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Analytical approaches to study Dental Caries

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ABSTRACT

Dental caries is an infection, bacterial in origin that causes demineralization and destruction of the hard tissues of the teeth. It is a result of the production of acid by bacterial fermentation of food debris accumulated on the tooth surface. If demineralization exceeds saliva and other remineralization factors such as from calcium and fluoridated toothpastes, these once hard tissues progressively break down, producing dental caries Today, caries remains one of the most common diseases throughout the world. Cariology is the study of dental caries. Early diagnosis of the caries lesion is important because the carious process can be modified by preventive treatment so that the lesion does not progress. If the caries disease can be diagnosed at an initial stage (e.g. white spot lesion) the balance can be tipped in favour of arrestment of the process by modifying diet, improving plaque control, and appropriate use of fluoride. This article describes the various technologies available to aid the dental practitioners in detecting and diagnosis of dental caries at the earliest stage of its formation, assessing the activities of the detected carious lesion, and quantitatively or qualitatively monitoring of the lesion over time.

Keywords: Dental caries, Diagnosis, Detection.

INTRODUCTION

Dental caries is an infectious microbiologic disease of the teeth that results in localized dissolution and destruction of calcified tissues. Development of caries require interaction between plaque microorganism, susceptible tooth, substrate and time. The infection results in loss of tooth minerals that begins on the outer surface of the tooth and can progress through the dentin to the pulp, ultimately compromising the vitality of the tooth. There are number of risk and modifying factors which affect the mineral equilibrium in one direction or another, i.e. towards remineralisation or demineralisation. There has been remarkable progress in the reduction of dental caries over the past 30 years. Changes have been observed not only in the prevalence of dental caries, but also in the distribution and pattern of the disease in the population. Specifically, it has been observed that the relative distribution of dental caries on tooth surfaces has changed, and the rate of lesion progression through the teeth is relatively slow for most people. The use of fluoride in public water supplies, in toothpaste, and in professional dental products, improved oral hygiene, and increased access to dental care have played major roles in this dramatic improvement. Nevertheless, dental caries remains a significant problem. Finding an accurate method for detecting and diagnosing any disease has been the goal of the healing arts since the time of Socrates.

Depending on the extent of tooth destruction, various treatments can be used to restore teeth to proper form, function, and aesthetics, but there is no known method to regenerate large amounts of tooth structure. Instead, dental health organizations advocate preventive and prophylactic measures, such as regular oral hygiene and dietary modifications, to avoid dental caries. Using non-invasive quantitative diagnostic methods it should be possible to detect lesions at an initial stage and subsequently monitor lesion changes over time during which preventive measures could be introduced.

Fig 1

Fig 2

The word caries derives from the Latin for rot or rotten. The earliest theory was the "tooth worm theory" proposed by the ancient Chinese in 2500 BC, where it posited a toothworm as the cause of this rottenness. 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. A chemico-parasitic process consisting of two stages, The decalcification of enamel, which results in its total destruction, and the decalcification of dentin. 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, Wendt, Koch & Birkhed (2007). The burden of dental caries lasts a lifetime be-cause once the tooth structure is destroyed it will usually require restoration and on-going maintenance throughout life.

