

Bacterial Flora of the Oral Cavity in Periodontal Diseases

Ambarkova Vesna*

Department for Preventive and Pediatric Dentistry, Faculty of Dentistry, University Ss. Cyril and Methodius, Skopje, Republic of Macedonia

***Corresponding Author:** Ambarkova Vesna, Department for Preventive and Pediatric Dentistry, Faculty of Dentistry, University Ss. Cyril and Methodius, Skopje, Republic of Macedonia.

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The environment of the subgingival sulcus is similar to that of the colony, the oxidation-reduction potential is 300 mV, the concentration of bacteria reaching 10^{12} ml, while the anaerobic bacteria account for 99% of the cultivated flora. In this area accumulation of the subgingival plaque leading to periodontal diseases. A specific and unique bacterial composition of the sub-gingival plaque has been identified in patients with periodontal diseases [1,2,3]. Many bacteria that are specifically associated with periodontal diseases such as *Porphyromonas gingivalis*, *Tannerella forsythia* and *Treponema denticola* have not been detected in individuals without clinical signs of periodontal diseases [4,5]. According to the Mane., et al. 2009 study, for the presence of oral bacteria in patients with periodontal diseases, the most commonly isolated bacteria are: *P. gingivalis* 48%, *F. nucleatum* 24%, *Pepto streptococcus micros* 23% and *Prevotella spec* 26% [6].

In parodontium of healthy individuals, the microflora of the sub-gingival plaque is consisted mainly of gram-positive organisms, such as *Streptococcus sanguinis* and *Actinomyces* spp. In the individuals with gingivitis, in the content of the sub-gingival flora increases the percentage of anaerobic gram-negative bacilli increased significantly, while *Prevotella intermedialis* is dominantly present [7]. In the patients with periodontal disease, a further increase in the complexity of the microbial plaque composition happens with the dominant presence of anaerobic gram-negative bacilli *Aggregatibacter (Actinobacillus) actinomycetemcomitans*, *Porphyromonas gingivalis*, *Prevotella intermedia*, *Treponema denticola* and *Tannerella forsythia* (formerly *Bacteroides forsythus*) [8].

Today's theory is that the supra-gingival plate serves as a reservoir of Gram negative pathogenic bacteria, which under favorable local conditions migrate into sub-gingival space and form a sub-genital biofilm that can be very damaging for periodontal structures. Pathogenic gram-negative bacteria, of which about 10 species are placed in the group of pathogens that cause periodontal disease, are the primary aetiological factor leading to the development and development of periodontal disease [5]. Socransky, et al. have grouped bacteria from dental plaque in five bacterial complexes, while two of them are responsible for the development and development of periodontal disease: "red" and "orange" complex. In addition, the remaining bacteria are grouped in the "green", "yellow" and "violet" complexes, such as bacterial colonies formed on the surface of the teeth and are preceded by colonization with the "orange" and "red" complexes. The "Red" complex consists of three closely related species: *Tannerella forsythia*, *Porphyromonas gingivalis* and *Treponema denticola*. This bacterial complex demonstrated an association with the depth of the periodontal pocket in patients with periodontal disease. The orange group in which *Prevotella intermedialis*, *Fusobacterium nucleatum*, *Pervotella nigrescens*, *Peptostreptococcus micros*, *Campylobacter rectus*, *Campylobacter gracilis*, *Campylobacter showae*, *Eubacterium nodatum* and *Streptococcus constellatus*, precede colonization from the "red" complex.

The Yellow Complex consists of strains of *Streptococcus*: *Streptococcus sanguis*, *Streptococcus oralis*, *Streptococcus intermedius*, *Streptococcus Gordonii* and *Streptococcus mitis*, while *Capnocytophaga ochracea*, *Capnocytophaga gingivalis*, *Capnocytophaga sputigena*, *Ekinela corrodens* and *Aggregatibacter actinomycetemcomitans* are an integral part of the “green” complex. The fifth and final complex, the Violet complex, composed of *Veillonella parvula*, *Actinomyces odontolyticus*, *Selenomonas noxia* and *Actinomyces naeslundii* genospecies 2. Specific factors of virulence of the parodonto-pathogenic bacteria, such as lipopolysaccharides and proteolytic enzymes, play an important role in the genesis of the periodontal disease [9,10]. However, host factors, such as aggregated inflammatory response, genetic predisposition, environmental factors such as smoking and poor nutrition, are also significant [11].

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