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Research Article

HUMAN ORAL MICROFLORA

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A B S T R A C T

Oral cavity is the first and foremost part of the gastrointestinal tract. The process of digestion starts from the oral cavity. Our food is metabolized initially in the oral cavity. Metabolism of the food substances in the oral cavity is brought about by the microflora present in different sites in the oral cavity. The oral cavity has a colony of microorganisms present even in healthy state, which when altered can lead to pathogenesis. Microbial flora is present at different sites in the oral cavity providing them habitats. This flora is classified into various categories of different species comprising of viruses, bacteria, yeasts and even on occasions, protozoa. The presence of flora is basically due to the specificity of organisms to each site which is termed as tissue specificity. The microflora exhibits tissue tropism and specific adherence responsible for the tissue specificity. The microflora present on the hard and soft surfaces exhibits biofilm formation wherein the pioneer species attach to the surface and form a network on which bacterial colonization occurs. The composition of microflora is affected by various factors such as temperature, redox potential, pH, endogenous and exogenous nutrients, innate and acquired host defence, host genetics and antimicrobial agents. Not all of the species in microflora are significant, however few species are important as they have significant role in both healthy as well as in disease state. Oral microbial species also form dental plaque, which can lead to dental caries on the tooth surface and periodontal diseases on the soft tissues of periodontium. Oral microbial species have been suggested in affecting cardiac health as bacterial endocarditis is caused by the streptococcus from the oral microflora.

INTRODUCTION

The oral cavity has many surfaces and every surface encompasses a wide diverse microflora. Oral cavity being the entry gate for the gastrointestinal tract provides a habitat for various microbial species inside the oral cavity such as bacteria, fungi, protozoa and few viruses. Bacteria being the most predominant species, is significant in causing diseases. Studies based on molecular ecology where 16s RNA was amplified from plaque samples revealed more than 600 bacterial species of which less than 50% are culturable. Members of this microflora are also participants in common oral cavity diseases like caries and periodontitis. There are many diseases where oral microbial species have been proved to be the culprit, such as, bacterial endocarditis, aspiration pneumonia, preterm low birth weight, osteomyelitis in children and cardiovascular diseases. Factors such as saliva, food intake, developing dentition and antibiotics also affect them. There are certain locations in the oral cavity such as gingival crevice which provide habitat for anaerobic species to survive.

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Tongue harbours mostly gram negative anaerobes and these microbes are responsible for periodontal diseases and malodour. Nutrients, water and moderate temperature provides the oral microbial species to survive within the oral cavity. These organisms causing dental diseases like dental caries, periodontitis and oral malodour with continued infection may even cause infectious disease of adjacent organ such as respiratory pneumonia.[1-19]

Factors which affects the growth of microorganisms in the oral cavity

- Temperature
- Anaerobiosis
- pH
- Nutrients
- Host Defences
- Host genetics
- Antimicrobial agents and inhibitors

Classification

Oral Bacteria Classification has been classified based on Gram's Staining

1. Gram Positive

2. Gram negative

Based on the effect of oxygen bacteria are classified as:

1. Obligate aerobe
2. Micro aerophilic
3. Facultative anaerobes
4. Obligate anaerobe

Table 1 Classification of bacteria based on Gram's Staining.

Gram positive		Gram Negative	
Cocci	**Rods**	**Cocci**	**Rods**
Abiotrophia	*Actinomyces*	*Moraxella*	*Campylobacter*
Peptostreptococcus	*Bifidobacterium*	*Neisseria*	*Capnocytophaga*
Streptococcus	*Corynebacterium*	*Veillonella*	*Desulfobacter*
Stomatococcus			*Desulfovibrio*
	Lactobacillus		*Eubacterium*
			Eikenella
	Propionibacterium		*Fusobacterium*
	Pseudoramibacter		*Haemophilus*
	Rothia		*Leptotrichia*

Few examples of anaerobic bacteria that are present in the oral cavity are: *Bifidobacterium, Lactobacillus, Actinomyces, Propionibacterium, Treponema, Veillonella, Arachnia, Bacteroides, Eubacterium, Fusobacterium, Leptotrichia, Peptococcus, Peptostreptococcus, Selenomonas* species. *Streptococcus salivarius* are the first group of bacterial species to colonize as soon as infants acquire new microflora, these microorganisms colonise on the tooth surface and gums followed by colonization of *Streptococcus sanguinis* and *Streptococcus mutans*. Species other than these adhere the soft tissues but not the teeth. Besides bacteria, oral cavity has major population of organisms constituting the oral microbiome. Studying separate species of organisms in this vast and diverse microbial communities is a bit difficult and hence a reduction approach is being used to study them differently.

