Oral microflora in health and disease conditions – A review

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Abstract
Caries in the tooth and periodontal related diseases which are oral diseases, has to be taken account as a result of consequences in imbalances of oral microbial biofilms which are ecologically driven. Classic microbial pathogens are not the cause for the two diseases rather it is the work by micro-organisms from the residing oral micro flora which are the causative factors. Microbial fermentation of carbohydrates at low PH can stimulate the population growth of strotococci and lactobacilli strains which are acid resistant and cause acid formation leading to deminilarization of hard tissue portion of tooth and hence caries. Periodontal diseases are the result of altered equilibrium of plaque community involving mixed anaerobic micro-organisms, hence inducing inflammation. An increase in the nutrition accompanied by PH raise and raised gingival crevicular flow stimulates peridontal pathogen growth and hence may leads to destruction of the periodontium.

Keywords: Oral Cavity, Microflora, Health, Disease.

Introduction
Disease and health have always held the attention of the human mind. In simple language, disease is opposite of health, i.e. what is not healthy is disease. But the terms health and disease are difficult to define. WHO defines health as “Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity.”

The study of Microorganisms which inhibit in the skin, mucous membrane of healthy oral cavity and GIT and the interaction of oral microorganisms with the other and host population of microorganisms is done in oral
microbiology. Majority of the indigenous normal microflora in the mouth are uncultivable.²

Staphylococci, Streptococci, Lactobacilli and Coryne bacteria are the common bacteria’s seen in oral cavity along with different anaerobes particularly bacteroides. Candida albicans are commonly found fungal species apart from few other normal commensals. There is presence of viruses during their infections or in their asymptomatic carrier stages. Periodontal lesions may also show presence of few protozoans. Microorganisms get their intrinsic nutrition from the materials around the tooth, gingival crevicular fluid, pus cells, degraded epithelial cells and the salivary components. There are 18 free amino acids found in saliva like, tryptophan, tyrosine, aspartic acid, threonine, glutamic acid, alanine, serine, phenylalanine, leucine, isoleucine, cystine, proline, valine, arginine, methionine, histidine, glycine and lysine. People with caries have more growth of Streptococcus mutans(type c) in their saliva than people without caries and this difference is influenced by some proteins in saliva from caries free mouth.³,⁴

The right time for the studies on indigenous oral flora in humans should begin at newborn since at that time oral cavity is exposed to millions of microorganism from which only a small number of them become included in normal microflora.⁶

The baby may have an oral cavity which is sterile or it might contain different microorganisms like gram positive rods, streptococci, coliform bacilli and staphylococci and the source of these bacteria depends on environment they are exposed to. In a later study it was found that the organism seen within 18 hours after birth was similar to seen in the mothers.⁷ Thus Mother can directly transfer S. salivarius to the infant.

Only after the eruption of teeth does the appearance of S. sanguis was demonstrated, whereas S. mutans wasn't isolated in the first year.⁸,⁹ There have been reports of isolation of S. mutans at the primary dentition period by the time of eruption of molars. Recently it was established that S. mutans could be seen in case of predental infants who have acrylate obturators for cleft palate and infants with primary incisors with Serotype being most common of S. mutans which were isolated.¹⁰,¹¹ The factors that hinder the qualitative and quantitative relationship of microorganisms in the oral cavity are, the type of the diet, the appearance of the dentition, the subject’s oral hygiene practices, the loss of the dentition, the degree of health & disease and the use of artificial dentures.

Biofilms have been defined by Costerton et al¹² as embedded microbial matrix populations which adhere to one another or/and to interfaces or surfaces. The bacteria that accumulate on surfaces with their extracellular products and the plaques forming on the surface of tooth form the biofilm¹³. These biofilms are stubbornly resistant to the action of antimicrobials¹⁴. They are made of channels traversing depths and highly structured creating circulatory system which is primitive.¹⁵

Studies done by FISH technique on plaque that develops on materials in periodontal pockets which are placed deep and are removable concluded that Gram-negative bacteria and spirochaetes colonized the deepest zones and Gram-positive cocci were found in shallower areas¹². Inflammation of margins of gingiva increased the chances of plaque formation in terms of thickness and surface of tooth covered as well. However the proper mechanism is still not clear. It's suggested that (i) The shelter for growing plaques is provided by inflammatory oedema of margins of gingiva. (ii) The inflammation produces enhanced gingival crevicular fluid which supply nutrients in excess for the bacteria which form the plaque.¹⁶
Phases of plaque formation

