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Microbial Symbiosis and Dysbiosis-an Overview of Dental Plaque

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Abstract

We clinicians assume that the clinical picture of dental disease is a net result of an interaction between the pathogenic dental plaque and host tissue response. Dental plaque biofilm cannot be eliminated. However, the pathogenic nature of the dental plaque biofilm can be reduced by reducing the bio-burden (total microbial load and different pathogenic isolates within that dental plaque biofilm) and maintaining a normal flora with appropriate oral hygiene methods that include daily brushing, flossing and rinsing with antimicrobial mouth rinse. This review is about the dental plaque and the association of microflora its symbiosis and dysbiosis in the oral niche.

Keywords: Symbiosis; Dysbiosis; Plaque; Microorganisms; Biofilm

Introduction

The oral cavity is a portal for entry of microorganism which alters the immunity of an individual. Oral bacteria are commensals, which become pathogenic in adverse conditions and cause infections [1]. Dental plaque is a structurally and functionally organised biofilm of diverse micro biota [2]. It is a matrix of polymers of bacteria and salivary origin which forms naturally on tooth and defends host by prevention of colonisation of exogenous pathogens [3]. Plaque is natural and contributes to the normal development of the physiology and defences [4].

Definition

A specific but highly variable structural entity resulting from colonisation of microorganisms on the tooth, restorations or other parts of the oral cavity composed of mucin, microorganisms, desquamated epithelial cells and debris all embedded in a gelatinous extracellular matrix.

Classification

Dental Plaque is classified into two categories [5].

- Supra-Gingival Plaque
- Sub-Gingival Plaque

Supra-gingival plaque: Found at and above the dentino-gingival junction of the crown of the tooth is commonly found at:

-Gingival third of the tooth

-Inter-proximal areas

-Pits and fissures and also on other such surface with irregularities.

Supra-gingival plaque also called as marginal plaque may lead to gingivitis.

Sub-gingival plaque: Below the dentino-gingival junction, is usually divided into:

-Tooth adherent zone

-Epithelial adherent zone

-Non adherent zone

Sub gingival plaque is further subdivided into tooth associated and tissue associated sub gingival plaque. Supragingival plaque and tooth associated sub gingival plaque may lead to Calculus formation & root caries. Sub gingival plaque (epithelium) may lead to tissue destruction – Periodontitis

Structure of dental plaque

The structure of dental plaque is compared to primitive circulatory system. Bacteria exist and proliferate within intercellular matrix. Matrix act as a barrier, bacterial substances produced are retained and concentrated – Metabolic interaction between different bacteria. The non-mineralised microbial accumulation of dental plaque adheres tenaciously onto the tooth surface [6]. It shows structural predominance of filamentous form composed of organic matrix derived from salivary glycoprotein. The dental plaque predominantly consists of bacteria, which are organised and interrelated. The origin of the dental plaque, its development and adaptation to changing the oral condition [7].

Development of micro biota of dental plaque

Earliest micro biota to colonize in the mouth of a new-born is of the mother's genitalia. Due to the absence of teeth, the microorganisms colonise and adhere to the available surfaces lined with epithelium [8]. *Streptococcous salivarious*is established within one day of birth. *Lactobacillus* is detected in the oral cavity at the time of birth which doesn't reappear until after the age of 2.*Streptococcus sanguis* preferentially colonise on the tooth surface. *Streptococcus mutans* also colonises on the tooth surface, it was found to be absent in the oral cavity of 91 predentate infants but 9 of 40 infants with only erupted primary incisors [9]. *Actinomyces naeslandii* predominant species of dental plaque *Actinomyces viscous* and *Veillonella* sp. gradually increase with age [10]. The most predominant species are gram positive, facultative

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microbiota is found after the emergence, with loss of tooth in old age, some ecological niches such as tooth surfaces and gingival sulcus that favours retention of certain species [6].

Steps in plaque formation

A certain sequence of events is observed in the formation of dental plaque biofilm [3, 5, 11, 12].