Sites where oral microbiota is in abundance are the interproximal areas and the gingival crevices. Pits and fissures are sites of largest microbial communities.[6],[19],[20]

Dental plaque

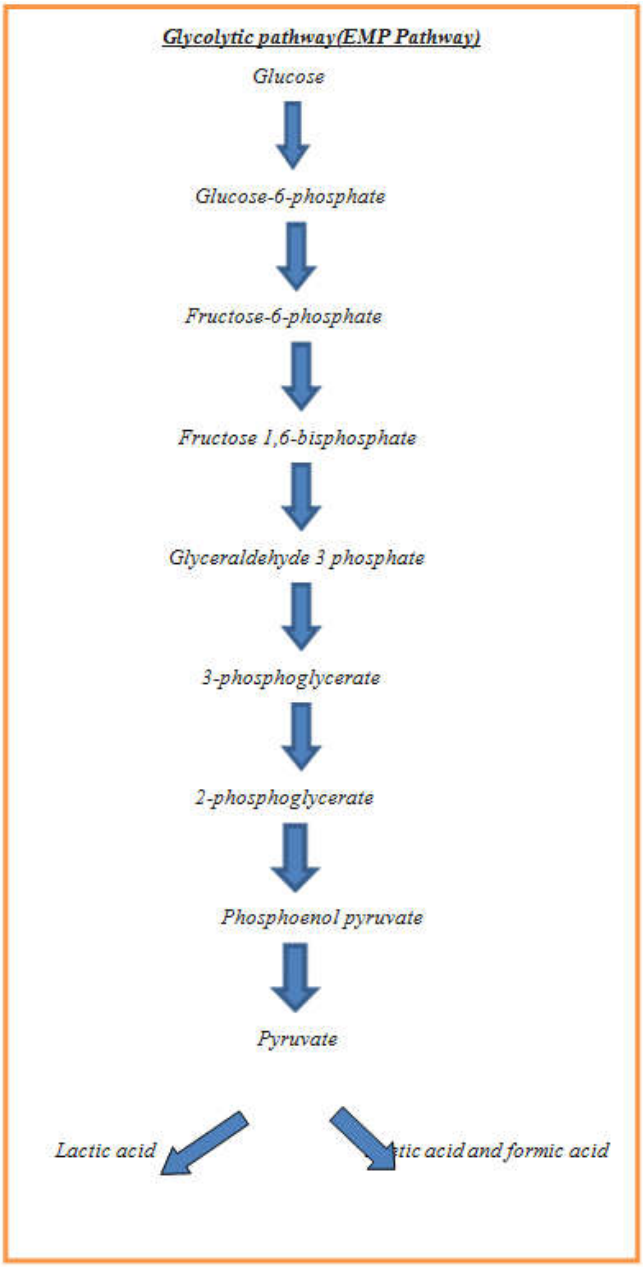
Dental plaque is a biofilm which develops not only on the tooth surface but also on dental restorations, tooth anomalies like enamel pearl, areas such as bifurcation, trifurcations, implants and prosthesis containing within the vast number of microbes. Its formation takes place within a matter of hours or days. There are stages of plaque formation as follows:

1. *Association*: Bacteria associated loosely with the pellicle by physical forces.
2. *Adhesion*: Primary colonizers like streptococcus and actinomycetes with the help of their surface molecules adhesins bind to pellicle receptors
3. *Bacterial proliferation.*
4. *Microcolonies*. Many streptococci secrete protective extracellular polysaccharides (e.g., dextrans, levans).
5. *Biofilm ("attached plaque"):* Microcolonies form complex groups with metabolic advantages for the constituents.
6. *Plaque growth—maturation*: The plaque begins to "behave" as a *complex organism*. Anaerobic organisms increase. Immune system is activated because of the metabolic end products.

These large number of microorganisms produce various metabolites which leads to dental diseases like dental caries, gingivitis and periodontitis if not prevented by oral hygiene practices such as tooth brushing and flossing.

