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DENTAL PLAQUE AS A BIOFILM – IMPLICATIONS FOR HEALTH, DISEASE AND MICROBIAL FACTION

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ABSTRACTS
Dental plaque is a structurally and functionally organized biofilm. The plaque is formed in an orderly manner and has a diverse microbial composition that remains relatively stable over time in health (microbial homeostasis). Predominant species from diseased sites differ from those occurring in healthy sites, although putative pathogens can often be detected in low numbers on normal pages. In dental caries, there is a shift towards community dominance of acidogenic and acid-tolerant species such as mutans streptococci and lactobacilli, although other species with relevant properties may be involved. Caries control strategies may include inhibition of biofilm development prevention of attachment of cariogenic bacteria, manipulation of cellular signaling mechanisms, delivery of effective antimicrobial agents, etc.) or enhancement of host defenses. In addition, these more conventional approaches could be extended by intervening in enabling factors cariogenic bacteria escape from the normal homeostatic mechanisms that limit their plaque growth and out competes the organisms associated with health. The evidence points to this regular low pH conditions in the plaque select streptococci and lactobacilli mutans. Therefore, Suppressing sugar catabolism and acid production by using metabolic inhibitors and non-fermentable artificial sweeteners in snacks or stimulating salivary flow could help in maintaining homeostasis in the plaque. Arguments will be presented that they will appreciate Ecological principles will enable a holistic approach to dental caries control.

INTRODUCTION
Dental plaque is a community of microorganisms is found on the surface of the tooth as a biofilm, stored in a matrix of polymers of host and bacterial origin.[1,2] clinical importance is the fact that biofilms are less sensitive to antimicrobial agents, while microbial communities can show increased pathogenicity (pathogenic synergism).[3] Plaque biofilm structure could they limit the penetration of antimicrobial agents, while the bacteria growing on the surface grow slowly and represent a novelty phenotype, one consequence of which is reduced sensitivity to inhibitors.[4] Plaque is natural and contributes (as well as the resident microflora of all other places in the body) to the normal development of physiology and host defense.[5]

Development of plaque biofilms on teeth
Dental plaque is formed through an orderly sequence of events, the result is structurally and functionally organized, species-rich microbial community.[2] Different phases in plaque formation includes: acquired pellicle formation; reversible adhesion involving weak long-range physicochemical interactions between the cell surface and the skin, which may lead to stronger adhesin-receptor mediated attachment; coadhesion leading to attachment secondary colonizers to already attached cells (Cisar – this symposium,[6] multiplication and biofilm formation (including the synthesis of exopolysaccharides) and further opportunity, separation. Increase in knowledge about mechanisms of bacterial attachment and coadhesion it can lead to control strategies or to influence the pattern biofilm formation (Cisar – this symposium). Analogues could be synthesized to block adhesin-receptor attachment or coadhesion and colonization properties surfaces could be chemically treated to reduce them contributing to microbial colonization. However, cells can express multiple types of adhesins,[7,8] so even if a the main adhesin is blocked, other attachment mechanisms may be used. Furthermore, although adhesion is necessary for colonization, the final proportions of a species in a mixed culture biofilm such as dental plaque will ultimately depend on the ability of the organism grow and compete with neighboring cells.

Once established, the overall composition of the climax plaque community is diverse and multispecies detected at individual locations. Molecular ecology approaches in which 16S rRNA genes are amplified plaque samples identified >600 bacteria and Archaea taxa, approximately

50% of which are currently uncultivable.[9] Once the plaque is formed, its species composition in a a place is characterized by a certain degree of stability or balance between component species, despite the regular minor environmental stress, e.g. from food components, oral hygiene, host defenses, daily changes in saliva flow etc. This stability (referred to as microbial homeostasis) is not because of some biological indifference among the population organisms, but it is due to the balance imposed by numerous microbial interactions, including examples of both synergism and antagonism.[10] These include conventional biochemical interactions such as those necessary for the removal of complex host glycoproteins and for the development of food chains, but also finer cell-cell signaling It can happen. This signaling can lead to a coordinated gene expression in the microbial community and these signaling strategies are currently considered potential targets for new therapeutics.[11,12]

