Abstract

Dental caries is a major oral and public health concern in the 21st Century. Despite a global decline in caries, the disease still remains persistent, particularly among poor and disadvantaged groups in developed and developing countries.\(^2\) According to the World Oral Health Report 2003, dental caries remains a major public health problem in most industrialised countries, affecting 60–90% of schoolchildren and the vast majority of adults.\(^2\)

The global burden of oral conditions is shifting from severe tooth loss toward severe periodontitis and untreated caries, increasing the burden on health care resources. Oral conditions affected 3.9 billion people, and untreated caries in permanent teeth was the most prevalent condition evaluated for the entire Global Burden of Disease 2010 Study with a global prevalence of 35% for all ages combined.\(^3\)

Our understanding of the aetiology of dental caries has evolved over the last 100 years with recent research highlighting the need to adopt a minimally invasive approach to the treatment and prevention of dental caries. This includes a focus beyond fluoride and diet, incorporating risk management techniques such as chairside evaluation of saliva buffering capacity, modulation of plaque ecology and the use of sugar free gum. This article presents management and prevention strategies for dental caries in the 21st Century.

Current concepts in the aetiology of dental caries

Our understanding of the aetiology of dental caries has evolved over the last 100 years from that of the non-specific plaque hypothesis (Miller 1890) through the specific plaque hypothesis (Loesche 1979) to our present day understanding of the ecological plaque hypothesis (Marsh 1991). The ecological plaque hypothesis proposes that carious disease can be attributed to changes in the oral environment that disrupt homeostasis between the plaque microflora and the host and is considered a modified hypothesis of Loesche and Miller.\(^4\)

This hypothesis states that bacteria of the non-mutans species are crucial for maintaining stability and balance of the oral microflora. In the presence of fermentable carbohydrates these bacteria within plaque, rapidly metabolise sugars, producing acids at the tooth surface. When formed in sufficient amounts and for adequate periods of time, this creates conditions that favour dissolution of calcium and phosphates from the tooth surface effecting demineralisation of enamel.\(^5\)

When plaque pH is lowered, the concentration of calcium and phosphate ions needed for salivary saturation increases, reaching “critical pH” at 5.5 to 5.7 when the hard tissues will start to dissolve to maintain salivary saturation levels.\(^6\) The extent of dissolution/demineralisation depends on the oral environment, specifically, the extent and duration of pH drop, the presence of fluoride, the presence and composition of saliva (buffering capacity), the frequency of sugar challenges and the presence of anti-bacteria. The prevention of caries therefore can be seen as maintaining a balance between pathological factors and protective factors.\(^7\) (Fig 1)

The protective capacity of saliva in mineralisation-remineralisation

Saliva plays an important role in buffering plaque acids and provides the medium for remineralisation. Saliva contains calcium and phosphate concentrations that supersaturate saliva with respect to hydroxyapatite, thereby neutralising acids.

Demineralisation occurs at below pH 5.5 when the oral environment is undersaturated with mineral ions, relative to the mineral content of enamel. Calcium and phosphate ions from hydroxyapatite are liberated, resulting in net loss of minerals.\(^3,6\)

Remineralisation occurs when dietary carbohydrate is removed and calcium and phosphate ions in saliva are re-incorporated in the enamel matrix. Once the pH of the biofilm is raised to approximately pH 7.0 demineralisation is arrested. An early carious lesion therefore may be arrested and prevented from reaching cavitation by altering the oral environment in favour of remineralisation.\(^8\)

The buffering capacity of saliva plays a critical role in helping restore a neutral pH at the tooth surface, modulating the Stephan Curve, providing mouth clearance and dilution of plaque acids and restoring the oral environment to a neutral pH. Kleinberg et al were able to demonstrate the effect of saliva in bringing plaque pH back to neutral more rapidly than in saliva deficient subjects.\(^9\)

Caries can therefore be considered a dynamic equilibrium between demineralisation and remineralisation mediated by saliva composition and dietary factors.

Bacterial adaptation in the aetiology of dental caries

Dental caries can be observed in the absence of pathogenic bacteria (i.e. S. mutans and Lactobacilli) as the acidic environment may select other acid thriving (aciduric) bacteria that are also capable of significant acid production, albeit at a slower rate.\(^9\)
The ability for cariogenic bacteria to adapt to an acid environment has also been observed. Pathogenic bacteria like *S. mutans* for example have adapted to enable them to be acid tolerant and thus survive and flourish in dental plaque.\(^{10}\) This has enabled *S. mutans* to dominate other non-pathogenic bacteria that do not possess this mechanism. The ability of *S. mutans* to survive at low pH is an important virulence factor in the pathogenesis of dental caries.\(^{11}\)

**The role of arginine in modulating the composition of plaque**

Other bacteria such as *S. sanguinis* have developed a mechanism known as the arginine deiminase pathway to break down arginine present in saliva into its (basic) alkali components, ammonia and carbon dioxide. The elevated pH induced by urea and arginine catabolism is thought to be important in inhibiting the development of tooth decay and in modulating the composition of plaque ecology.\(^{12}\)

A correlation between higher concentrations of arginine in saliva and a lower incidence of dental caries has been demonstrated\(^{13}\) further strengthening the idea that base production is one of the key factors in oral health and oral biofilm pH homeostasis. These base substances directly neutralise acids in plaque.