Fact Box #1. Of Clinical revelance

Analogs must be developed to prevent the attachment of adhesins with the receptors thereby preventing the co-adhesion and similarly chemicals can be used to discourage their colonization which will act against the signals which motivate the organisms to catabolize complex glycoproteins derived from food.[5]



Biofilm formation

It is the network or meshwork formed by the microbes where food accumulation and other microbes rest. These microbes developing on hard surfaces acquire biofim phenotype which makes them different from other planktonic organisms at molecular and physiologic level.

The microbes interact with each other via their metabolic processes, exchange of nutrients and they even do chemical communication during quorum sensing and biofim formation.[20]

Microflora of Infants

Infants have a sterile oral cavity but later they are transferred with microbes through milk, saliva and external environment. The oral cavity of infants is highly selective and only few organisms are in common with the microbes of adult flora. As soon as microflora develops in the oral cavity of infants, lactobacilli and candida are first to colonise. Predominant species in the infants are streptococci i.e; *S. mitis*, *S. oralis* and *S. salivarius*.[19-23]

Probiotics

Oral microbes such as streptococci act as potential probiotics. Their role is to provide inhibition or competition to pathogens. They also modulate the mucosal immune system and sometimes can enhance the growth of beneficiary organisms.[20]

Functions of the resident oral microflora in health

Resident microflora plays important role in defence system. It acts against pathogens and maintains health. Bacteriocin or salivaricin produced by *Streptococcus salivarius* acts against many Lancefield group A streptococci. These byproducts produced by the resident microflora act against pathogens. Other byproducts like hydrogen peroxide, volatile fatty acids tend to change the local environment (pH, redox potential) thereby reducing the quantity of opportunistic pathogens. One such example is inhibition of dental plaque formation by A. actinomycetemcomitans because of hydrogen peroxide formation by Streptococcus mitis. Similarly, *Fusobacterium nucleatum* is present in the gingival crevicular fluid in both health as well as in disease and thus are considered as opportunist pathogen. However, *F.nucleatum* in addition with other organisms such as *Streptococcus gordoni* favours colonization of more pathogenic organisms such as *Porphyromonas gingivalis* and plays a role in initiation and progression of chronic periodontitis.[19],[24-26]

Ecological niches

It is referred to as the separate function performed by each microrganisms in the habitat including the organisms of supragingival plaque, subgingival plaque and tongue coating. Tooth surfaces are coated with salivary components such as proteins and glycoproteins.

Supragingival microrganisms are sacharolytic and utilize carbohydrates derived from food and produce lactic, acetic, succinic and other organic acids through EMP (Embden-Meyerhoff Parnas) pathway. The acidic environment not only increases the acidurance (ability to survive in acidic environment) of these organisms but also the acidogenicity (ability to produce acid). Though primarily brought by mutans streptococci but some strains of non-mutans streptococci (e.g. *S. mitis* biovar1 and *S. oralis*) can still metabolize sugars to acid at a moderately low environmental pH at rates comparable to those achieved by mutans streptococci. *Streptococcus mutans* upregulates specific proteins under acidic conditions as encountered during carious disease which enhances their survival. Preventive measures must be targeted at inhibiting plaque acid production by fluoride containing products which acts against enzymes involved in glycolytic pathway. Meals and drinks containing fermentable sugars between main meals should be avoided. Saliva flow should be stimulated by chewing sugar free gum.[27,5]

Microbial Shift

Non mutans streptococci and actinomyces are the pioneers or initiators to create acidic environment and adapt to acidic environment hence producing cariogenicity. It is in this acidic environment that the microbial shift occurs wherein mutans streptococcus colonize, adapt and promote the process of caries activity.

Subgingival microflora

It has a stable tooth surface and an unstable epithelial surface. As the gingival crevices deepens it creates a more neutral and anaerobic environment. These microrganisms are asacharolytic/ proteolytic and anaerobic such as *Fusobacterium, Eubacterium, Campylobacter, Prevotella and Porphyromonas*. Gingival crevices has gingival crevicular fluid which is derived from blood plasma and rich in nitrogenous compounds such as aminoacids, peptides and proteins.

