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Introductory Chapter: Dental Biofilms Associated with Caries

Emilija Stefanovska and Efka Zabokova Bilbilova

1. Introduction

Caries is a localized, progressive, destructive process in which the mineral component of dental hard tissues dissolves. It is the result of multiple disturbances of the balance between the oral environment and the teeth. Due to this disturbed balance, microbial acid production occurs and the pH value decreases.

Dental caries is still the most common chronic infectious disease, especially in endangered and poor populations [1, 2], but not so prevalent in the industrialized world and developing countries. Dental caries is a disease associated with biofilm. Plaque control is an important caries prevention strategy, because biofilm bacteria are the driving force of demineralization and caries development [3, 4].

The course of dental caries development depends on several host factors, including location, morphology, composition, ultrastructure, and age of the tooth after eruption [5]. The very environmental conditions that exist at each site of the tooth explain the highly localized and complex nature of the caries process. The occlusal pits and cracks of the molars with their morphological characteristics create a retention area for the formation of biofilm and food retention, making them more prone to caries forming on the tooth surface in children.

The process of dental caries begins within the bacterial biofilm that covers the surface of the tooth. Numerous episodes of mineral loss and gain (demineralization and remineralization) occur on the enamel surface. If demineralization prevails over remineralization, the result will be a permanent and irreversible mineral loss, void formation, and continuous destruction of hard tissues [6, 7]. Signs and symptoms of the disease range from the slightest underground loss of minerals to severe tooth destruction.

Clinical causes of caries include:

1. the presence of plaque containing acid-producing bacteria,
2. consuming carbohydrates that are easily fermented on a frequent basis (for example, sugar),
3. low saliva production or reduced saliva capacity to act as a buffer and.
4. genetic factors which make the host more susceptible to caries [8].

2. Components of dental plaques

The oral microbiome, the unique natural ecology of the mouth is an ecosystem or community of microorganisms that live in symbiosis with one another and with

their host, which contribute positively to the individual's health. It is composed of many different bacteria, neutrophils leukocytes, macrophages, monocytes and lymphocytes. The largest portion of dental plaque is liquid and consists of water while 70% of the dry weight is made up of bacteria and the rest is polysaccharide and glycoprotein matrix adherent to the tooth surface [9].

Polysaccharide and glycoprotein matrix mainly consist of 10–20% water-insoluble glucan, 1–2% fructan, approximately 40% bacterial and salivary proteins, variable quantities of lipid, calcium, phosphorus, magnesium and fluoride – the rest is water. The remaining microorganisms are later colonizers that join in the dental biofilm and with their metabolic activities have a competitive advantage in the community [10].

The dental bio-plaque formation is a complex structure, with a highly organized setting of functional symbiotic interactions between microorganisms [11].

The maintenance of health is closely dependent on the balance between the microorganisms that practice commensalism or mutualism between themselves and in relation to the host.

In addition to these interactions, under the influence of certain circumstances, internal or external, this homeostasis may be disturbed, resulting in changes in the microbial population and the induction of certain pathological conditions. An elevation or a depression in the relative presence of certain microorganisms often leads to disruption of homeostasis [12].

A common feature that can disrupt the microbial ecosystem is the import of nutrients, such as sugars. Carbohydrates induce the reproduction of microorganisms that ferment sugars, such as *Streptococcus mutans* and *Lactobacilli* species. Through metabolizing these carbohydrates, the bacteria generate large amounts of lactic acid, resulting in the selection and dominance of acidogenic bacteria.

The acidogenic oral bacteria present in the dental biofilm, have the ability to generate acids, which are responsible for tooth structure demineralization. The biofilm, related to dental caries, functions in several ways. It is a result of bacterial proliferation and growth, acid/ base regulation at the tooth surface, and a calcium ion exchange between the tooth and the saliva [13].

