

Microbial Flora of Oral cavity

¹Dr. Fathima Abdul Jabbar, Department of Oral and Maxillofacial Pathology, Karpaga Vinayaga Institute of Dental Sciences, Chinnakolambakkam, Madhuranthagam, Chengalpattu, Tamilnadu – 603308

²Dr. R Mahalakshmi, Department of Oral and Maxillofacial Pathology, Karpaga Vinayaga Institute of Dental Sciences, Chinnakolambakkam, Madhuranthagam, Chengalpattu, Tamilnadu – 603308

³Dr. S Ramya Srinivasan, Department of Oral and Maxillofacial Pathology, Karpaga Vinayaga Institute of Dental Sciences, Chinnakolambakkam, Madhuranthagam, Chengalpattu, Tamilnadu – 603308

⁴Dr. S Kanimozhi, Department of Oral and Maxillofacial Pathology, Karpaga Vinayaga Institute of Dental Sciences, Chinnakolambakkam, Madhuranthagam, Chengalpattu, Tamilnadu – 603308

⁵Dr. P Karthiga, Department of Oral and Maxillofacial Pathology, Karpaga Vinayaga Institute of Dental Sciences, Chinnakolambakkam, Madhuranthagam, Chengalpattu, Tamilnadu – 603308

⁶Dr. M Sathish Kumar, Department of Oral and Maxillofacial Pathology, Karpaga Vinayaga Institute of Dental Sciences, Chinnakolambakkam, Madhuranthagam, Chengalpattu, Tamilnadu – 603308

Corresponding Author: Dr. Fathima Abdul Jabbar, Department of Oral and Maxillofacial Pathology, Karpaga Vinayaga Institute of Dental Sciences, Chinnakolambakkam, Madhuranthagam, Chengalpattu, Tamilnadu – 603308

How to citation this article: Dr. Fathima Abdul Jabbar, Dr. R Mahalakshmi, Dr. S Ramya Srinivasan, Dr. S Kanimozhi, Dr. P Karthiga, Dr. M Sathish Kumar, “Microbial Flora of Oral cavity”, IJMACR- November – December - 2022, Vol – 5, Issue - 6, P. No. 30 – 37.

Copyright: © 2022, Dr. Fathima Abdul Jabbar, et al. This is an open access journal and article distributed under the terms of the creative commons attribution noncommercial License 4.0. Which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

The oral cavity being the collection of variety of microbes, promotes the growth of microbial organisms, such as on the mucosa and teeth. These organisms are beneficial when present in right numbers. A delicate balance is maintained in the microbial ecosystem, with these organisms contributing to normal development and defenses. However, any change or disruption in the microbial profile due to any factors can result in an

unfavorable shift toward pathogenic organisms triggering various diseases. The objective of the article is to benefit the student population as the concept of oral microbial flora has been put forward in a simplified format.

Keywords: Microbial flora, oral cavity, oral disease.

Microbial ecosystem in oral cavity

The mouth is a place of a huge, diverse microorganisms. 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. 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 bacterial community and the human immune system. The oral cavity of the new-born is devoid of bacteria but is rapidly colonized with *Streptococcus Salivarius*. With the onset of the first teeth, *Streptococcus mutans* and *Streptococcus Saguinis* appear. Various anaerobes predominantly inhabit gingival sulcus, while *Bacteroides* and spirochaetes appear in the mouth during puberty. 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 extracellular products.

Types of oral flora

Indigenous flora

It refers to the organisms' present > 1% of total viable count. They are in a compatible relationship with the host and do not compromise the survival of the host. E.g., streptococcus, Actinomyces & Neisseria

Supplemental flora

It refers to the organisms identified in a significant amount (< 1%) in certain individuals. Egg lactobacillus

Transient flora

This flora comprises of organisms that may be present in the oral cavity for very short periods (hours to days) of time.