Proteolytic organisms have cell bound or extracellular proteases to breakdown these nitrogenous compounds into aminoacids and peptides which are later used as metabolic substrates. Proteases degrade the host tissues and host defense system by attacking complements, immunoglobulin and blood coagulation system. Metabolic end products such as short chain fatty acids (propionic acid, butyric acid), aminoacids and sulfur compounds(H_2S and mercaptan) cause damage to host tissue and host defence system.[27]

Microbial shift

Firstly the *F.nucleatum and P.intermedia* colonize in a shallow gingival pocket where the pH is variable and sometimes acidic but later these organisms creates a neutral environment. Bacterial colonization promotes inflammation and thus increased production of GCF gingival crevicular fluid rich in nitrogenous compounds as a result of which colonization of more proteolytic and anaerobic organisms like *P.gingivalis* occurs which also induces pathogenicity of *P.intermedia*.[27]

Tongue coating and oral malodour

A wide range of bacteria including *Actinomyces, Streptococci, Veillonella, Fusobacterium* and *Prevotella* are found and the tongue may act as a reservoir or cradle of oral bacteria.

Bacterial metabolism of sulfur amino acids produces foul smelling volatile sulfur compounds(VSC's). VSC's are mainly H_2S, methylmercaptan and dimethylsulfide.[27]

> *Fact Box # 2*
> *Organisms causing malodour associated with periodontal diseases :* ***Porphorymonas and Prevotella***
> *Organisms causing malodour not associated with periodontal disease:* ***Veillonella and Actinomyces.***

Factors affecting growth of oral microorganisms

Neutral pH: Normal healthy pH of gingival crevice is 6.9 but in diseased state it reaches to 7.2-7.4, this change in pH leads to altered gene expression which favours growth of anaerobic organisms such as *P.gingivalis.*

- **Urea** can significantly increase the pH of the microcosm which can decrease the incidence of dental caries**.**

- **Bioactive glasses** which are the replacement of bone or bone substitiutes when in contact with saliva for a long time can liberate ions such as $Ca^{2+,}$ Na+, PO_4^{3-} and Si^{4+} causing rich in pH of the saliva and hence acts on the dentogingival margins against organisms such as *Actinobacillus actinomycetemcomitans, P. gingivalis, Actinomyces naeslundii, S. mutans,* and *Streptococcus sanguis*. This property of bioactive glasses is being utilised in the production of toothpastes.
- **Diet** has a major role in influencing the microbial community
- Growth of some organism affect the ecology by generating oxidizing and reducing conditions hence creating an anaerobic environment.[20]

Our defence system

Saliva has a buffering action against acid produced as a result of metabolism of carbohydrates by microorganisms. Buffering is produced with the help of carbonates, phosphates, peptides and proteins.

Antimicrobial*:* Mucin, lactoferrin, lysozyme, histidine rich proteins and sialo peroxidase have antimicrobial action.

Innate and humoral immunity*:* Viral activity is checked by immunoglobulins (IgA and IgG) Peptides- Such as cystatins and defensins are histidine rich polypeptide.

Indigenous Microbiota*:* Act against any external pathogen as in the case of *Pseudomonas aeurogenosa* attempt for biofilm formation is prohibited by oral microflora.

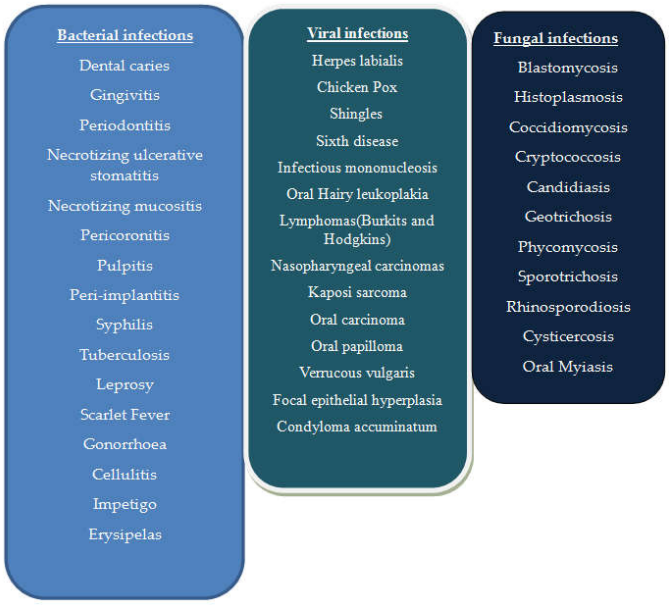
Oral Mucosa

1. Continuous exfoliation of oral mucosal epithelium prevents the growth of microbes.
2. Human β-defensins1(H β-D1) and human β-defensins2 (H β-D2) from epithelial cells along with chemokines attract monocytes and neutrophils activating the innate immune system.
3. Acquired immune system includes immunoglobulins, Langerhans cells and intraepithelial lymphocytes.
4. Stratum granulosum secretes membrane coating granules which has its role in inhibiting microbial deposition.
5. Intraepithelial dendritic Langerhans cells are antigen presenting cells which present the antigen complexed with the MHC Class II to helper T cells.