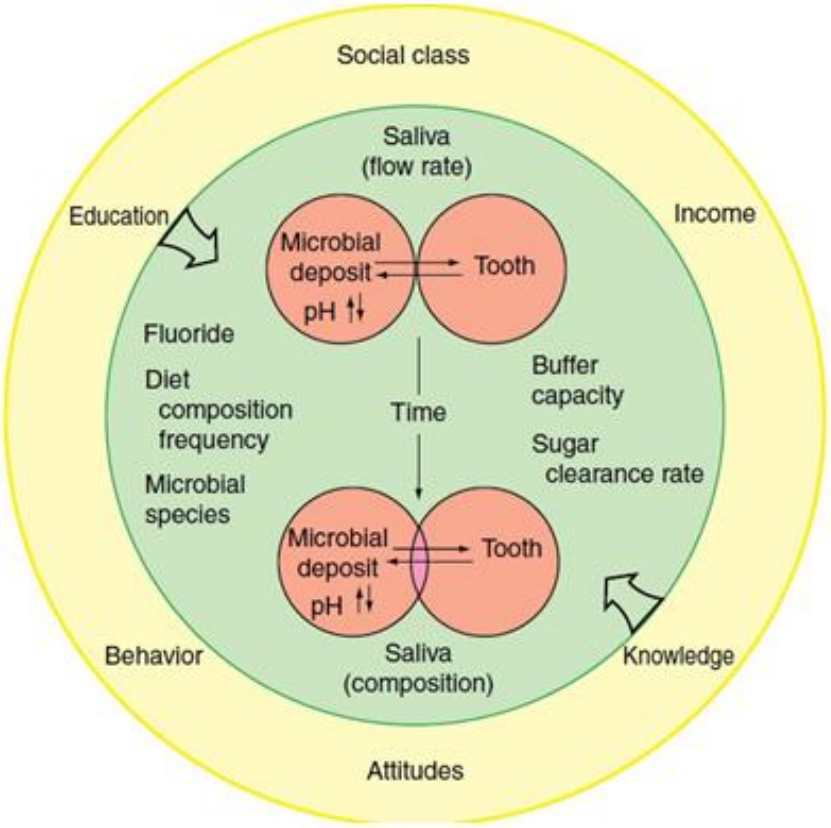

Figure 3: Schematic diagram of the determinants of the carious process

CAUSE FACTORS OF CARIES

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).

Saliva

The 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.

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, acting as a substrate for the production of extracellular polysaccharides and acids. Cariogenic streptococci produce glycan (water-insoluble) from sucrose, which in addition to facilitating initial adhesion of the organisms to the tooth surface serve as a nutritional source and a matrix for further plaque development. Recent data indicate that high lipid content in saliva enhances caries activity. On the other hand, it is demonstrated that a caniogenic diet becomes less caniogenic when it is combined with cheese or milk products, probably because of the content of calcium phosphate in these products (Johansson et al., 2009). Dental caries cannot occur in the absence of dietary fermentable carbohydrates and, therefore, it has been characterized as a "dietobacterial" disease (Bowen & Birkhed, 1986) .Since the original observations of Miller, (1902) researchers have recognized fermentable carbohydrates as the "fuel" for the caries process, and in the 1940 and 1944 Stephan demonstrated the relationship between caries and sugar exposure, leading to the acidification of dental plaque. Moreover, Weiss and Trithart 1960 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.

Tooth decay in young children is of particular concern for a number of reasons:

- It is painful for the child, disturbs eating and sleeping patterns and is distressing for both child and parent;
- Treatment is challenging and often requires secondary and specialist care under conscious sedation or general anaesthesia;
- It may impact on the developing permanent dentition, self esteem and aesthetics;
- It highlights that an opportunity has been missed to prevent what should have been preventable.

PREVENTIVE STRATEGIES

Dairy products have properties that protect teeth against caries and eating cheese after exposure to sugar rapidly neutralizes plaque acidity. A wide range of sugar substitutes have low or no cariogenic potential. For example, sucralose is a high-intensity non-cariogenic sweetener and xylitol has been reported to have anticariogenic properties. Chewing sugar-containing gum increases caries risk but chewing sugar-free gum after meals can reduce caries risk. Some food additives may have protective properties that reduce cariogenicity; for instance, cranberries can reduce bacterial adherence and glucosyl transferase activity of S. mutans and tea extracts inhibit salivary amylase activity.

The conventional approach to the treatment of dental caries was to remove and replace diseased tissue with an inert restoration. This approach made no attempt to cure the disease and the patient often returned some months later.

Dental caries is a dynamic dietomicrobial disease involving cycles of demineralization and remineralization. The early stages of this process are reversible by modifying or eliminating etiologic factors 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.

Our understanding of caries has changed markedly in the last century. A National Institutes of Health consensus statement Domenick (2009) acknowledged that tooth restoration does not stop the caries process and emphasized the need for improved diagnosis, prevention and management of caries in its early (that is, noncavitated) stages. Still, dental practitioners and researchers alike have an incomplete understanding of the natural history of caries. Cognizant of the limitations of current clinical diagnostic methods and concerns about potential disease progression, dentists tend to err on the side of more aggressive operative treatment than often might be warranted.

- Parents and cariers should be advised that cheese is a good high energy food for toddlers as it is non-cariogenic and may be actively protective against caries.
- Children should have their teeth brushed with fluoride toothpaste. Toothbrushing should commence as soon as the primary teeth erupt.