Factors contributing to normal microbial growth

Anatomical

Shape, topography and malalignment of teeth, Poor quality of restoration, non - keratinized sulcular epithelium

Saliva

Adsorption of salivary glycoprotein results in formation of salivary pellicle which in turn facilitates bacterial adhesion, Acidic saliva promotes growth of cariogenic bacteria. It is a rich source of food

Gingival crevicular fluid(gcf)

GCF flushes microbes out of crevice, It also acts as a primary source of nutrients to the crevicular bacteria, Maintain pH of gingival crevice, It has specific and non-specific defense factors like IgG, Neutrophils involved in GCF are involved in phagocytosis

Microbial factors

There is competition for receptors among different microbes resulting in prevention of attachment by 'late comers', Production of metabolic end products by certain bacteria lower the pH, some bacteria use the metabolic end products of other bacteria for nutritional purposes, Coaggregation of same or different species e.g., bacteria-corn-hub formation²

Miscellaneous factors

Local Environment Ph, Oxidation-reduction potential, antimicrobial therapy, Diet, Iatrogenic factors, Diurnal variations, Drugs, Extraction of teeth, Intraoral appliance²

Role of dental plaque

The oral cavity houses various habitats for microorganisms like, the mucosal surfaces (such as the lips, cheek, palate and tongue) and teeth which support the growth of microbial communities.⁶ Dental plaque is the community of microorganisms found on a tooth surface as a biofilm. Plaque formation is a normal phenomenon which contributes to the host's normal development and defenses. Problems arise as a result of disruption in the homeostasis existing between microbial communities in the plaque. Two common examples

explaining this imbalance are dental caries and periodontal disease. In the former a change in the nutrient status like excess carbohydrates (increased supragingival plaque) results in the balance tilting toward more aciduric and acidogenic bacteria (e.g., *Streptococcus mutans*) to thrive, resulting in the disease. In the latter, accumulation of plaque, due to poor oral hygiene can result in deepening of pockets, creating anaerobic environments, thus favoring the proliferation of pathogenic bacteria (anaerobic species like *Fusobacterium*, *Prevotella*) leading to periodontal disease. Thus, any change in the dynamics within this ecosystem can alter the flora, increase its potential pathogenicity, and subsequently initiate and promote oral diseases.¹

Plaque related disease- ‘periodontitis and periimplantitis ‘

Periodontitis

Periodontal disease affects the great majority of the adult population. Teeth are the ideal substrate for plaque formation because of their non-exfoliating surfaces. Chronic Periodontitis is defined as an infectious pathology leading to slow or moderately slow, progressive loss of attachment and bone. It occurs mainly in adult patients in either a localized or generalized form but can nonetheless affect children and adolescents. Predisposing factors such as cigarette smoke, stress and local (e.g., tooth-related, or iatrogenic) or systemic (e.g., diabetes mellitus or HIV infection) conditions can enhance the destructive effects of the microbiota. Aggressive Periodontitis, on the other hand, is characterized by rapid loss of attachment and bone destruction in otherwise clinically healthy patients and presents familial aggregation.

Secondary features are represented by: High levels of A. actinomycescomitans and, in some subjects, P. gingivalis in the associated plaque, Discrepancies between plaque deposits and severity of attachment and bone loss, Abnormalities in the host response, such as decreased PMN chemotaxis due to lower levels

Periimplantitis

Peri-implantitis can be considered the “twin-sister” of periodontitis, even though some important differences between natural teeth and dental implants must clearly be borne in mind, the most important being that implants are not surrounded by periodontal ligament and therefore present different biomechanics and defensive cell recruitment.

Biofilm

Bacteria colonize the oral cavity soon after birth and form organized, co-operating communities called biofilms within specific ecological niches, for example, the tongue, tooth, subgingival sulcus, tonsil and buccal mucosa. These biofilms allow the bacteria to live in a nutrient-rich environment that is protected from environmental insults, antimicrobial agents and frictional forces. These biofilms perform two important functions: (i) they prevent pathogenic colonization and (ii) they educate the immune system to recognize ‘friend and foe’. In a state of health, equilibrium exists between biofilm antigens and toxins and the host immune response.⁴ Inclusion in a biofilm brings about profound changes in microorganisms, which may therefore exhibit important differences from their planktonic counterparts, including physiological properties, susceptibility to antimicrobial agents, interaction with host tissues and immunological response. Initially, the aspecific plaque theory dominated, but later it became evident that every biofilm differs from others, and that only the selection of

some specific species can lead, under certain circumstances, to disease.

Clinical gingivitis is associated with the development of a more organized dental plaque. The species involved vary depending on local environmental characteristics, but the colonization pattern is always the same. The shift from gingivitis to periodontitis does not come about automatically, either in every patient or every site, but depends on three main factors: host susceptibility, pathogenic bacteria and “protective bacteria”.