Phase 1: Acquired pellicle formation and Transport to the surface

Phase 2: Initial adhesion

Phase 3: Attachment

Phase 4: Colonization and plaque maturation

Acquired pellicle formation and transport to the surface: In the first phase, acquired pellicle formation is seen when bacterial and host products present in the saliva and gingival crevicular fluid come in contact with the tooth surface. In supra-gingival areas, this layer is covered with molecules like salivary glycoproteins, histatin, proline-rich proteins and alpha-amylase. Glucosyl transferases and glycan are also found in the acquired pellicle. Bacteria are transported to the surface of the surface by Random contact– Brownian movement, Sedimentation or active bacterial movement.

Initial adhesion: Some gram positive streptococci like *Streptococcus sanguinis*, *Streptococcus oralis*, *Streptococcus mitis* and *Neisseria* are said to be the primary colonizers. Initially, these organisms make non-specific, long range Vander Waals bonds with the molecules. Later, they develop stronger, irreversible, short range adhesion with receptors in the acquired pellicle. Streptococcal oral bacteria have glucosyl transferases for adhesion whereas; Actinomyces species uses their fimbriae [13].

Attachment: There is a firm anchorage between pellicle and bacteria. The bonding between pellicle and bacteria are mediated trough specific extracellular proteins of the MO (adhesins) and complementary receptors present on the pellicle (cryptitopes).

Colonization and plaque maturation- There's an increase in dental plaque biofilm due to multiplication of primary colonizers and co-aggregation of secondary colonizing bacteria. While primary colonizers are aerobic or facultative arrives, secondary colonisers are gram negative species such as Actinomyces species, *Fusobacterium nucleatum, Prevotella intermedia* and *Capnocytophaga species.*

The dental plaque has a 'corn-cob' appearance and 'test-tube brush' appearance due to the adherence of cocci to filamentous bacteria. During this stage, plaque bacteria secrete EPS, which forms the scaffold for the dental plaque biofilm [14]. Some gram negative anaerobic bacterial species colonize and within 7 days, give rise to the 'tertiary colonizers'. Tertiary colonizers include *Porphyromonasgingivalis* and *Aggregati*bacteria, *Actinomycetemcomitans* and Spirochetes such as *Treponema denticola*. The bacteria and micro colonies tend to communicate with one another. This is done by the process of 'Quorum sensing'.

Quorum sensing or cell density mediated gene expression in the bacteria is the regulation of expression of specific genes by accumulation of signaling compounds that mediate intercellular communication. Signaling by quorum sensing involves a signaling pathway that is mediated by response to cell density. This is seen in both Gram positive and Gram negative microorganisms. The stimuli of quorum sensing systems 'auto inducers' are produced at basal constant level and their concentration is a function of microbial density. Quorum sensing gives the biofilm their distinct properties. They also help the potential to encourage the growth of beneficial bacteria and discourage growth of competitors [15].

Quorum quenching refers to all processes involved in the disturbance of Quorum sensing. Quorum quenching molecular actors are diverse in nature (enzymes, chemical compounds), mode of action (Quorum sensing-signal cleavage, competitive inhibition, and so on) and targets, as all main steps of the quorum sensing pathway that include synthesis, diffusion, accumulation and perception of the Quorum sensing signals may be affected. Some parameters, like temperature and pH may affect the half-life of quorum sensing signals. This natural mechanism is evolved either by quorum sensing-emitting organisms for the recycling or clearing of their own signals or by the quorum quenching organisms from their competitive relationship with quorum signal-emitting organisms.

The enzymatic degradation of QS signal is by soil isolates of *Variovorax* and *Bacillus* genera. They represent four catalytic classes: The lactonases, the amidases that cleave AHLs at the amide bond and release fatty acid and homoserine lactone, the reductases and cytochrome [16].

Factors affecting dental plaque formation

Environmental factors

Temperature

Oxygen tension

Availability of nutrients

Host factors

Saliva: One of the key properties of saliva is its action as an effective buffer, adjusts the pH of most surfaces. Saliva normally has pH 6.75 -7.25. Many bacteria found in healthy plaque can withstand exposure to low pH, but are killed or inhibited by prolonged exposure. Saliva contains important growth factors, proteins and minerals that can be used in the metabolism of bacteria.

