



REVIEW ARTICLE

Overview on concepts of dental caries

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ARTICLE INFO

Article history:

Received 26th June 2015

Accepted 10th Sept 2015

Keywords:

Dental caries, Current concepts,
Remineralisation, Demineralization

ABSTRACT

Epidemiologic data in the literature indicates that understanding of caries has changed in the last century. Scientific advances in cariology in the past 150 year have led to the understand the dental caries as a chronic, dietomicrobial, site-specific disease caused by a shift from protective factors favouring tooth demineralization to destructive factors leading to demineralization. This paper focuses on the history of dental caries with an emphasis on relevant developments in understanding of different concepts and its implication. It provides a brief overview of important concepts, and scientific developments that have shaped our current understanding of one of the most common diseases in humans. The primary emphasis was placed on the importance of appreciating caries as a common, complex, chronic disease whose deleterious effects can be mitigated best with ongoing use of appropriate, risk-based protective measures based on the current concept.

Introduction

It is very strange that the hardest tissue of the body, the enamel, which is indestructible otherwise, can disintegrate in the oral environment.

The word caries is derived from Latin meaning rot or decay. It is similar to the Greek word Ker meaning death.

Benjamin Franklin stated that “Hot things, sharp things, sweet things, cold

things, all rot the teeth and make them look like old things”.

Dental caries is perceived as plagues of modern times affecting human race. In quantitative sense this is true. The sugar laden diets of many technologically advance society can be associated with increased prevalence of dental decay.

Dental caries is a microbial disease of the calcified tissues of the teeth, characterized by demineralization of the inorganic portion and

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destruction of the organic substance of the tooth. Miles Markley, leader of preventive dentistry stated that “loss of even a part of a human tooth should be considered a serious injury” and that dentistry’s goal should be to preserve healthy natural tooth structure. Therefore, there have been calls for changing in paradigm from “drilling and filling” to managing the disease as an infection.

Historical Perspective

The earliest concept of caries is tooth worm theory, which originated in Egypt in 12th century B .C and persists in some cultures until present day¹.

The first of the modern concept was the, view that, dental decay was brought about by inflammation .In later part of 19th century W. D Miller demonstrated that, it was not possible to produce inflammatory process in hard structure of the teeth^{2,3}.

Most of the 19th century was dominated by two separate schools of thoughts –one viewed dental caries in terms of effect of acid, and other considered dental caries as the direct result of bacterial attack⁴.

In 20th century the concept of caries has changed, it is regarded in two aspects –one as bacterial disease⁵.Second, as not only as bacterial disease but it is specific bacterial disease produced by *Streptococcus mutans*⁶.

Therefore caries is considered as infectious, specific bacterial transmissible disease.

Current concepts states that caries as a multifactorial, reversible, complex disease caused by an imbalance in physiologic equilibrium between tooth mineral and bio film fluid⁷.

The original approach to the treatment of dental caries was purely surgical. The demineralised area of the tooth structure was eliminated and rebuilds it with an inert restoration. G.V. Black developed the surgical approach. It is considered, highly destructive. G.V. Black in 1908 pointed out that “fillings are not curative” but treatment of a symptom rather than a disease^{1,8}

Over a time, modern dentistry has evolved to a minimally invasive approach, in which caries is managed as an infectious disease, deferring operative intervention as long as possible⁹.

Under Standing Of Caries

The oral cavity houses more than 250 microbial species. The tooth morphology have many inaccessible areas to physiological clearance mechanism. Thus a tooth becomes an ideal place for stubborn adherence for many of these species. For many years, either all plaque flora were collectively considered as being pathogenic (nonspecific plaque hypothesis-

NSPH) or certain specific organisms were considered pathogenic (specific plaque hypothesis-SPH). Under NSPH, the target was to remove the entire plaque, but it was slowly realised that it was impossible to remove this natural accumulation of microbes on the tooth, even after brushing or professional cleaning. Under SPH, the target was to eliminate specific pathogen with antimicrobial treatment. This hypothesis was failed to substantiate the inability to detect specific organisms in the presence of disease¹⁰. In 1991, a new hypothesis was proposed called the ecological plaque hypothesis. According to this dental caries is a “complex disease caused by an imbalance in physiologic equilibrium between tooth mineral and bio film fluid”^{11,12}. Lesion develops where bio films are allowed to mature and remain for prolonged period of time. When the physiologic equilibrium between the tooth and bio film is disturbed, results in net loss of mineral. A frank cavity represents a site of ecological niche where the bio film composition adapts to decline PH environment. It facilitates further caries development.