Dental caries

Dental caries is a chronic disease that progresses slowly in most individuals and is characterized by localized destruction of the tooth following long contact/interaction with acidic products that result from the bacterial fermentation of dietary carbohydrates.¹ The most common chronic infectious disease, bacteria as main pathogen. It can lead to chronic and progressive destruction of dental hard tissue under many factors. Have a wide range and high incidence. Occur at any age. And the early childhood caries is the most harmful and has become a prevalent public health problem among preschool children globally, which it has many factors influence the incidence of, including oral microbiome³.

Microflora alteration can cause a disease. The normal microflora usually consists of non-mutans streptococci like the salivarius group (e.g., *Streptococcus salivarius*) on the root surface, mitis group (e.g., *S. sanguis*) in the pit and fissures and also a small number of microbes of the mutans group (not enough to induce caries).³ As the supragingival plaque accumulates or if there is an increased supply of carbohydrates, an acidic and anaerobic environment is created. The normal flora adapts to this change in the environment and can become aciduric. These conditions now become more favorable

for aciduric organisms like mutans streptococci thus demineralizing the tooth surface.¹

Periodontal diseases

The subgingival crevice is flooded with gingival crevicular fluid (GCF) which creates a neutral/alkaline environment due to the presence of nitrogenous compounds, such as amino acids, peptides and proteins.¹ Periodontal diseases frequently occur in human mouth, and can be divided into two categories, gingival diseases and periodontitis. It cause destruction of periodontium (tooth-supporting tissues such as gingiva and alveolar bone) and constitute a potential risk factor for certain systemic diseases.³ As the gingival sulcus deepens, this environment is established and under these conditions, a saccharolytic and anaerobic and/or proteolytic bacteria, such as *Fusobacterium*, *Eubacterium*, *Campylobacter*, *Prevotella* and *Porphyromonas* are found. Proteolytic bacteria can degrade nitrogenous compounds into small peptides and amino acids by cell membrane-bound and/or extracellularly secreted proteases, for subsequent use as metabolic substrates. These enzymes secreted by the micro-organisms for degrading the nitrogenous compounds, induce inflammation and immunoreactions¹. The red complex bacteria, *Fusobacterium nucleatum* (*F. nucleatum*) were the most prevalent with very high levels in all groups.

The green and blue complex bacteria were less prevalent compared with red and orange complex, except *Aggregatibacter actino mycetomcomitas* was detected in all localized aggressive periodontitis groups.³ *P. intermedia* and *F. nucleatum* are capable of growth at acidic and neutral pH, and are frequently found in supragingival plaque. In addition, *P. intermedia* and *F. nucleatum* are capable of neutralizing the acidic environmental pH by changing the acid-base balance

through amino acid metabolism. *F. nucleatum* and *P. Intermedia* colonize a shallow gingival pocket (where the pH is variable and sometimes becomes acidic) and then promote the establishment of a neutral pH environment. This induces inflammation and an increase in GCF, inducing and promoting the growth of more proteolytic bacteria like *P. gingival* is, and enhance the pathogenicity of *P. intermedia* through the increase in proteolytic activity and cytotoxic end products.¹

Cross-sectional studies have demonstrated that dentate patients, those with poor oral hygiene, and those who do not regularly visit their dentist are more likely to develop pneumonia, suggesting a potential link between poor oral health and lung disease. Dental plaque of hospitalized subjects with pneumonia has been shown to be a reservoir for *P. aeruginosa*. Thus, there appears to be an association between periodontal pathogens and the incidence of cardiovascular disease.³

Lactobacillus

Absent from oral cavity of new born appear during first year of life. Their presence depends of numerous factors such as presence of ecological niches e.g., natural anfractuositities of the teeth, 42.3% came from tongue 11.8% from gums and species *L. fermentum* and *L. plantarum* are predominant on tongue; *L. rhamnosus* is predominant on gum.² A correlation also exists between *Lactobacillus* rates in dental plaque and in saliva. One factor that could influence the rate of salivary lactobacilli during childhood is the carbohydrate intake. Higher counts have been found in subjects harbouring removable prostheses and in subjects consuming a lot of medicine. On the other hand, a low level of *Lactobacillus* count seems to indicate, with a good probability, a low carious activity. The clinical significance of the *Lactobacillus* count is also more

reliable when applied to huge samples than to one person. *Actinomyces* species promoted growth of key *Lactobacillus* species in a biofilm, as did *S. mutans* to a lesser extent, and that the ability of individual bacteria to form mono-culture biofilms is not necessarily an indicator of their survival and pathogenic potential in a complex multispecies biofilm community.⁹