Gingival fluid: which contains proteins, and amino acids, minerals, vitamins and glucose? It has the protective functions for the host due to the effect of rinsing (gums and teeth), and the high number of antibodies

Diet: The presence of carbohydrates such as lactose, glucose, maltose leads to increased plaque. Also frequent consumption of these substances increases the chances of caries on the tooth surface. Some foods remain between the teeth or adhering to the Occlusal surfaces of the teeth. So bacteria have "food" and multiply by secreting products of their metabolism, which is detrimental for our teeth [3].

Microbial factors

This includes adherence, retention, and co aggregation, microbial inter and intra species interactions and virulence mechanism [3].

Tooth related factors

Surface irregularities are responsible for formation of plaque so it is called individualized plaque growth pattern. This pattern may change severely when the tooth surface contains irregularities that offer a favorable growth pattern. Rough intraoral surface accumulate and retain more plaque in terms of thickness, area and colony-forming

unit. In the dental arch, more difference in plaque growth rate can be detected. Plaque formation generally occurs faster in lower jaw when compared with upper Jaw [3].

Characteristics

Macroscopic Surface Characteristics

Macroscopic cell surface characteristics relevant for adhesion include Surface energy, Zeta potential and hydrophobicity, which are the interrelated phenomena [17].

Surface energy: Critical surface tension of interfacial surface energy been postulated as a driving force for initial adhesion of microorganisms to solid surfaces [18].

Zeta potential: The zeta potential of organisms has been described as adhesion. Being important surface characteristics in adhesion [19].

Hydrophobicity: Although microbial hydrophobicity is ill-defined. Hydrophobicity of microorganisms has been implicated for adherence [19].

Microscopic surface characteristics

Salivary components: Several salivary components have been shown to aggregate microorganism. The aggregating ability has been taken to support a role of certain salivary component in microbial adhesion to pellicle covered tooth surfaces.

Microbial adhesion: The prevalence of microbial species on a surface has been found to correlate with the organism's inheritance ability to adhere to surface [20].

Intermicrobial co-aggregation: Species or strain-specific co-aggregation based on cell-to cell recognition has been occurring among a diversity of diversity of micro-organisms. The so called 'corn cob' structures have been observed microscopically in dental plaque have been taken as evidence for the importance of such coaggregation in vivo plaque formation.

Plaque hypothesis

Nonspecific plaque hypothesis

The idea that the overall activity of the total microflora could lead to disease, was enriched by taking into account difference in virulence among bacteria. The noxious product elaboration by the entire plaque flora. The host metabolism nullifies the noxious substance produced by the oral micro biota. The increased production of the noxious substance will overwhelm host defense mechanism [21].

Traditional non-specific plaque hypothesis (T-NSPH)

T-NSPH postulated that it was the quantity of plaque that determined the pathogenicity without discriminating between the levels of virulence of bacteria. Believing this, the host would have a threshold capacity to detoxify bacterial products (e.g., saliva neutralizing acid) and disease would only develop if this threshold was surpassed and the virulence factors could no longer be neutralized [22].

Updated nonspecific plaque hypothesis (U-NSPH)

(U-NSPH) took into consideration that some indigenous sub gingival bacteria can be more virulent than others and that plaque composition changes from health to disease. Unlike the classic NSPH, the updated NSPH could explain this by taking into account that differences in the plaque microbial composition could lead to differences in pathogenic potential. Hence the total amount of plaque in mouth is directly proportional to the periodontal disease [21].

Specific plaque hypothesis

The importance of the qualitative micro biota composition is stated. Specific bacterial pathogens provoke the periodontal disease. Acceptance of *A.actinomycetemcomitans* in pathogenesis of localized aggressive periodontitis proved that specific bacteria act as pathogens [22].