The use of genetically engineered, ammonia-producing oral streptococci as potential agents for the control of dental caries has been demonstrated. This may provide the foundation for the potential use of arginine to moderate plaque acidification and to control the emergence of a cariogenic biofilm.\(^{14}\)

A new technology based on arginine and insoluble calcium in a fluoridated toothpaste was released offering superior caries prevention and modification of plaque biofilm composition.\(^{15,16}\)

**21st Century management and prevention strategies**

The acid tolerance of bacteria in the carious lesion is much higher than that of healthy commensal (ie non-cariogenic) bacteria. The resting plaque pH in the healthy mouth is therefore higher than that with active carious lesions.

The long term eradication of oral biofilms is not possible as this would adversely affect the oral ecology. Beneficial and commensal bacteria compete with pathogens for colonisation.

The key to maintaining caries balance is to focus on rebuilding and maintaining a healthy biofilm and influencing the oral environment. Healthy biofilm acts as a storage reservoir for fluoride (and other ions such as calcium and phosphate) causing enhanced fluoride retention and exchange between these ions and tooth enamel, increasing the length of remineralisation time to combat caries.\(^{17}\)

The essential aspects of prevention and management of caries involve assessing the underlying caries risk factors, which cannot be detected during a routine examination. The main risk factors implicated in the oral environment include:

**Diet:** the frequency of sugar and acid hits. The most common method is via dietary analysis with the patient. It is important that the dental team update their knowledge on the nutrition facts and the cariogenic potential of various foods.

**Bacteria:** the composition of the individual’s plaque e.g.: *Streptococcus mutans* count and an evaluation of bacterial activity as measured by lactic acid production. Chair-side assessment methods are commercially available that are based on a species-specific monoclonal antibody response, where the results are obtained within 30 minutes. An increased Mutans streptococci level is usually evident in the presence of more incipient lesions. Lactobacilli require retentive areas to proliferate and hence, a high count is usually associated with the presence of frank cavitations.\(^{18}\)

**Fluoride:** past and current exposure. Fluoride inhibits demineralisation, by penetrating and coating the enamel crystals to prevent dissolution, and by enhancing remineralisation, resulting in enamel with a higher F content and lower acid solubility.\(^{19}\)

When fluoride is present, the critical pH for demineralisation is lowered, meaning a lower plaque pH can be tolerated before demineralisation occurs.\(^{20}\) The formation of calcium fluoride on the tooth surface acts as a reservoir and is driven into hydroxyapatite in response to significant drops in pH.\(^{21}\) Whilst fluoride is efficient in providing protective factors that reduce the susceptibility of the tooth surface to demineralisation and cavitation, it does not reduce pathological factors by inhibiting plaque formation or by altering the microbiology of plaque biofilms in favour of non-pathogenic organisms.

**Saliva composition:** Salivary flow rate is the most important clinical parameter affecting dental caries susceptibility. With a reduced quantity of saliva, the oral clearance of microorganisms and food remnants is impaired.\(^{22}\) In addition, the pH and buffering capacity is reduced and a more acidic environment results in the growth of the aciduric cariogenic organisms.\(^{19}\) The calcium and phosphate content is also lower with reduced saliva, and thus the remineralisation capacity is compromised. A low salivary flow rate accentuates the pH decrease in dental plaque.\(^{23}\) The buffering capacity of the saliva and its ability to reduce the acidity can be measured by chair-side saliva testing.

**The role of sugarfree gum in caries management**

Saliva stimulation from chewing gum helps in the management of caries by neutralising plaque acids and remineralising tooth enamel. The chewing of sugar-free gum stimulates saliva production which can last up to 2 hours with increases in both flow rate and pH being observed following chewing for 20 minutes.\(^{24,25}\) Stimulated saliva also contains increased levels of salivary electrolyte and protein concentrations, in particular bicarbonate and calcium ions. This allows recovery of plaque pH above 5.5 within 20 minutes compared to two hours without gum.\(^{26}\)

In terms of caries prevention, a randomised clinical study from Beiswanger et al\(^ {27}\) demonstrated an 11% drop in DMF scores in children using sugarfree gum after meals. A further study has also shown a 30% reduction in DMF scores over a two year period.\(^ {28}\)

**The modern approach to caries prevention**

Minimal intervention and management of the oral environment is a modern approach to caries prevention. The public is also aware that restorative dentistry is not enough to ensure good oral health. It is important for the profession to now consider how new treatment and prevention modalities can be incorporated into everyday clinical practice.

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Over this four day congress, modern concepts in the aetiology, diagnosis, prevention and management of caries were explored, along with evidence-based strategies and contemporary therapeutic approaches for the prevention and management of this global health issue: caries.

This report contains information from the following presentations:

Hien Ngo: Managing caries in clinical practice; the oral environment.
References


14. M Dobbs: Saliva and oral health; the science behind the chewing gum claims.

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