Gingival Crevicular Fluid*:* GCF increases by 30 fold.[20]

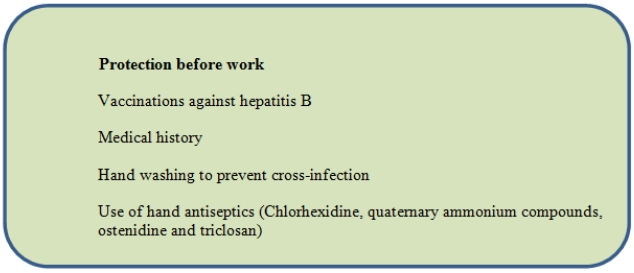
How microflora can be of help?
Useful in diagnosis (Diagnostic Indicators): Few microbes such as *Capnocytophaga gingivalis, Prevotela melaninogenica* and *Streptococcus mitis* have been found in patients suffering from OSCC.[20]

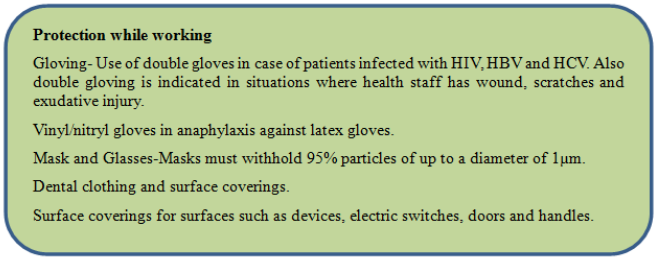
Oral Microbial Infections*[28]*



Sterilization and cross-infection control in the dental practice*[29]*

It is imperative for dental health care facility to follow the basic principles of cross-contamination barriers and infection control measures bacterial cell to cell attachment provides synergistic effect on its pathogenicity. Preventive steps such as development of analogs which can prevent binding of adhesins to receptor or chemically treatment of tooth surfaces so that it becomes less conducive to microbial colonization. Antimicrobial peptides are recognized as antimicrobial however that is not just its role but its other functions are complex and multifunctional as it has to do with the immune functions.





Cleaning and sterilization of dental instruments

Step 1: Chemical disinfection.
Step 2: Mechanical wash in machine washer.
Step 3: Autoclaving

Sterilization of hand pieces and Burs
Hand pieces

Step 1: Operate for 5-10 seconds after use.
Step 2: Wash and brush it under running water.

Step 3: Dry it with absorbent paper.
Step 4: Handpiece is reconnected with tubes and operated for 3-5 seconds.
Step 5: Handpiece is lubricated and kept in a special pouch.
Step 6: Sterilization- Autoclaving 121°C for 20 minutes/127°C for 13 minutes/134°C for 3 minutes.
Step 7: Re-lubrication.

Sterilization of Burs

Step 1: Meticulous cleaning
Step 2: Ultrasonic devices (Baths) with suitable fluids with the addition of enzymes with proteolytic action. At 60°C for 60-80 khz for 15 minutes.
Step 3: Dried.
Step 4: Sterilization – Dry heat oven sterilization, chemical vapour sterilization and ethylene oxide sterilization are preferred.

Preparing impression for the lab

Step 1: Wash the tray to remove blood, mucous and saliva.
Step 2: Spray suitable disinfectant.
Step 3: Keep the impression in a suitable plastic box.

CONCLUSION

Oral microflora contains predominantly bacteria. These bacteria attempt to colonize at every possible location inside the oral cavity. Colonization leads to biofilm formation and this microbial meshwork has specific members assigned for different location and for different time frame. Plaque formation remains stable for a period of time exhibiting what is known as microbial homeostasis.

Inhibition of dental caries can be brought about by strategies aimed to prevent biofilm formation by intervening at cell to cell adhesion and their signalling mechanism. Caries can be significantly lowered if pH changes after sugar metabolism be reduced. One must note that from the whole oral microbiota only a few are actively involved in the disease as demonstrated in the resident plaque microflora samples and this phenomenon states the specific plaque hypothesis.

Conflict of Interest

There is no conflict of interest.

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