Microbiological aspect of dental caries

D. عذراء مصطفى

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• Dental caries is the result of the metabolic activities of bacteria in microbial communities on teeth termed dental biofilms (often referred to as dental plaque). Hence, the presence of microbial communities on the tooth surface is a prerequisite for caries lesions to develop. However, teeth may be covered by biofilms without always presenting visible signs of caries.

• Resident microflora Acquisition of the resident microflora occurs from birth and is a natural process, during which all environmentally exposed surfaces of the body become colonized.

• Once established, the resident microflora has a diverse composition, consisting of a wide range of Gram-positive and Gram-negative bacterial species, as well as yeasts and other types of microorganism.

• In addition, the composition of the oral microflora will change as the biology of the mouth alters over time.

• Benefits of the resident microflora

The resident microflora contributes directly and indirectly to the normal development of the host, and functions as part of the innate host defenses by acting as a barrier ('colonization resistance') to permanent colonization by
transient organisms, some of which are potentially pathogenic. Mechanisms involved in colonization resistance by resident organisms include:

- saturation of microbial attachment sites
- more effective competition for essential nutrients
- creation of conditions unfavorable to the growth of invading microbes
- the production of inhibitory factors, e.g. bacteriocins and hydrogen peroxide.

**Transmission of cariogenic flora**

Oral infection in children is related to frequent contact with large numbers of bacteria from the mother (“cuddle effect”) and other caregivers within the discrete period between 18/19 and 31 months (the “window of infectivity”). But the presence of caries testifies that children aged 9–10 months are also already infected and S. mutans was detected in 25% of one sample of pre-dentate children.

The correlation between numbers of mutans streptococci in maternal and children’s saliva is 70% of 3-yearold children harbored S. mutans, compared with 40% when mothers’ microflora was suppressed for a period spanning a few years. If 15-month-olds harbored S. mutans, the probability of caries at the age of 3 years was 75%. In one study, children free of mutans streptococci until age 5 had more sound teeth at age 11 than children who acquired the bacteria earlier.

When the number of S. mutans in a mother is reduced permanently, her child’s mouth is colonised at a later stage or not at all. Bacteria newly introduced in the mouth are more likely to get established in children than in adults with their highly colonisation-resistant, optimally adapted flora. In adults, a new bacterium rarely spreads from the inoculation site and becomes undetectable
within a few weeks. In contrast, tooth eruption creates conditions favourable for colonisation. In 19–31-month-old children a new bacterial colony is established relatively easily.

**Oral environment**

Adherence is a key ecological determinant for oral bacteria to survive and persist. The mouth is unique in the human body in possessing non-shedding surfaces (teeth) for microbial growth, leading to extensive biofilm formation (dental plaque). In contrast, desquamation ensures that the bacterial load is relatively light on mucosal surfaces.

**Oral habitats**

Buccal mucosa, dorsum of the tongue, teeth, crevicular epithelium, and prosthodontics and orthodontics appliances.

**Factors modulating microbial Growth**

a-anatomical factor  
b-saliva  
c-gingival crevicular fluid  
d-microbial factors  
e-miscellaneous  
f-nutrition of oral bacteria  

Teeth do not provide a uniform habitat for microbial growth which is optimal for the growth of many micro-organisms. Saliva has a profound influence on the ecology of the mouth for example, its ionic composition promotes its buffering properties and its ability to remineralize (i.e. repair) enamel.
In addition, the organic components (glycoproteins and proteins) can influence the establishment and selection of the oral microflora by either coating oral surfaces, and adaptive immunity (e.g. sIgA) and so can directly inhibit some exogenous micro-organisms.

Saliva plays other roles in regulating the growth and metabolic activity of the oral microflora. Saliva helps to maintain the pH in the oral cavity at values around 6.75-7.25 and the temperature at around 35-36°C.

The tongue has a highly papillated surface providing protection in the crypts to fastidious bacteria including obligate anaerobes. The tongue act as reservoir for many species that are commonly found in dental plaque. So the biological and physiological properties of each site result in only a subset of the organisms (often 20-30 distinct types) being able to predominate at an individual site.

A carbohydrate-rich diet increases the acid production and growth rate of many oral bacteria.

