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Review Article

Biological Features of Dental Caries

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Abstract

The purpose of this paper is to address some actual basic concepts about the etiology and aspect of dental caries; aiming the reasoning and understanding of several factors involved in the disease pathogenesis and provides its control in a simple and effective way. For so much, caries is mentioned as a localized disease, consequent of acid's action from the metabolism of anaerobic bacteria on retention sites of tooth surface. Thus, disorganization of biofilm, through efficient mechanical control of plaque (toothbrushing and mastication) constitutes the most important factor in disease's control.

ABBREVIATIONS

EDJ: Enamel Dentin Junction

INTRODUCTION

Data from the NIH Human Microbiome project (2009) shows that we are colonized by 10 trillion bacterial cells, as opposed to 1 trillion human cells that make up the body itself. Among this colonized body, the sites that harbor more bacterial cells comprehend gastrointestinal tract, oral cavity and skin [1]. Within this high abundance and diversity, these bacteria coexist in homeostasis and are necessary for our survival.

In relation to oral cavity, recent publications show that it harbors more than 700 different bacterial species, many of which unknown or traditionally cultivated by culture methods [2,3]. These bacterial species are indigenous on various oral surfaces, including tongue, buccal mucosa, gingiva, hard palate, supra- and sub-gingival dental biofilm [4] and can be found in different abundances, according to each oral site and even distinct surfaces on the same tooth [5]. This knowledge has been possible due to the advance use of techniques involving molecular biology to identify bacterial genera and species by sequencing the 16S rRNA gene, such as HOMINGS and the open-ended approaches 16S rRNA Illumina sequencing and 454 pyrosequencing [6].

With the advance of these new techniques, it is also possible to understand the spatial and sequence organization of oral biofilms. Initially teeth and oral mucosa are covered by a thin film of saliva, which is moving at different speeds, depending on the anatomical region. Indigenous microbiota needs a place to adhere and reproduce, and the retentive surface of choice is obviously the tooth surface. Thus, it initiates the adsorption of microorganisms that carry predominantly aerobic metabolism enamel. With proliferation of microorganisms, the formed biofilm becomes thicker, thus decreasing oxygen tension especially in the deeper layers, which favors the use of anaerobic route of metabolism, forming lactic acid, the principal substance involved in tooth decalcification. This is responsible for an ecologic shift to an imbalanced biofilm, towards disease development [6,7]. Recent data from Dige, Grønkjær and Nyvad (2014) showed, for the first time, the spatial distribution and functions of intact microbial communities on tooth surfaces. The composition of bacteria in the bottom of occlusal fissures seemed less diverse than the biofilm at the entrance of the fissures, with *Actinomyces* spp. And few colonies of *Streptococci, Fusobacterium* spp., *Veillonella* spp., *Lactobacillus* spp. and *Bifidobacterium* spp. was not frequent residents inside the shallow fissures. In active cavitated enamel lesions in occlusal surface, the structural composition of the biofilm was similar to that observed inside the shallow fissures. However, *Lactobacillus* spp. and *Bifidobacterium* spp. were prominent. In active cavitated lesions, *S. mutans* was observed, while *Bifidobacterium* spp. and *Lactobacillus* spp. were also seen in the outer layers of the biofilm at the entrance to the cavity. Only at advanced stages of the caries process, with cavity formation, bacterial invasion with penetration into the dentinal tubules could be observed. [8]

What is dental caries?

Dental caries is the most prevalent disease in the world, affecting children and adults, and representing a serious public health issue. Data from the US Department of Health and Human Services reports that caries in pre-school children remains a problem in both developed and developing countries, and it is the most prevalent chronic disease in children in USA [9], where more than 40 percent of children have caries by the time they reach kindergarten [9,10]. In contrast to declining prevalence of dental caries among children in older age groups, the prevalence of caries in poor USA children under the age of five is increasing. Among adults, USA reports a small decrease in coronal caries, with an incidence from 95% in 1988-1994 to 92% in 1998-2004, and the largest decline being seen in the 20- to 34-year age group [11].