Paradigm shift also considers, dental caries is “reversible multifactorial process”. This is based on factors such as bacteria, sugar, saliva, fluoride etc. In early stage dental caries is reversible. The development of lesion is dynamic process of demineralization of dental

hard tissues by acid products of bacterial metabolism alternating with period of remineralisation. The bacteria in the plaque intact tooth surface metabolize the available sugar and produce acid. This acid, which penetrates permeable tooth surfaces, drives calcium & phosphate out of the subsurface tissue, resulting in demineralization. This result in white spot lesion .If the tooth surface is intact, the reverse biochemical process can occur. Saliva buffers the low PH in plaque; calcium and phosphate are driven back into tooth there by remineralising lesion. This remineralisation is enhanced by application of fluorides, eliminating pathogenic bacteria, maintaining favourable oral environment¹³. Based on these paradigm shift dental caries is defined as “infectious, communicable disease resulting in destruction of tooth structure by acid forming bacteria found on dental plaque in intra oral biofilm in the presence of sugar”¹⁴.

Current concept of dental caries

The traditional model of caries is that decay is one-way process of acidic demineralization of susceptible tooth surface. The process is initiated by a combination of plaque and frequent consumption of refined carbohydrate.

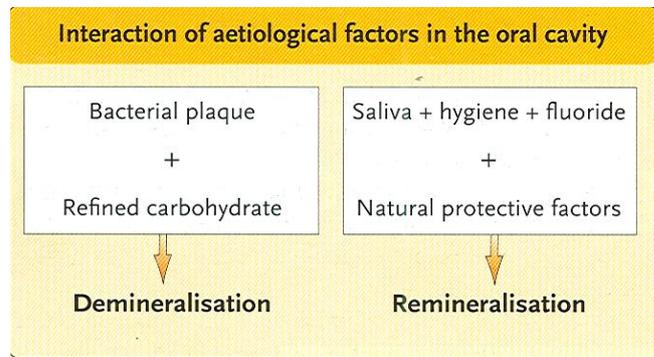
Current knowledge about decay requires us to rethink the traditional model and to realize that process is much more complex than the model suggests.

Ernest Newbrun stated that “caries is not simply a continuous and unidirectional process of the demineralization of the mineral phase but appear to be cyclic with periods of demineralization immediately following metabolism of fermentable substrate by plaque flora interspersed with period of remineralization”.

Modern concept suggests demineralization – remineralisation cycle of the chemical reaction that occurs on the tooth structure. Diet and plaque are considered to be major demineralization factors and fluoride and saliva the main factor facilitating protection and repair

This model allows the dental practitioner to understand the tooth environment as a homeostatic system, where factors promoting remineralisation should balance factors causing demineralization¹⁵.

To understand the mechanism of carious process it is necessary to understand the basic nature of chemical reaction that occurs at the tooth surface.



Demineralization:

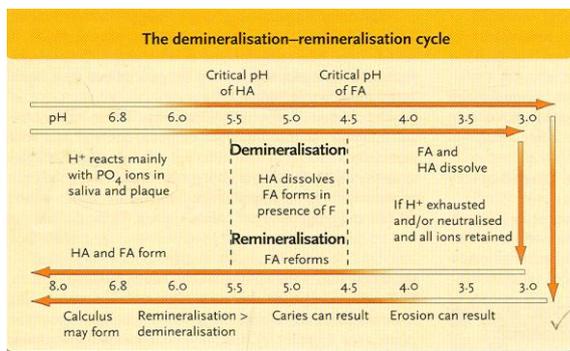
The mineral component of enamel, dentin & cementum is hydroxyapatite $\text{Ca}_{10}(\text{PO}_4)_6(\text{OH})_2$. In a neutral environment, hydroxyapatite is in equilibrium with the local aqueous environment, which is saturated with calcium (Ca^{+2}) & phosphate (PO_4^{3-}) ions. Hydroxyapatite is reactive to hydrogen (H^+) ion at PH 5.5 and below. H^+ reacts with phosphate group. It forms HPO_4^{2-} and H^+ being buffered at the same time. HPO_4^{2-} is then not able to contribute to the normal Hydroxyapatite equilibrium. Because it contains PO_4 and Hydroxyapatite crystals dissolves i.e. dissolution of Ca^{2+} and HPO_4^- from tooth surface to saliva. This is termed demineralization. This process is an active transport and energy is derived from hydrogen ion diffusion.