- Children's teeth should be brushed last thing at night, before bedtime and on at least one other occasion.
- Eating directly after brushing should be avoided, to prevent fluoride from being washed out of the mouth prematurely.
- 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.

It is important that there is a recommended maximum level for consumption of free sugars because when free sugars consumption by a population is low, dental caries levels are low. Population goals enable the oral health risks of populations to be assessed and health promotion goals monitored. The best available evidence indicates that the level of dental caries is low in countries where the consumption of free sugars is below 15–20 kg/person/yr. This is equivalent to a daily intake of 40–55 g and the values equate to 6–10% of energy intake. It is of particular importance that countries which currently have low consumption of free sugars (15–20 kg/person/yr) do not increase consumption levels. For countries with high consumption levels, it is recommended that national health authorities and decision makers formulate country-specific and community- specific goals for reduction in the amount of free sugars, aiming towards the recommended maximum of no more than 10% of energy intake, in addition to the development of oral health programmes to protect from dental caries.

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

Conclusion

To bring about a reduction in caries levels in populations a focus beyond the purely biological to the societal setting is required. A focus on making healthier choices easier and unhealthy ones more difficult is required (e.g. it should be usual for toothpaste to be fluoridated). Two fundamentally different approaches to prevention are discussed: a high-risk strategy that targets efforts at those considered to be high risk, versus a whole-population strategy that targets everyone. The arguments for the whole population approach are persuasive. Finally, and perhaps most interesting and persuasive of all, is the common risk factor approach to prevention. Hygiene, diet and tobacco cessation are relevant to many diseases, so that in future dentists may find themselves promoting health in general, rather than dental health in particular.

References

[1]. Klinke T, Kneist S, de Soet JJ, Kuhlisch E, Mauersberger S, Forster A, (2009). Acid production by oral strains of
[2]. Candida albicans and lacto-bacilli. Caries Res; 43(2):83–91. CrossRefMedline.
[3]. Miller W. (1902). The presence of bacterial plaques on the surface of teeth and their significance. Dent Cosmos; 44:425–446.
[4]. Johansson-788S, Diet and dental caries" The Journal of the American Dental Association. V. 140 no. suppl 1 25S-34S.
[5]. Bowen W, Birkhed D. (1986). Dental caries: dietary and microbiology factors. In: Granath L, McHugh WD, eds.
[6]. Stephan R. (1940). Changes in hydrogen-ion concentration on tooth surfaces and in carious lesions. JADA;27(5):718–723.
[7]. Weiss RL, Trithart AH. (1960). Between-meal eating habits and dental caries experience in preschool children.
[8]. Bowen WH, Young DA, (1990). Pearson SK. The effects of sucralose on coronal and root-surface caries. J Dent Res ; 69(8):1485–1487.
[9]. 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.
[10]. Bowen WH, Young DA, Pearson SK. (1990). The effects of sucralose on coronal and root-surface caries. J Dent Res;69(8):1485–1487.
[11]. 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.
[12]. Splieth CH, Flessa S. Modelling lifelong costs of caries with and without fluoride use. European Journal of Oral Sciences 2008; 116: 164-169.

[13]. Bagramian RA, Garcia-Godoy F, Volpe AR. The global increase in dental caries. A pending public health crisis. American Journal of Dentistry 2009; 22: 3-8.
[14]. Longbottom CL, Huysmans MC, Pitts NB, Fontana M. Glossary of key terms. Monographs in Oral Science 2009; 21: 209-216.
[15]. Kidd EA, Ricketts DN, Pitts NB. Occlusal caries diagnosis: A changing challenge for clinicians and epidemiologists. Journal of Dentistry 1993; 21: 323-331.
[16]. Bagramian RA, Russell AL. Epidemiological study of dental caries experience and between-meal patterns. Journal of Dental Research 1973; 52: 342–7.
[17]. Bergendal B, Hamp SE. Dietary pattern and dental caries in 19-year-old adolescents subjected to preventive measures focused on oral hygiene and/or fluorides. Swedish Dental Journal 1985; 9: 1–7.
[18]. Larsson B, Johansson I, Ericson T. Prevalence of caries in adolescents in relation to diet. Community Dentistry and Oral Epidemiology 1992; 20: 133–7.