use of sorbitol- and xylitol-sweetened chewing gum in caries control (published correction appears in *JADA* 2006;137[4]:447). *JADA*;137(2):190–196.
- Caufield PW, Griffen AL. 2000. Dental caries. An infectious and transmissible disease. *Pediatr Clin North Am*;47:1001-19,v.
- Daniels TS, Silverman S, Michalski JP, Greenspan JS, Sylvester RA, Talal N. 1975. The oral component of Sjogren's syndrome. *Oral Surg*;39:875–85.
- Dental and Oral Health Problems: Prevention and Services 1 -II-233.
- Domenick T. et al. 2009. The Journal of the American Dental Association September vol. 140 no. suppl 1 25S-34S.
- Finn SB, Klapper CE, Voker JF. 1955. Intra-oral effects upon experimental hamster caries. In: RF Sognaes (ed). *Advances in experimental caries research*. Washington, DC: American Association for the Advancement of Sciences;:155–68.
- Frank RM, Herdly J, Phillippe E. 1965. Acquired dental defects and salivary gland lesions after irradiation for carcinoma. *J Am Dent Assoc*;70:868–83.
- Glass RL. 1981. Effects on dental caries incidence of frequent ingestion of small amounts of sugars and stannous EDTA in chewing gum. *Caries Res*;15(3):256–262. Medline
- Grenby TH, Mistry M, Desai T. 1990. Potential dental effects of infants' fruit drinks studied in vitro. *Br J Nutr*; 64(1):273–83.
- Gustafsson BE, Quensel CE, Lanke L Set al. 1954. The Vipeholm dental caries study: the effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for five years. *Acta Odontol Scand*;11(3–4):232–264.
- Harper DS, Osborn JC, Clayton R, Hefferren JJ. 1987. Modification of food cariogenicity in rats by mineral-rich concentrates from milk. *J Dent Res*; 66(1):42–45.
- Klock B. Krasse B. 1978. Effect of caries preventive measures in children with high numbers of *S. mutans* and lactobacilli. *Scand J Dent. Res*;86:221.
- Koo H, Nino de Guzman P, Schobel BD, Vacca Smith AV, Bowen WH. 2006. Influence of cranberry juice on glucan-mediated processes involved in *Streptococcus mutans* biofilm development. *Caries Res*;40(1): 20–27.
- Lakshman Samaranayake . 2006. *Essential microbiology for Dental students*, pp.267
- Li Y, Wang W. 2002. Predicting caries in permanent teeth from caries in primary teeth: an eight-year cohort study. *J Dent Res* .81: 561-6.
- Marinho VCC, Worthington HV, Walsh T, Clarkson JE. 2013. Fluoride varnishes for preventing dental caries in children and adolescents. *Cochrane Database of Systematic Reviews*., 7:279.
- Marinho VCC, Worthington HV, Walsh T, Clarkson JE. 2013. Fluoride varnishes for preventing dental caries in children and adolescents (Review) *The Cochrane Collaboration*.
- Marinho VCC, Worthington HV, Walsh T, Clarkson JE. 2013. Fluoride varnishes for preventing dental caries in children and adolescents (Review) *The Cochrane Collaboration*.
- Milnes AR, Bowden GH. 1985. The microflora associated with developing lesions of nursing caries. *Caries Res*;19:289-97.
- National clinical guideline (NCQ) Prevention and management of dental decay in the pre-school child. A national clinical guideline, 83, pg.24.
- Newbrun E. 1982. Sugar and dental caries: a review of human studies. *Science*; 217(4558):418–23.
- Nurelhuda NM, Al-Haroni M, Trovik TA, Bakken V. 2010. Caries experience and quantification of *Streptococcus mutans* and *Streptococcus sobrinus* in saliva of Sudanese schoolchildren. *Caries Res*;44:402-7.
- Persson LA, Holm AK, Arvidsson S, Samuelson G. 1985. Infant feeding and dental caries, a longitudinal study of Swedish children. *Swed Dent J* ; 9(5):201–6.
- Petersen PE. 2009. Global policy for improvement of oral health in the 21st century – implications to oral health research of World Health Assembly 2007, World Health Organization. *Community Dent Oral Epidemiol*; 37: 1-8.