The "Specific Plaque Hypothesis" (SPH), postulated that dental caries was an infection with specific bacteria in the dental plaque of which the most relevant were "mutans streptococci" main species: *Streptococcus mutans* and *Streptococcus sobrinus* and lactobacilli [21]. This also suggested that "specific-pathogens" are part of the indigenous microflora and unlike foreign pathogens cannot be eliminated from the oral cavity. Potential periopathogens included: protozoa, spirochetes, streptococci, and actinomyces. In addition, Gram-negative, anaerobic rods including black-pigmented *Bacteriodes* such as *Bacteriodesmelaninogenicus* (renamed to *Prevotelamelaninogenica*) and others from the genus *Wolinella* (re-classified as *Campylobacter*) and facultative anaerobic, Gram-negative rods of the genera *Capnocytophaga, Eikenella* and *Actinobacillus. This identification led to the idea that specific pathogens cause specific disease.*

Ecologic plaque hypothesis

A hypothesis that combines key concepts of the earlier two hypotheses: The "Ecological Plaque Hypothesis" (1994), which proposes that disease, is the result of an imbalance in the microflora by ecological stress resulting in an enrichment of certain disease-related micro-organisms [21]. Total amount of dental plaque + Specific micro biota = Healthy tissue to diseased tissue. Dental plaque biota is relatively stable and dynamic in equilibrium. The changes in microbial composition to changes in ecological factors such as the presence of nutrients and essential cofactors, pH and redox potential [21]. For example, frequent exposure to a low pH, for instance as the result of sugar fermentation, leads to a relative increase of acid-tolerant species. When tempered by inflammation the gingivocrevicular fluid is reduced. This reduction in the GCF induces shift in the micro biota called *dysbiosis*. The interspecies competition keeps the microbial micro biota balanced. Bacterial growth is dictated by the environment, which in turn is influenced by bacterial metabolism, leading to mutual dependencies in health but also a chain of events that lead to diseases.

Keystone pathogen hypothesis

This hypothesis proves that even low abundance microbial pathogen can make a normally benign micro biota into a dysbiotic one. *Porphyromonasgingivalis* is shown to be able to manipulate the native immune system of the host. By doing so it was hypothesized that it does not only facilitate its own survival and multiplication, but of the entire microbial community. In contrast to dominant species that can influence inflammation by their abundant presence, keystone pathogens can trigger inflammation when they are present in low numbers. Importantly, even though their absolute number increases, keystone pathogens can decrease in levels compared to the total bacterial load which increases as plaque accumulates in periodontitis. Porphyromonas Gingivalis is considered as the Key Stone Pathogen a per this hypothesis.

Role of P. Gingivalis as a Key Stone Pathogen

P. gingivalis exerts its keystone effects through bacterial synergy

as well as modulation of the host. In terms of host modulation, *P. Gingivalis* facilitates the colonization and growth of other organisms, for example, *F. nucleatum*, by delaying neutrophil recruitment by transiently inhibiting the initiation of chemokine's like gingival IL8 and T-cell chemokine-like IP 10. It has also shown to affect the function of neutrophils by activating toll-like receptor (TLR) 2 and C5aR. However, the persistence of *P. gingivalis* in the periodontium is dependent on the instigation of incendiary crosstalk seen between receptor of complement C5a and TLR 2 and also the ability of its gingipains to produce C5 convertase activity, which has shown to retard the annihilating ability of leukocytes. This was substantiated by a study in which dysbiosis and periodontitis could not be caused by *P. gingivalis* in C5aR sans host (mice) [23-25].

Conclusion

In the view of the foregoing information, it seems appropriate to conclude that the clinical picture of dental disease is a net result of an interaction between the pathogenic dental plaque and host tissue response. Dental plaque biofilm cannot be eliminated. However, the pathogenic nature of the dental plaque biofilm can be reduced by reducing the bio-burden (total microbial load and different pathogenic isolates within that dental plaque biofilm) and maintaining a normal flora with appropriate oral hygiene methods that include daily brushing, flossing and rinsing with antimicrobial mouth rinse.

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Conflict of Interest

I declare to have no conflict of interest.

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