Dental caries can be defined as a localized disease, resulting from localized bacterial activity that leads to an ecologic shift within the dental biofilm. This change of the biofilm's metabolic activity, from a balanced population of microorganisms to an acidogenic, aciduric and cariogenic microbiological population, is developed and maintained by lack of regular mechanical disruption of the biofilm and frequent consumption of fermentable dietary carbohydrates, which results is an imbalance between the dental mineral and fluid of the biofilm, between demineralization and remineralization, leading to net mineral loss within dental hard tissues [12].

The importance of the biofilm

It must be understood that not all biofilm is capable of developing disease. On the contrary, biofilm, when in homeostasis, is necessary to oral health maintenance. This non-cariogenic biofilm can be defined as plaque that suffers recurrent episodes of disorganization; therefore, aerobic metabolism is predominant, which does not have acid as the final metabolic production. Conversely, cariogenic biofilm can be defined as microbial accumulation that remains non-disorganized for long periods, leading to microbial succession and where anaerobic metabolism predominates, what culminates with acid production and pH decrease of the biofilm.

Classic references [13-15] reinforce the role of biofilm in caries etiology. Backer-Dirks showed that, in erupting permanent molars, carious lesions are always linked to areas without occlusion, where is difficult thus receiving a self-cleaning (during chewing) or even brushing, and with full eruption and the establishment of occlusion, most carious lesions were naturally arrested [14]. In the study of von der Fehr et al., the authors developed caries in students, in the presence or absence of sucrose, but with no brushing for 30 days. This showed the dental caries is not sucrose-dependent but hygiene-dependent. Sucrose, instead, has an important contribution, once it increases the speed of caries progression. [15]

Holmen et al., placed modified orthodontic bands (there was a space between the band and the tooth so that biofilm could accumulate) in homologues premolars that would be extracted for orthodontic reasons. One of the bands was weekly removed and the tooth was cleaned with a rubber cup or cotton swabs. Band was then re-cemented. After the fourth week the teeth were extracted and analyzed. Results indicated that the teeth on which the band was removed weekly and teeth's surface were cleaned, there was no caries progression. In contrast, among the bands that were not cleaned, surface's showed classic signs of demineralization. This study showed that etiology of dental caries is based on the biofilm accumulation and activity and thus, the more effective way to prevent it is cleaning the teeth. [16]

From the microbiological point of view, *Streptococcus mutans* and *Streptococcus sobrinus* are known as the most cariogenic species related to initial caries development [17-19]. Classic studies have shown that particular biochemical characteristics of the mutans streptococci, such as high acid production, high tolerance to environmental and intracellular acidification [20] and production of extracellular polysaccharides [21,22] are related to their cariogenic role. However, due to advances in molecular microbiology studies, recent studies involving "OMICs" technology have shown that other species may play an important role in the community leading to caries development. Aas et al., examined pooled biofilm samples and found other species significantly abundant in caries initiation, including *Actinomyces*spp, *Abiotrophia* spp, *Atopobium* spp, *Bifidobacterium* spp, *Lactobacillus* spp and *Veillonella*spp. *Veillonella* spp. was associated with caries and especially with total acid-producing bacteria, and the presence of *Veillonella* spp., but not of *Streptococcus mutans*, was predictive of caries risk [23]. Gross et al., showed that *Streptococcus mutans* was not statistically associated with the severity of caries, once it was often found in high numbers in the early stages of caries and in some healthy subjects; instead, *Propioni bacterium* spp was associated with caries progression [24]. Simon-Sóro et al., showed that bacterial species, other than *Streptococcus mutans* and *Streptococcus sobrinus*, may also play important roles in caries initiation and biofilm community interactions. This study again found *Streptococcus mutans* in high numbers in many subjects, but some subjects with caries had no *Streptococcus mutans* [25].

Some host factors will influence the bacterial community development and thus, may influence dental caries progression rate. First, the tooth structure, where some areas of the same tooth are prone to biofilm accumulation (pit and fissures, proximal surfaces, tooth irregularities, such as hypoplasias). Second, saliva quality and quantity and its protective role, such as salivary flow rate, buffering capacity to neutralize acids, antimicrobial activity, microorganism aggregation and clearance from the oral cavity, immune surveillance, fluoride delivery and calcium phosphate binding proteins all interact to inhibitor reverse demineralization of exposed tooth surfaces [26]. Normally, independently of those host factors, once the human microbial communities are established, it remains stable for months, and possibly years. However, common aspects of the lifestyle, including antibiotics, high-fat diets, alterations in saliva and even actions and experiences can persistently alter commensal microbial communities [27].