Remineralization:

The demineralization process can be reversed, if the PH is neutral and there are sufficient Ca^{2+} & Po_4^{3-} ions in the environment. Calcium and phosphate ion in saliva inhibit process of dissolution through the common ion effect. This

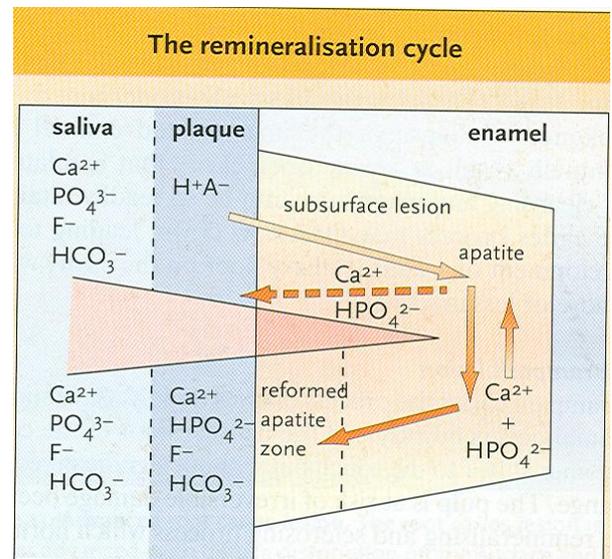
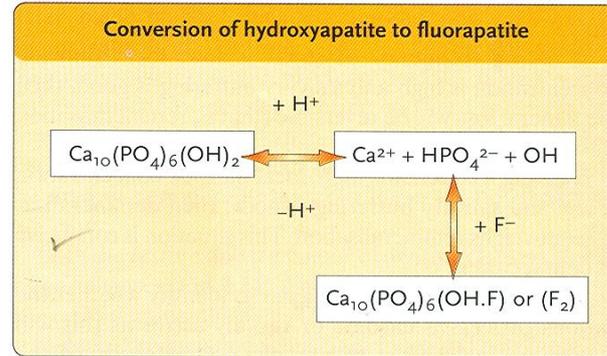
enables rebuilding of partly dissolved apatite crystal and it is termed remineralisation. This interaction can be greatly enhanced by the presence of fluoride ion at the reaction site. It results in formation of fluorapatite, which has critical PH 4.5. This means it resists acidic dissolution.

Demineralization - Remineralization cycle:



As the PH decreases, the acid ion reacts principally with phosphate in saliva and plaque until the critical PH for dissociation of hydroxyapatite is reached at approximate PH 5.5 –5.2. Further decrease in PH results in progressive interaction of the acids ion with phosphate group of hydroxyapatite, causing partial or full dissolution of the surface crystallites. Stored fluoride released in the process reacts with calcium & phosphate ion breakdown products forming fluorapatite or fluoride enriched apatite. If the PH decreases further below 4.5, which is the critical PH for fluorapatite dissolution, it will dissolve. If the acid ions are neutralized, the calcium &

phosphates ions are retained in this hypothetical model, the reverse process of remineralization are able to occur.



Main contributing factors to the de-remineralization balance are:

Destabilizing factors	Protective factors
Diet & plaque = plaque acids	Saliva
Reduction in salivary flow	Buffering capacity
Low buffering & oral clearance	Ca ²⁺ & PO ₄ ³⁻ levels
Acidic saliva	Buffering and

	remineralisation
Erosive acids	Oral clearance Proteins / glycoproteins
	Fluoride contact Developmental and topical application

. By maintaining favourable oral environment in oral cavity, the caries process can be reversed and stabilized through this concept [16].

Different concept of dental caries and their implications:

The concept of dental caries influences our strategies for control and treatment of disease. Key's model is known for its simplicity (Fig.1). This model is easy to appreciate and to explain to the public when arguing for the choice of preventive strategy.

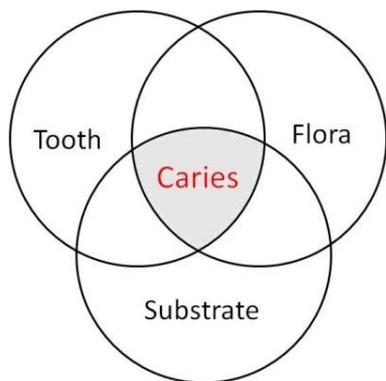


Fig:1: Key's model

It is evident that the mere existence of the three factors operating together does not result in instantaneous mineral loss and therefore a fourth circle is often added to stress the time dimension taken for dental caries to develop.(Fig.2)

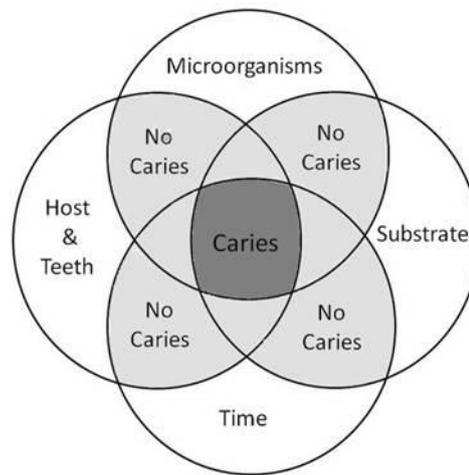


Fig: 2: Modification of Key's model

It is generally agreed that 'a clean tooth never decays' that is the existence of microbial deposits on the tooth surface plays a key role in development of caries. If it is thought that one particular microorganism is responsible for caries, it will be important to identify the microorganism in high number, and treatment strategies involves controlling caries by use of anti microbial and vaccination. In fact, all microorganism in the microbial deposits are belongs to the resident human microflora. Therefore, it is questioned whether it is biologically appropriate at all to interfere with the delicate oral eco system by using

antimicrobials, antibiotics etc in attempts to interfere with oral disease.

