Review

The mouth is a habitat of a large, diverse and complex microbial environment. Different types of bacteria live on different surfaces in the mouth, accumulating the hard and soft tissue in the form of biofilms, affecting the ecological filaments of the dental surface and the gingival epithelium [1,2]. More than 750 types of bacteria or phylotypes, of which 50% are not yet cultivated, are detected in the oral cavity. They developed mechanisms of host adaptation and mechanisms of modification and avoidance to the host immune response. But the human immune system constantly monitors their growth and reproduction, preventing their invasion of the surrounding tissue and the development of disease. In fact, there is a dynamic equilibrium between the bio-communities of the bacteria and the human immune system, and oral health depends on the integrity of all-natural barriers that normally block the activity of microorganisms.

The oral cavity of the newborn does not contain bacteria but is rapidly colonized with Streptococcus salivarius. With the onset of the first teeth, Streptococcus mutans and Streptococcus sanguinis appear. Various anaerobes predominantly inhabit gingival sulcus, while bacteroids and spirochaetes appear in the mouth during puberty [2]. The concentration of bacteria in the saliva is about 108/ml with 90% of anaerobic bacteria, where the predominant organism is Veillonella parvula [3]. In a healthy oral cavity, Streptococcus, Pepto streptococcus, Veillonella and diphtheroids account for more than 80% of the total microbial flora. Many clinically significant oral anaerobic gram-negative bacilli previously included within the “oral Bacteroides” group are now reclassified as Porphyromonas or Prevotella species [4].

Oral cavity cannot be considered a unique uniform environment. Although representatives of various types of microorganisms may be isolated from several areas of the mouth, however, certain organisms tend to colonize certain predilection sites. As for example Fusobacterium, pigmented Prevotella, and anaerobic spirochaetes are concentrated in the gingival sulcus. Bacterial adherence and interaggregate, local environmental conditions, such as the presence of oxygen, pH, and other host factors seem to regulate these unique patterns of colonization and affect the composition of the oral flora. As and Pasteur research has confirmed that different bacteria live on different surfaces in the mouth because of specific bacterial cell adhesion molecules that are associated with complementary specific oral surface receptors [5,6].

Some of the bacteria in the mouth are responsible for oral diseases such as caries and periodontal disease, which are one of the most common diseases in humans. At least 35% of adults between 30-80 years in the United States have some form of periodontal disease [7,8]. Specific oral bacterial species are also considered to have a role in systemic diseases such as bacterial endocarditis [9], aspiration pneumonia [10], osteomyelitis in children [11] and cardiovascular diseases [12,13].

Bacteria in the oral cavity have evolved over time, with the aim of protecting individual bacterial organisms, in supra and sub-gingival bacterial communities, that is, plaque. The dental plaque is a dense, non-mineralized, complex mass of bacterial colonies that live in a gel-intermixing matrix and adheres to the tooth. Contains bacterial cells, salivary polymers and bacterial extracelluler products [11]. The flora of the plaque formation involves the presence of 2.5x107 aerobic bacteria/mg plaque and about 4.6x107 anaerobic bacteria/mg plaque [14]. The dental plaque begins to accumulate within 24 hours without regular tooth brushing. The dental plaque has an extra-polysaccharide that surrounds it and protects it from the penetration of an antibiotic. After the formation of the dental plaque, visible gingival inflammation develops in 2-4 days due to the production of various metabolites such as endotoxins, lipoteic acid, mucopolptides, metabolic products, and proteolytic agents such as hyaluronidase, and chondroitinase that can reach the gingival tissue directly or act indirectly through the stimulation of the immune system which further leads to further destruction.

Plaque accumulation can be stimulated by increased production of gingival cervical fluid that contains growth factors of various bacteria including gram negative anaerobes such as Porphyromonas gingivalis, Prevotella melaninogenica and Fusobacterium nucleatum.
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