Clinically, since it is not worth to conduct microbiological identification and profiling to every single patient dentists should make all efforts to identify areas of biofilm stagnation, prior to follow lesions assessment. Identifying the quality and distribution of the biofilm will help the clinician to diagnose initial lesions and also monitor patient's compliance in follow-up appointments. Ribeiro proposed a clinical index to obtain data about the extent and quality of visible dental biofilm [28]. Figure (1) shows a flowchart of clinical proceedings to address the index score. According to it, dental biofilm was classified as thin when only seen after the dental surface had been dried with a gauze, without scratching it; it was recorded as thick when it could be seen clinically without the use of gauze; and it was considered to be firmly attached when the thick layer of bacterial accumulation was resistant to complete removal with a gauze. This index has shown to be useful to access patient's quality of tooth cleaning

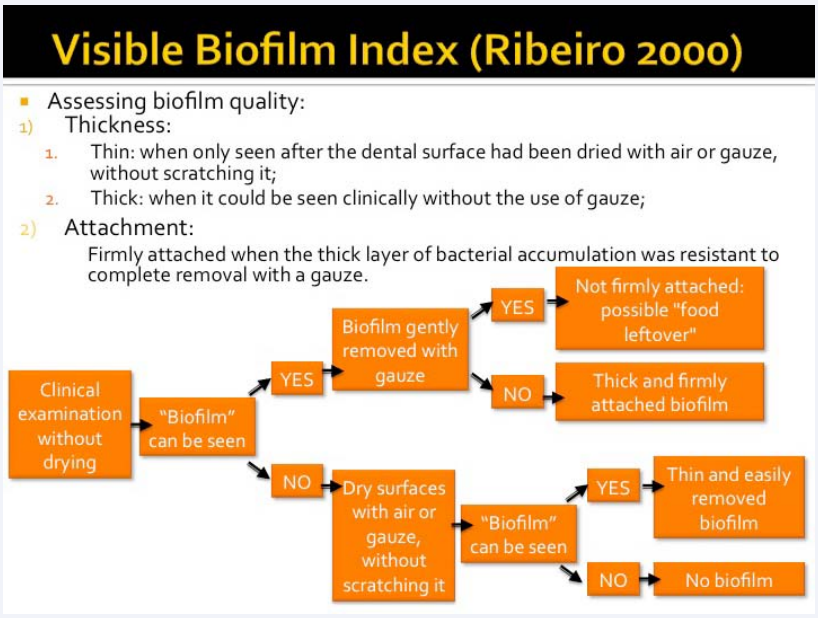

Figure 1 The Visible Biofilm Index criteria for clinical assessment of the quality of dental biofilm (Ribeiro, 2000).

and compliance, and demonstrated good correlation with presence of gingivitis and caries activity [28-31].

Dental caries diagnosis: basis on biological features of the disease

Once the professional identifies the local areas with cariogenic biofilm, professional cleaning should be performed to remove it so surfaces can be classified according to clinical appearance, which is related to pathogenesis of the disease in enamel and dentin, and in smooth of pit and fissures surfaces.

DENTAL CARIES IN PROXIMAL SURFACES

The relationship between gingivitis and caries in proximal surface is narrow [31]. The anatomy of the gingival papillae in proximal surface takes up all the interdental extension except the contact point. However, local gingival changes in this area will let to a protected surface for biofilm accumulation [12]. Thus, through clinical examination (mainly at the lingual surface of the teeth, where there is a greater extent visible papillae), clinicians should look for gingival changes and suspicious of problems in hard tissues in areas between the contact point and gingival margin (Figure 2).

Ribeiro et al., investigated the performance of methods for detecting proximal caries lesions in primary molars. A total 209 approximal surfaces were examined by visual-tactile examination using the Nyvad criteria, DIAGNOdent pen 2190 bitewing radiographs. After tooth exfoliation, surfaces were directly examined by computed microtomography as a reference standard. This study showed that the diagnosis of proximal surfaces can be influenced by the contact point, although visual-tactile examination, using the Nyvad criteria, showed better results in detecting approximal caries lesions in primary molars than DIAGNOdent pen and bitewing radiographs. The authors suggested the placement of orthodontic separating elastics on the contact points can be useful for caries diagnosis in proximal surfaces [32].