- Powell LV. 1998. Caries prediction: a review of the literature. *Community Dent Oral Epidemiol*; 26: 361-71.
- Richard J. Lamont, Howard F. Jenkinson , 2010. *Oral Microbiology at a Glance* pp.7
- Richard J. Lamont, Howard F. Jenkinson . 2010. *Oral Microbiology at a Glance*, pp.38
- Richard J. Lamont, Howard F. Jenkinson .2010. *Oral Microbiology at a Glance*, pp:37
- Schachtele CF, Jensen ME. 1984. Can foods be ranked according to their cariogenic potential? In: Muhlemann HR, Guggenheim B, eds. *Cariology Today*. Basel, Switzerland: Karger; 136–146.
- Schafer TE, Adair SM. 2000. Prevention of dental disease. The role of the pediatrician. *Pediatr Clin North Am*;47:1021-42,v-vi.
- Selwitz RH, Ismail AI, Pitts NB. 2007. Dental caries. *Lancet*; 369: 51-9.
- Sheiham A. 2006. Dental caries affects body weight, growth and quality of life in pre-school children. *Br Dent J*; 201: 625-6.
- Skeie MS, Raadal M, Strand GV, Espelid I. 2006. The relationship between caries in the primary dentition at 5 years of age and permanent dentition at 10 years of age – a longitudinal study. *Int J Paediatr Dent*; 16: 152-60.
- Sofia D. Forssten , Marika Björklund and Arthur C. 2010. *Ouwehand. Streptococcus mutans, Caries and Simulation Models Nutrients*, 2, 290-298;
- Sofia D. Forssten , Marika Björklund and Arthur C. Ouwehand. 2010. *Streptococcus mutans, Caries and Simulation Models Nutrients*, 2, 290-298;
- Stephan R. 1940. Changes in hydrogen-ion concentration on tooth surfaces and in carious lesions. *JADA*;27(5):718–723.
- Stephan R. 1944. Intra-oral hydrogen-ion concentrations associated with dental caries activity. *J Dent Res*;23(4):257–266.
- Stookey GK. 2008 The effect of saliva on dental caries. *JADA*;139 (5 suppl):11S–17S.
- Tanzer JM, Livingston J, Thompson AM. 2001. The microbiology of primary dental caries in humans. *J Dent Educ*;65:1028-37.
- Tove I. Wigen and Nina J. Wang, 2012 . Parental influences on dental caries development in preschool children. An overview with emphasis on recent Norwegian research ,*Norsk Epidemiologi*; 22 (1): 13-19
- Van Houte J, Gibbs G, Butera C. 1982. Oral flora of children with “nursing bottle caries”. *J Dent Res*;61:382-5.
- Van Houte J. 1981. Experimental odontogenic infections-effects of inoculation methods, dietary carbohydrates, and host age. In: Tanzer JM, editor. *Animal models in cariology (special supplement to Microbiology, Abstracts-Bacteriology)*; 231–8.
- Weinstein P, Domoto P, Wohlers K, Koday M. 1992. Mexican-American parents with children at risk for baby bottle tooth decay: pilot study at a migrant farm workers clinic. *ASDC J Dent Child*; 59(5):376–83.
- Weiss RL, Trithart AH. 1960. Between-meal eating habits and dental caries experience in preschool children. *Am J Public Health Nations Health*;50(8):1097–1104. Medline.
- Wendt LK, Hallonstein AL, Koch G. 1991. Dental caries in one- and two-year-old children living in Sweden. Part I – A longitudinal study. *Swed Dent J*; 15(1):1–6.
- Winter GB. 1980. Problems involved with the use of comforters. *Int Dent J*; 30(1):28–38.
- Xhemnica L, Sulo D, Rroco R, Hysi D. 2008. Fluoride varnish application: a new prophylactic method in Albania. Effect on enamel carious lesions in permanent dentition. *Paediatric Dent* ., 9(2): 93-96.
- Zero DT. 2008. Are sugar substitutes also anticariogenic? *JADA*; 139(5 suppl):9S–10S.