Dental Plaque and Disease

Numerous studies have been undertaken to determine the composition of the plaque microflora from diseased sites in order to try and identify those species directly implicated in causing pathology. Interpretation of the data from such studies is difficult because plaque-mediated diseases occur at sites with a pre-existing diverse resident microflora, and the traits associated with cariogenicity (acid production, acid tolerance, intracellular and extracellular polysaccharide production) are not restricted to a single species. A comparison of the properties of strains representing several streptococcal species have shown considerable overlap in the expression of these cariogenic traits[14] (see below).

Microorganisms in biofilms such as plaque are in close physical contact, and this can increase the probability of interactions, some of which can modulate the pathogenic potential of cariogenic bacteria (for example, Kuramitsu and Wang – this symposium). Similarly, the consequence of acid production by cariogenic species can be ameliorated by the development of food chains with Veillonella spp., or due to base production by neighboring organisms. Not surprisingly, therefore, there has been only limited success in using the presence of specific species as diagnostic or prognostic indicators of disease. The advent of microarrays, in which the presence of all of the possible groups of micro-organisms in plaque can be determined, may enable particular microbial profiles (or molecular "signatures") to be identified that correlate with caries or periodontal disease (Stahl, this symposium), although markers of biochemical activity might also be needed.

Source of cariogenic pathogens

The origin and role of oral pathogens is the subject of much debate. Resolution of this debate is indeed key to the development of effective plaque control strategies. Earlier studies using conventional culture techniques often failed to recover suspected pathogens from healthy sites or, if pathogens were present, included only a small fraction of the microflora. However, the recent application of more sensitive molecular techniques has led to the frequent detection of low levels of several pathogens (participating in caries and periodontal diseases) in a wide variety of sites.[15] Bacterial typing schemes have shown that identical strains of putative cariogenic bacteria can be found in the plaque of mothers (or other close caregivers) and infants,[16] implying that transmission of such bacteria may occur. In either situation (i.e., naturally low levels of "pathogens" or low levels of exogenously acquired "pathogens"), these species would have to outcompete already established microflora residents to achieve an appropriate degree of numerical dominance to cause disease. . As noted above, for this to occur, normal homeostatic mechanisms would have to be disrupted, which is likely to occur only if there is a major disruption to the local environment . This suggests that plaque-mediated diseases result from an imbalance in the resident microflora resulting from the enrichment of the microbial community of pathogens due to the action of strong selective pressures. If so, interfering with these drivers could prevent pathogen selection and reduce disease incidence

Factors responsible for the disruption of microbial homeostasis

Studies of a range of habitats have provided clues as to the type of factors capable of disrupting the internal homeostasis that exists in microbial communities. A common feature is a significant change in nutrient status, such as the introduction of a new substrate or major chemical disturbance at the site. For example, it is recognized in environmental microbiology that nitrogen fertilizers washed from agricultural land into lakes and ponds can promote algal overgrowth. Algae can consume dissolved oxygen in the water, leading to a loss of aerobic microbial, plant and insect life (eutrophication). Similarly, atmospheric pollution with sulfur dioxide and nitrogen oxides can result in acid rain, which causes damage to plants and trees and the loss of aquatic life.

The local environment is known to change into plaque during disease. Tooth decay is associated with a more regular intake of fermentable carbohydrates in the diet, and therefore plaque is more often exposed to low pH. The effect of such environmental changes in nutrient availability and pH on gene expression by oral bacteria prevalent in health or disease has shown that organisms such as mutans streptococci are better adapted to low pH and upregulate a number of genes. Which protects against acid stress. For example, S. mutans cells up-regulate a number of specific proteins and functions when exposed to sublethal pH values (approximately 5.5). This increases survival in acidic conditions such as those found in caries. These differences in phenotype will alter the competitive ability of the bacteria in the plaque. Laboratory model studies involving diverse but defined communities of oral bacteria have been conducted to answer specific questions regarding the

consequences of such changes on the relative competitiveness of individual species and the impact on community stability. Analysis of these studies led to the formulation of an alternative hypothesis related to the role of oral bacteria in dental disease and to the identification of factors that disrupt the natural balance of resident plaque microflora.