Many studies have confirmed the role of *Streptococcus mutans* and some anaerobic bacteria like *Fusobacterium* and *Actinobacillus* as a primary colonizer in the initial attachment on the tooth surface, responsible for the formation of the premature biofilm community [14].

The composition of bacterial species like *Streptococcus mutans* and some anaerobes like *Fusobacterium* and *Actinobacteria* varies in the different areas in the mouth. In accordance with the condition and localization of biofilm in the oral cavity there are subclasses of biofilm, sub gingival and supra gingival dental biofilm. Supra gingival plaque usually comprises of aerobic bacteria, thus it is filled with oxygen. On the other hand, sub gingival plaque found in interdental spaces which are devoid of oxygen, consists of anaerobic bacteria.

Changing the conditions in the environment, pH levels, salivary flow, temperature, oxidation–reduction reactions, the growth and development of microorganisms also changes [15]. The normal pH value in the oral cavity varies from 6 to 7. Small deviations from these salivary pH values are a gateway for the multiplication and growth of pathogenic microorganisms, thereby changing the dental plaque formation. The various nutrients in saliva such as proteins and amino acids are an excellent breeding ground for bacterial growth [16]. Deviations from the normal temperature in the oral cavity ranging between 35 and 36 degrees also alter the chemical processes that affect bacterial metabolism. Such a persistent acidic environment within the biofilm results in the demineralization of tooth enamel [17].

However, it should be pointed that streptococcal mutans that produce glucans and biofilm are the primary and major etiological factors in the pathogenesis of dental caries [18].

3. The role of sucrose in biofilm cariogenicity

Numerous epidemiological and experimental studies confirm the causal relationship between sucrose and dental caries [19–22].

Sucrose causes an increased level of *Streptococcus mutans* and *Lactobacilli* and at the same time, a decrease in *S. sanguinis* levels thus leading to biochemical and physiological changes in the dental biofilm formation and enhancing their caries-inducing properties [23]. The cariogenic effect of sucrose is closely dependent on its concentration and duration of exposure [24, 25].

Increasing the frequency of carbohydrates results in plaque accumulation whose persistence below critical pH values will cause demineralization of the enamel [26]. Compared to glucose and fructose, sucrose has the dominant cariogenic potential [27]. As a substrate for the production of extracellular glucan by *Streptococcus mutans* glycosyltransferases, sucrose takes precedence as a unique cariogenic carbohydrate [28].

4. Dental plaque, a trigger factor in the development of caries and periodontal disease

Scientific research cannot definitively define the concept of a basic, normal oral microbiome that could be associated with a healthy oral cavity for a number of reasons. First of all, because individual dental biofilms are unique, with varying susceptibility to disease. Then with the passage of time and the aging process, the microbial community changes as a result of the evolutionary processes, the ecological environment and the immune response of the individual. However, Gram-positive microorganisms belonging to the streptococcus and actinomyces species are predominantly present in the oral microbiome with certain deviations.

When the transformation of the healthy microbiological community begins and caries occurs, changes in the oral microbiological environment happen, along with changes in the development of the disease [29].

The acidogenic properties of cariogenic microorganisms and the ability to form extracellular polysaccharides from sucrose maintain the acidity of the environment, thus reducing the diversity of microbial flora.

The acidogenic capacity of these microorganisms is the mechanism that is scientifically widely accepted in the process of demineralization of dentinal and enamel tooth structures, causing the development of carious lesions [30].

Dysbiosis in periodontal diseases is also associated with an increase in microbial diversity. The main pathogens in periodontal diseases consist of dominantly gram-negative microorganisms including *Porphyromonas gingivalis*, *Treponema denticola*, *Tannerella forsythia* and *Aggregatibacter actinomycetemcomitans* [31].

The periodontal microbial community, as dominant in anaerobic conditions, is capable of disrupting the host's inflammatory response by causing nutritional imbalance and impaired availability of potential gingival fluid and blood substrates. The microbial communities in periodontal disease and caries are in symbiosis with the host. How this balance is maintained depends on the nutritional interdependence of the bacteria as well as the salivary glycoproteins which are a constant nutrient source of dental biofilms [29].