Endodontic infections

The microbial flora of root canals has been studied extensively over the years, by using different sampling techniques and identification method¹. Infected root canals are colonized by a consortium of bacterial species. Certain *Streptococcus* spp. persists in treated root canals. Diversity of bacterial species across geographical locations has confirmed that there are shared, location specific and exclusive bacterial species.⁵

Candidiasis

Candidiasis or oral candidosis is one of the most common human opportunistic fungal infections of the oral cavity¹³. Candidiasis is caused by a yeast like fungus, *Candida albicans*. Other species like *C. tropicalis*, *C. Parapsilosis*, *C. stellatoidea* and *C. krusei* may also be involved (Table 5). *Candida* is a component of the normal oral microflora, with 30 to 50% of people carrying it in their oral cavity and is kept under control by means of specific and nonspecific defense mechanisms and also by the competition of the microbes in the normal flora. Colonization in the newborn occurs from the mother's vaginal flora or other exogenous sources. Most people usually carry a distinct strain of *Candida* and the colonizer is usually the culprit (infecting strain) if at all infection occurs¹.

Conclusion

Oral microbial flora, beyond doubt, have a very important role to play in maintenance of homeostasis of the ecosystem in the oral cavity. It is crucial for clinicians to be aware of this fact and they should focus their treatment toward control of this flora rather than eliminating it. A thorough knowledge of the normal and altered flora and the mechanics behind how the change can happen and what it might lead to would give us a fair idea of how various oral diseases could be controlled and preventive strategies be developed.¹

Despite the large bacterial flora that inhabits the oral cavity, microbiological diagnosis of oral disorders remains largely confined to research laboratories. However, advances in technology indicate that some of the traditional as well as novel techniques could readily be employed to aid clinicians at point of care. Comprehensive and miniaturized kit-based methods of traditional microbiological testing methods, could help identify pathogen(s) even by novices with a basic knowledge in microbiology.⁵

References

1. Patil S, Rao RS, Sanketh DS, Amrutha N. Microbial flora in oral diseases. The journal of contemporary dental practice. 2013 Nov 1; 14 (6):1202.
2. Patil S, Rao RS, Amrutha N, Sanketh DS. Oral microbial flora in health. World J Dent. 2013 Oct; 4 (4): 262 -6.
3. Gao L, Xu T, Huang G, Jiang S, Gu Y, Chen F. Oral microbiomes: more and more importance in oral cavity and whole body. Protein & cell. 2018 May; 9 (5): 488 -500.
4. Kumar PS. Oral microbiota and systemic disease. Anaerobe. 2013 Dec 1; 24:90-3.
5. Parahitiyawa NB, Scully C, Leung WK, Yam WC, Jin LJ, Samaranayake LP. Exploring the oral bacterial flora: current status and future directions. Oral diseases. 2010 Mar;16(2):136-45.
6. Tlaskalová-Hogenová H, Štěpánková R, Hudcovic T, Tučková L, Cukrowska B, Lodi nova -Žádníková R, Kozáková H, Rossman P, Bártová J, Sokol D, Funda DP. Commensal bacteria (normal microflora), mucosal immunity and chronic inflammatory and autoimmune diseases. Immunology letters. 2004 May 15;93(2-3):97-108.
7. Sbordone L, Bortolaia C. Oral microbial biofilms and plaque-related diseases: microbial communities and their role in the shift from oral health to disease. Clin Oral Investing. 2003;7(4):181-188. doi:10.1007/s00784-003-0236-1
8. Badet C, The baud NB. Ecology of lactobacilli in the oral cavity: a review of literature. Open Microbiol J. 2008; 2:38-48. doi:10.2174/1874285800802010038
9. Könönen E. Development of oral bacterial flora in young children. Ann Med. 2000; 32 (2): 107 - 112. Doi: 10.3109 /0785 389000 9011759
10. Suresh, BS & Shailaja, S. (2014). Endodontic Microflora- A Review. Journal of Oral Health and Community Dentistry. 8. 160 -165. 10.5005/ Joh cd – 8 - 3 - 160.
11. Garcia-Cuesta C, Sarrion-Pérez MG, Bagan JV. Current treatment of oral candidiasis: A literature review. Journal of Clinical and Experimental dentistry. 2014 Dec; 6 (5): e576. s