Impact of environmental change – Mixed culture modeling studies

As noted earlier, individuals who frequently consume sugar in their diet generally have increased levels of cariogenic bacteria such as streptococci mutans and lactobacilli in their plaque and are at greater risk of tooth decay. Whether the increase in cariogenic bacteria is due to the sudden availability of sugar per se (e.g. due to more efficient sugar transport systems in these bacteria) or is a response to the inevitable low pH conditions following sugar consumption can never be determined in animal studies or epidemiological surveys in humans. Exploiting the unique advantages of parameter control in the chemostat, together with the reproducibility of a defined mixed culture inoculum, allowed for the first time to separate these coupled effects. Two mixed culture chemicals were inoculated with 9 or 10 species (representing healthy and diseased individuals) in a growth medium at pH 7.0 in which mucin was the major carbohydrate source; under these conditions, S. mutans and Lactobacillus rham nosus were noncompetitive and composite <1% total microflora). Once the consortium was stably established, both chemostats were pulsed with a fermentable sugar (glucose) daily for ten consecutive days. In one chemostat, the pH was automatically maintained throughout the study at neutral pH (as found in a healthy mouth) to determine the effect of fermentable sugar addition on culture stability, while in the other the pH was allowed to fall by bacterial metabolism for six hours after each pulse (how it occurs in vivo); The pH was then returned to neutral for 18 h before the next pulse.[17] In contrast, when the pH was allowed to change after each pulse, progressive selection of cariogenic (a acid-fast) species at the expense of bacteria associated with dental health. After the last pulse of glucose se the community was dominated by species involved in the development of dental caries (S. mutans and L. rhamnosus made up approx. 55% microflora). When this study was repeated, however The drop in pH was limited after each glucose pulse to any pH 5.5, 5.0 or 4.5 in independent experiments, similarly enrichment of cariogenic species at the expense of healthy species, but their increase was directly proportional to the extent of the pH decrease. collectively, these studies showed conclusively that it was low pH arising from the metabolism of sugars rather than the ability of sugar availability, which led to the breakdown of microbial homeostasis in dental plaque. This finding is important implications for caries control and prevention; data suggest that the selection of cariogenic bacteria could be prevent if pH changes after sugar metabolism could be reduced.

CONCLUSION

The key to a more complete understanding of the role of microorganisms in dental diseases such as dental caries may depend on a paradigm shift away from concepts that have evolved from studies of classical medical infections with a simple and specific (eg, single-species) etiology to understand. ecological principles. The development of plaque-mediated disease at the site can be seen as a breakdown of the homeostatic mechanisms that normally maintain a beneficial relationship between the resident oral microbiota and the host. When assessing treatment options, it will enable the enlightened practitioner to assess the ecology of the oral cavity to take a more holistic approach and take into account nutrition, physiology, host defenses and the overall well-being of the patient as these will be affected by the balance and activity of the resident oral microflora. Future episodes of disease will occur if the cause of any homeostasis disturbance is not recognized and addressed. For example, a side effect of many medications is a decrease in saliva flow. This will have a detrimental effect on sugar clearance and buffering capacity, thereby promoting the growth of acid-tolerant and potentially cariogenic bacteria. The identification of such critical control points can lead to the selection of appropriate caries prevention strategies that are tailored to the needs of individual patients. In this way, the doctor not only treats the end result of the dental caries process, but also tries to identify and intervene in factors that, if left unchanged, will inevitably lead to further diseases.

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