The scientific advances in molecular biology have shed light on the bacterial communities present in the oral cavity, providing evidence that the prevalence of oral infections is due to impaired host homeostasis [32–34].

5. Dental indices

Dental indices are a numerical expression for assessing the prevalence and incidence of certain dental conditions, and for determining the need for treatment as well. They can be simple and cumulative. The simple indices determine only the presence or absence of a certain state, while the cumulative ones determine the current state, but also previous, past states. There are also so-called irreversible indices, which determine immutable conditions, such as caries. Reversible indices, in turn, note changing conditions, such as the amount of dental plaque accumulation.

5.1 Oral hygiene index (OHI)

Proposed and developed by John C. Greene and Jack R. Vermillion (1960), [35]. This index is composed of Debris and Calculus Index. Rules for scoring are: 1) Only permanent teeth are scored, 2) Third molars or incompletely erupted teeth are not scored, 3) The buccal and lingual debris scores are both taken on the tooth in a segment having the largest surface area covered by debris. 4) Interpretation: The minimum number of points for all segments in either debris or calculus score is 0. The maximum number of points for all segments in either debris or calculus score is 36. The higher the score, the poorer the oral hygiene.

5.2 Simplified oral hygiene index (OHI-S)

This index is a modification of the original OHI index, also developed by John C. Green and Jack R. Vermillion (1964), [35], in order to reduce the number of decisions made by researchers, as well as to shorten the time of inspection. Unlike the original OHI index, rules for scoring are at least two of the six possible tooth surfaces which must be examined. The third molars are involved only as long as they are functional. Natural teeth with crown restorations and tooth surfaces reduced in height by caries or trauma are not recorded.

5.3 Plaque index (PI)

The PI as developed by Silness and Loe [36], assesses the thickness of plaque at the cervical margin of the tooth (closest to the gum). Four areas, distal, facial or buccal, mesial, and lingual, are examined. Each tooth surface should be well dried beforehand, and the examinations are performed with a dental mirror, an explorer and a well-light probe. The probe is passed around the cervical third of the tooth to detect the presence of dental plaque. A disclosing agent can also be used to visualize dental plaque. The results are expressed in four values. A zero indicates no plaque present; 1 indicates a film of plaque present on the tooth; 2 represents moderate accumulation of soft deposits in the gingival pocket or on the tooth that can be seen by the naked eye; 3 represents an abundance of soft matter within the pocket or on the tooth. Each area of each tooth is assigned a score from 0 to 3. Scores for each tooth are totaled and divided by the four surfaces scored. To determine a total PI for an individual, the scores for each tooth are totaled and divided by the number of teeth examined. Four ratings may then be assigned: 0 = excellent, 0.1–0.9 = good, 1.0–1.9 = fair, 2.0–3.0 = poor.

5.4 Gingival index (GI)

Also attributed to Loe and Silness [37], the GI assesses the severity of gingivitis based on color, consistency, and bleeding on probing. Each tooth is examined at the mesial, lingual, distal, and facial (or buccal) surface. A probe is used to press on the gingiva to determine its degree of firmness, and to run along the soft tissue wall adjacent to the entrance to the gingival sulcus. Four criteria are possible: 0, normal gingiva; 1, mild inflammation but no bleeding on probing; 2, moderate inflammation and bleeding on probing; 3, severe inflammation and ulceration, with a tendency for spontaneous bleeding. Each surface is given a score, then the scores are totaled and divided by four. That number is divided by the number of teeth examined to determine the GI. Ratings are 0 = excellent; 0.1–1.0 = good; 1.1–2.0 = fair; 2.1–3.0 = poor.