DENTAL CARIES IN FREE SMOOTH SURFACES

Once caries lesions are dependent of biofilm accumulation, initial caries lesions will be localized in areas of the smooth surfaces that are protected from physiological cleaning, which are located close to gingival margin and follow its shape. It means that initial caries in enamel will have a “banana shape”.

Active incipient caries (active white spot lesions) are characterized by an enamel surface with a whitish/yellowish opaque lesion with loss of luster. Clinician will feel rough when the tip of the probe is moved gently across the surface, and the area generally was covered with biofilm. Inactive caries with intact enamel smooth surfaces are characterized by whitish, shiny, hard and smooth enamel, when the tip of the probe is moved gently across the surface. No clinically detectable loss of substance can be observed, and this lesion typically is located at some distance from gingival margin (Figure 3) [33].

Also, the straight relation between biofilm accumulation and

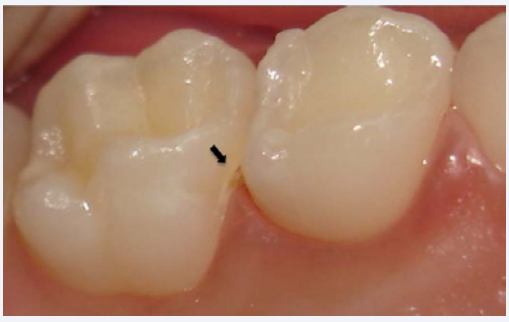

Figure 2 Incipient caries lesion in proximal surface (arrow).

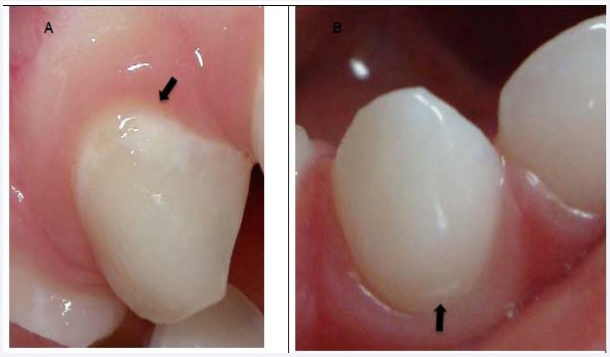

Figure 3 (A) Active white spot lesion in smooth surface (arrows). (B) Inactive (arrested) white spot lesion in smooth surface (arrows).

gingivitis can represent a useful parameter during the clinical examination [31]. It is not difficult to have a patient that brushes his teeth vigorously because he knows he has an appointment with the doctor, so biofilm accumulation is not always evident. But the signs of its stagnation can be verified by presence of provoked gingival bleeding, helping with the final diagnosis of the smooth surface.

DENTAL CARIES IN OCCLUSAL SURFACES

Traditionally, the diagnosis of occlusal surfaces has been based in probing, despite the evidence-based dentistry has already showed the damage caused by probing: often pits are broken, leading to a greater biofilm accumulation and lesion progression. In the *in vitro* study from van Dorp, Exterkate, ten Cate, they evaluated the effect of the use of the explorer on occlusal surfaces with demineralized enamel areas. After a week, they showed that the lesions increased vertically and horizontally, some reaching the enamel-dentin junction (EDJ). None of the demineralized surfaces that were not probed reached the EDJ. After two weeks, all probed lesions reached dentin, while not probed surfaces didn't reach the EDJ [34]. Yassin (1995) showed, *in vitro*, the mechanical damage of early carious lesion in artificial U-shaped grooves caused by a sharp dental explorer. Surface layer in demineralized enamel grooves were broken down after being assessed by explorers, converting the white spot lesion with apparently a sound surface layer into a cavity. [35]

When assessing occlusal surfaces, the clinician must remember that pit and fissure areas are protected from physiological cleaning and, thus, favor biofilm accumulation. So, initial caries lesions will be localized at pit and fissures (Figure 4). The assessment of the natural history of decay on the occlusal surface cannot be made without remembering the physiology of the eruption of these teeth. The research from Carvalho et al., showed erupting teeth accumulate more plaque than fully erupted teeth, and thus teeth at this stage have a greater risk of developing caries. [36]

Active incipient caries in occlusal surfaces will appear as whitish lesions with intact fissure morphology. The lesion will be extending along the walls of the fissure. In inactive incipient caries, generally a brownish or black fissure with an intact morphology will be observed, and the lesion extending along the walls of the fissure [33].