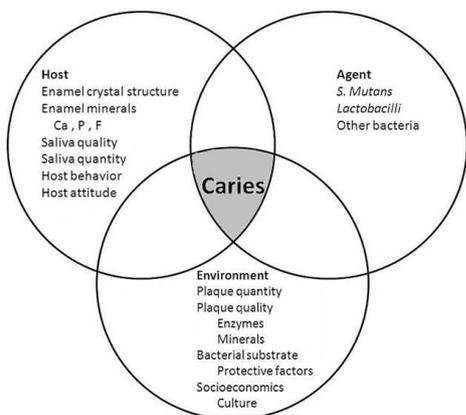


Fig: 3: Multifactorial model

Frequent exposure to carbohydrates was strongly associated with the high prevalence of decay in individual with limited oral hygiene. Change in diet, with introduction of refined flour and increased availability of sucrose leads to gradual increase in caries. Hence the concept “sugar is the arch criminal in dental caries” as proposed. Sucrose favours the growth of mutans streptococci and forms extracellular glucans, which is capable of attaching to the tooth surface, and makes it more pathogenic. Thus the concept presented in fig 2 & 3 led in many societies to the idea that the battle against sugar was the most important and if microorganism were to be attacked, it would be advantageous to go for mutan streptococci.

Clinical studies have shown that not all individual with poor oral hygiene & frequent

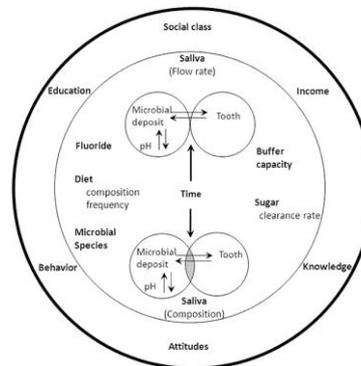


Fig. 4: illustrates the relationship between dental plaque and the multiple biological determinants, which influence the likelihood of caries lesion to develop

sugar consumption develop caries. This gives rise to the concept that enamel was considered as important factors. Fluoride concentration in water supplies or through saliva makes enamel resistant to decay.

In oral cavity a microbial deposit will cover tooth surfaces and plaque remains undisturbed over a period of time in the protected area of tooth, where caries develops. The microbial interaction on the tooth results in pH fluctuation due to release of acid. This in turn leads to loss and gain of minerals from the underlying dental hard tissues.

This metabolic process is strongly influenced by multitude of factors in oral cavity such as saliva, Ca^{+2} , PO_4^{+} , fluoride, buffer capacity, flow rate of saliva etc. This will strongly influence dynamic equilibrium between microbial deposit and the tooth surface.

Socio economic & behavioural factors in an outer periphery of the model indicated the confounding factors, which may not always be same in all societies.

Applying this model to understand dental caries, it is clear why individual with high caries experience may not share the same characteristics ¹⁷.

Conclusion

“Dental caries is as old as mankind.”

- Dental caries is a multifactorial process involving substrate, specific bacteria, host factors and time. One factor by itself will not initiate caries.
- The carious process may be thought of as a chemical reaction with a number of intermediate steps. It begins with the intake of cariogenic foods and may end with a cavitated lesion if allowed to proceed to its end.
- If the disease progression is prevented from producing a cavitated lesion in the tooth, this process may be reversible and curable.
- The plea of G. V Black made in 1909, to study and understand early carious lesions should receive our utmost attention. We need to shift our focus from defining dental caries as CAVITES

to detecting the stages of the caries process.

- The consequences of paradigms are to appreciate the risk of developing new lesion is never zero. Therefore dental caries can never be 100% preventable because of its complex nature.
- The caries-balance model is a useful adjunct for understanding the nature of the interactions among numerous protective factors and caries-risk factors. Other researchers have constructed interactive models that demonstrate the differential impact of various risk and protective factors within a dynamic, multifactorial framework. Caution should be used, however, in extrapolating the quantitative aspects of this model to populations beyond those on which the model is based.
- The emphasis should be focused on effective preventive practice and non-operative caries management should change the face of routine clinical practice. Perform minimal intervention surgical procedures as required. Repair, rather than replace, defective restorations. However, dental caries remains a significant problem for many human beings, and we look forward to

the day when people of all ages and backgrounds view dental caries as a disease of the past.

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How to cite this article: Hema B S, Goenka S, Verma P. Overview on concepts of dental caries. *JOADMS* 2015;1(2):61-69.
Source of Support: Nil, Conflict of Interest: None