6. Conclusion


Dental biofilm, as a proven complex biostructure of microorganisms is the primary etiological factor for the development of the two most diseases in the oral cavity - caries and periodontal diseases. Despite the fact that it cannot be completely eradicated, it can be controlled. So, the risk assessment for its occurrence is essential for the preservation of oral health. An individual adaptive anti-plaque program should be applied to each patient with a recommendation for appropriate home oral hygiene, practicing daily tooth brushing with a fluoride tooth paste, interdental cleaning and a hygienic-diet regimen with reduced carbohydrate intake. By doing this, there is an optimistic possibility that these cost-effective preventive strategies may minimize the incidence of caries and periodontal diseases and their impact on specific systemic conditions.

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References

- [1] Dantas Cabral de Melo MM, de Souza WV, Tavares MC, de Lima ML, Jamelli S, Couto GB. Social conditions and high levels of dental caries in five-year-old children in Brazil. *J Dent Child (Chic)* 2015; 82:29-35.
- [2] Sinha N, Singh B, Chhabra KG, Patil Comparison of oral health status between children with cerebral palsy and normal children in India: a case-control study. *J Indian Soc Periodontol* 2015;19: 78-82.
- [3] Takahashi N, Nyvad B. Caries ecology revisited: microbial dynamics and the caries process. *Caries Res* 2008 42, 409-418.
- [4] Ccahuana-Vásquez RA, Cury JA. *S. mutans* biofilm model to evaluate antimicrobial substances and enamel demineralization. *Braz Oral Res* 2010; 24: 135-141.
- [5] Guido JA, Martinez Mier EA, Soto A, Eggertsson H, Sanders BJ, Jones JE, Weddell JA, Villanueva Cruz I, de la Concha A, Luis J. Caries prevalence and its association with brushing habits, water availability, and the intake of sugared beverages. *International Journal of Paediatric Dentistry* 2011;21(6):432-440.
- [6] Caufield PW, Li Y, Dasanayake A. Dental caries: an infectious and transmissible disease. *Compend Contin Educ Dent* 2005; 26 (5 Suppl 1):10-6.
- [7] Takahashi N, Nyvad B. The role of bacteria in the caries process: ecological perspectives. *J Dent Res* 2011; 90: 294-303.
- [8] Kutsch VK. Dental caries: an updated medical model of risk assessment. *J Prosthet Dent* 2014; 111: 2805.
- [9] Marsh PD, Moter A, Devine DA. Dental plaque biofilms: communities, conflict and control. *Periodontol* 2000 2011; 55:16-35.
- [10] Bowen WH, Koo H. Biology of *Streptococcus mutans*-derived glucosyltransferases: role in extracellular matrix formation of cariogenic biofilms. *Caries Res* 2011; 45:69-86.
- [11] Marsh PD, Bradshaw DJ. "Dental plaque as a biofilm". *J Ind Microbiol* 1995; 15(3): 169-175.
- [12] Kolenbrander PE. "Oral Microbial Communities: Biofilms, Interactions, and Genetic Systems". *Annu Rev Microbiol* 2000; 54(1):413-437.
- [13] Bradshaw DJ, McKee AS, Marsh PD. Effects of carbohydrate pulses and pH on population shifts within oral microbial communities in vitro. *J Dent Res* 1989; 68(9):1298-302.13.
- [14] Chetrus V, Ion IR. "Dental Plaque - Classification, Formation, and Identification." *Int J Med Dentistry* 2013; 3(2):139-143.
- [15] Mc Dermid AS, McKee AS, Marsh PD. "Effect of Environmental pH on Enzyme Activity and Growth of *Bacteroides gingivalis* W50". *Infect Immun* 1998; 56(5): 1096-100.
- [16] Humphrey SP, Williamson RT. A review of saliva: normal composition, flow, and function *J Prosthet Dent* 2011 85(2): 162-169.
- [17] Marsh PD, Devine DA (2011) "How is the development of dental biofilms influenced by the host?" *J Clin Periodontol* 38 (s11): 28-35.
- [18] Zhou M, Zhu F, Dong S, Pritchard D, Wu H. A novel glucosyltransferase is required for glycosylation of a serine-rich adhesin and biofilm formation by *Streptococcus parasanguinis*. *J Biol Chem* 2010; 285:12140-8.