DISEASE TREATMENT AND CONTROL

Given the biological and pathologic basis for dental caries as a localized, biofilm-dependent disease, both prevention of disease development and management of existing lesions should focus primarily on control or management of the biofilm. Once caries lesions have already been developed, lesion stages and activity might be assessed before planning treatment, once they require different management. As stated by Schwendicke et al., caries lesions management should aim: (1) inactivation/control of the disease process, (2) preservation of dental hard tissue, (3) avoidance of initiating the cycle of restoration, and (4) preservation of the tooth for as long as possible [37].

For disease control, it is imperative to share responsibilities with the patient. Improve daily biofilm removal by adequate brushing technique, use of fluoridated toothpaste and adjustment to a healthy diet are home-based tools for caries control. From a biological point of view, even after a carefully performed toothbrush by the patient, some residual plaque capable of a moderate pH drop can be found in tooth fissures and/or irregularities and proximal surfaces [38]. However, from a clinical point of view, a regular mechanical removal of the biofilm with fluoridated toothpaste favors biofilm disruption and maturation and controls caries progression in enamel and dentin [13,15,37] Management of initial, non-cavitated, active lesions in enamel should be towards its inactivation, by mechanical biofilm control (toothbrushing and flossing) with fluoridated toothpaste (high frequency of low concentration of fluoride). Professional use of topical fluoride should also be indicated (low frequency of high concentration of fluoride), remineralization or by sealing [37]. In occlusal lesions, this will be through placement of fissure sealants [39,40], while on proximal or smooth surfaces with pits, this will involve other methods of sealing or lesion infiltration with resin [41]. However, once biofilm accumulation is the main etiologic factor of the disease, it is imperative to achieve a good mechanical cleaning of the lesion, otherwise it won't be controlled.

Enamel cavitated lesions that are potentially cleansable lesions (i.e., assessed as being cleansable by the patient) can be inactivated and not require further treatment, as their

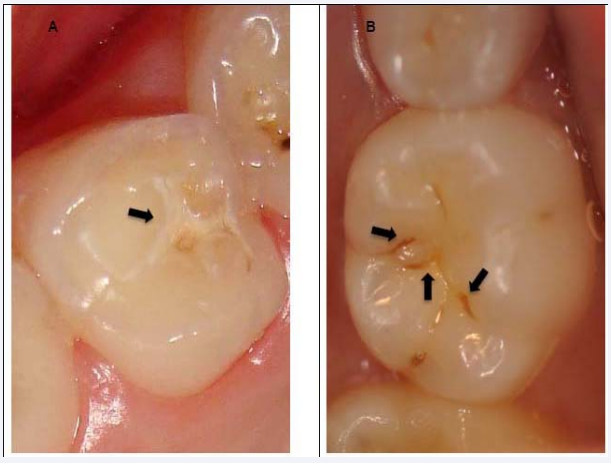

Figure 4 (A) Active white spot lesion in occlusal surface (arrow). (B) Inactive (arrested) lesion in occlusal surface (arrows).

progression is unlikely and can be managed non restoratively (noninvasively; i.e., via biofilm removal and remineralization). On the other hand, lesions that are not cleansable are likely to be active and progress [37]. Sealing over them and depriving the bacteria within the lesion of carbohydrates might arrest them [39].

Dentin lesions are a result of progress following cavitation of enamel lesions, due to continuously accumulation of biofilm. If the lesion is either accessible or opened for cleaning by the patient or parent, the lesion can be arrested [42]. However, most of these lesions are closed, favoring food stagnation and biofilm accumulation. These cavities should then be sealed, without the need of complete removal of the infected dentin. Sealing of infected dentine by selective caries removal prior to placing a well-sealed filling will arrest the lesion. In deep carious lesions in symptomless, vital teeth, vigorous excavation of infected (soft) dentine increases the risk of pulp exposure and makes root canal treatment necessary. Thus, complete excavation is considered overtreatment and should be avoided. [37,42]

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