**Plaque development**

- Soon after polishing a tooth, a biofilm of negatively charged salivary glycoproteins, the salivary pellicle, is seen to adhere to the tooth. Salivary bacteria ($\sim 2 \times 10^8$/mL) are attracted towards this pellicle, and the sparse, reversible colonisation of the pellicle with a few species soon stabilises: microcolonies develop, which produce an interbacterial matrix that encloses other bacteria which do not have a capacity to adhere to the pellicle

- The complexity of the plaque microbial population strongly depends on the salivary properties, crevicular fluid, mechanical factors, the substrate and other plaque-related factors (e.g. age of the plaque). It takes a few days for newly formed plaque to become cariogenic.
Composition of plaque

• Already at birth, the mouth is colonised by bacteria. The oral epithelium is in a state of continuous replacement; thus, bacteria that need to adhere to stable surfaces to multiply (e.g. S. mutans) only colonise the oral cavity permanently after tooth eruption. The then more complex plaque that forms contains bacteria associated with caries.

• Bacteria comprise 70% of plaque; the remainder 30% is composed of intercellular material derived chiefly from the bacteria, salivary proteins and epithelial cells, and also plaque fluid with calcium and phosphate, and, rarely, food remnants.

Factors affect the amount and rate are
tooth brushing, sugar consumption, antimicrobial features of saliva, etc.

In experimental studies, germ-free animals fed on a cariogenic diet developed caries only after transfer of microflora from other animals. Long-term use of antibiotics or chemotherapeutic agents restricted the oral microflora and reduced the frequency of occurrence of carious lesions.

• The plaque composition determines its pathogenicity and this is different for different types of tooth surface.

• Pits and fissures

harbor many bacterial genera, but 80% are Gram-positive cocci, among which are large numbers of S. mutans, S. sanguis and Lactobacillus strains. Actinomyces species are also present.

After progression of the carious lesion into the dentine, S. mutans remains important but many other bacteria are also present.
• Approximal Plaque
accumulating underneath the contact points has a more varied and locally different composition. In particular it contains actinomycetes, followed by fewer numbers of Gram-negative bacteria and even fewer streptococci, but the numbers of S. mutans increase when caries develops.

• Free smooth surfaces
Here, the mechanical action of the cheeks, tongue, mastication and tooth brushing restrict plaque growth. Only bacteria that are able to strongly adhere to the teeth initially become established. S. mutans, preceded by S. salivarius and Actinomyces, are important in smooth surface caries

• Cervical
The most complex and thickest plaque is found cervically. The crevicular fluid and saliva maintain the plaque, which consist of more than 50 species, including Actinomyces and strict anaerobic bacteria in large quantities. Some of these play a role in root caries.

• In children, as the rate of crevicular fluid flow is low, the cervical plaque resembles the approximal plaque.

• Subgingival plaque
differs from supragingival plaque and has a high percentage of anaerobes. the quality and quantity of the microflora (plaque) at a site that may be considered vulnerable to caries determines whether the disease develops or not
Virulence factors

The amount of acids, the rate of production and the time for which they remain on the tooth surface co-determine the cariogenicity of plaque. In the presence of sugar, plaques with larger numbers of mutans streptococci quickly produce much acid

- The plaque hypothesis to explain role of dental biofilm bacteria in the etiology of dental caries

- The specific plaque hypothesis proposed that, out of the diverse collection of organisms comprising the resident plaque microflora, only a single or very small number of species were actively involved in disease. This proposal has been easy to promote because it focused efforts on controlling disease by targeting preventive measures and treatment against a limited number of organisms, such as by vaccination or gene therapy or by antimicrobial treatment

- non-specific plaque hypothesis considered that disease is the outcome of the overall activity of the total plaque microflora, so not just those that make acid, but also species that produce alkali or consume lactate need to be considered. Thus, a heterogeneous mixture of microorganisms could play a role in disease.

- Explanation: of non specific theory
  1-there is extreme variation in supragingival plaque
  2-other plaque bacteria have the same biochemical characteristics
  3-bacteria associated with caries other than mutans streptococcus and lactobacillus
  4-the presence of lactate-consuming species (e.g. Veillonella)
5-the production of alkali to raise the local pH (e.g. by ammonia production from urea or arginine by S. salivarius and S. sanguinis, respectively).

This has a great significance for caries prevention, since implicit in the hypothesis is the concept that disease can be controlled by targeting the putative pathogens (mutans streptococci and other acidogenic/aciduric species) through interference with the factors that are driving the deleterious shifts in the balance of the microflora. Identification of such critical control points (e.g. mechanical biofilm removal, saliva stimulation and/or dietary control) can lead to the selection of appropriate caries-preventive strategies that are tailored to the needs of individual patients.

- The ecological plaque hypothesis proposes that the organisms associated with disease may also be present at sound sites, but at levels too low to be clinically relevant. Disease is a result of a shift in the balance of the resident microflora driven by a change in local environmental conditions. In the case of dental caries, repeated conditions of low pH in plaque following frequent sugar intake (or decreased sugar clearance following low salivary secretion) will favor the growth of acidogenic and aciduric species, and thereby predispose a site to caries.