- [19] Cury JA, Rebelo MA, Del Bel Cury AA, Derbyshire MT, Tabchoury CP. Biochemical composition and cariogenicity of dental plaque formed in the presence of sucrose or glucose and fructose. *Caries Res* 2000; 34:491-497.
- [20] Cury JA, Francisco SB, Del Bel Cury AA, Tabchoury CP. In situ study of sucrose exposure, mutans streptococci in dental plaque and dental caries. *Braz Dent J* 2001; 12:101-104.
- [21] Cury JA, Marques AS, Tabchoury CP, Del Bel Cury AA. Composition of dental plaque formed in the presence of sucrose and after its interruption. *Braz Dent J* 2003; 14:147-152.
- [22] Zero DT. Sugars-the arch criminal? *Caries Res* 2004; 38:277-285.
- [23] Minah GE, Lovekin GB, Finney JP. Sucrose-induced ecological response of experimental dental plaques from caries-free and caries-susceptible human volunteers. *Infect Immun* 1981; 34:662-675.
- [24] Aires CP, Tabchoury CP, Del Bel Cury AA, Koo H, Cury JA. Effect of sucrose concentration on dental biofilm formed in situ and on enamel demineralization. *Caries Res* 2006; 40:28-32.
- [25] Paes Leme AF, Dalcico R, Tabchoury CP, Del Bel Cury AA, Rosalen PL, Cury JA. In situ effect of frequent sucrose exposure on enamel demineralization and on plaque composition after APF application and F dentifrice use. *J Dent Res* 2004; 83:71-75.
- [26] Marsh PD. Sugar, fluoride, pH and microbial homeostasis in dental plaque. *Proc Finn Dent Soc* 1991; 87:515-525.
- [27] Cury JA, Rebelo MA, Del Bel Cury AA, Derbyshire MT, Tabchoury CP. Biochemical composition and cariogenicity of dental plaque formed in the presence of sucrose or glucose and fructose. *Caries Res* 2000; 34:491-497.
- [28] Bowen WH. Do we need to be concerned about dental caries in the coming millennium? *Crit Rev Oral Biol Med* 2002; 13:126-131.
- [29] Mira A, Simon-Soro A & Curtis MA. Role of microbial communities in the pathogenesis of periodontitis and caries. *Journal of Clinical Periodontology* 2017; 44: S18, 23-38.
- [30] Simón-Soro A & Mira A. Solving the etiology of dental caries. *Trends in Microbiology* 2015; 23:76-82.
- [31] Camelo-Castillo AJ, Mira A, Pico A, Nibali L, Henderson B, Donos N & Tomás I. Subgingival microbiota in health compared to periodontitis and the influence of smoking. *Frontiers in Microbiology* 2015; 24:119.
- [32] Darveau RP. Periodontitis: a polymicrobial disruption of host homeostasis. *Nat Rev Microbiol* 2010; 8(7):481-490.
- [33] Kuramitsu HK, He X, Lux R, et al. Interspecies interactions within oral microbial communities. *Microbiol Mol Biol Rev* 2007; 71(4):653-670.
- [34] Von Fraunhofer JA, Loewy ZG (2009) Factors involved in microbial colonization of oral prostheses. *Gen Dent* 57(2): 136-143.
- [35] Greene JC. and Vermillion JR. The Simplified Oral Hygiene Index. *The Journal of the American Dental Association* 1964; 68:7-13.
- [36] Silness P, Løe H. Periodontal disease in pregnancy II. *Acta Odontol Scand*. 1964; 22:121-6.
- [37] Løe H, Silness P. Periodontal disease in pregnancy I. *Acta Odontol Scand* 1963; 21:533-51.