Formation of biofilms is a step-by-step process which commences by adhesion of planktonic microorganisms to a surface. Later steps involve co-adhesion, colonization, colonization, and growth, and finally detachment of few microorganisms.17

In the gingival crevice18

<table>
<thead>
<tr>
<th>Group</th>
<th>Genera and/or species commonly found</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gram - positive facultative cocci (28.8 %)</td>
<td>Staphylococci</td>
</tr>
<tr>
<td></td>
<td>Enterococci</td>
</tr>
<tr>
<td></td>
<td>Streptococcus mutans</td>
</tr>
<tr>
<td></td>
<td>Streptococcus sanguis</td>
</tr>
<tr>
<td></td>
<td>Streptococcus mitis</td>
</tr>
<tr>
<td></td>
<td>Peptostreptococcus</td>
</tr>
<tr>
<td>Gram - positive anaerobic cocci (7.4%)</td>
<td>Corynebacterium</td>
</tr>
<tr>
<td></td>
<td>Lactobacillus</td>
</tr>
<tr>
<td></td>
<td>Nocardia</td>
</tr>
<tr>
<td></td>
<td>Odontomycesviscosus</td>
</tr>
<tr>
<td>Gram - positive facultative rods (15.3%)</td>
<td>Bacterionemamatomachotii</td>
</tr>
<tr>
<td>Microbial Group</td>
<td>Bacterial Species</td>
</tr>
<tr>
<td>---------------------------------------</td>
<td>--------------------------------------------------------</td>
</tr>
<tr>
<td>Gram - positive anaerobic rods (20.2%)</td>
<td>Actinomycesbifidus, Actinomycesisraelii, Actinomycesnaeslundii, Actinomycesodontolyticus, Propionibacterium acnes, Leptotrichiabuccalis, Corynebacterium</td>
</tr>
<tr>
<td>Gram - negative facultative cocci (0.4%)</td>
<td>Neisseria</td>
</tr>
<tr>
<td>Gram - negative anaerobic cocci (10.7%)</td>
<td>Veillonellalaccaesens, Veillonellaparvula</td>
</tr>
<tr>
<td>Gram - negative facultative rods (1.2%)</td>
<td>--</td>
</tr>
<tr>
<td>Gram - negative anaerobic rods (16.1%)</td>
<td>Bacteroidesmelanogenicus, Bacteroidesoralis, Vibrio sputorum, Fusobacteriumnucleatum, Selenomonassputigena</td>
</tr>
<tr>
<td>Spiral organisms (1 to 3)</td>
<td>Treponemadenticola, Treponemaoralis, Treponemamacrodentium, Borelliavincenti</td>
</tr>
</tbody>
</table>

**Tongue microflora**

<table>
<thead>
<tr>
<th>Microbial Group</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Facultative streptococci</td>
<td>38.3%</td>
</tr>
<tr>
<td>Facultative diphtheroids</td>
<td>13.0%</td>
</tr>
<tr>
<td>Micrococci-staphylococci</td>
<td>6.5%</td>
</tr>
<tr>
<td>Fusobacterium</td>
<td>0.8%</td>
</tr>
<tr>
<td>Vibrio</td>
<td>2.1%</td>
</tr>
<tr>
<td>Bacteroides</td>
<td>5.3%</td>
</tr>
<tr>
<td>Unidentifiable gram-negative cocci</td>
<td>2.6%</td>
</tr>
</tbody>
</table>
Anaerobic diphtheroids 7.4%
Veilonella 14.5%
Peptostreptococcus-peptococcus 4.2%
Gram-negative rods Unidentifiable 3.2%
Neisseria 2.3%

Approximate proportional distribution of bacteria on various oral surfaces

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>Gingival crevice</th>
<th>Coronal plaque</th>
<th>Tongue dorsum</th>
<th>Buccal mucosa</th>
<th>saliva</th>
</tr>
</thead>
<tbody>
<tr>
<td>Streptococcus salivarius</td>
<td>&lt;0.5</td>
<td>&lt;0.5</td>
<td>20</td>
<td>11</td>
<td>20</td>
</tr>
<tr>
<td>Streptococcus mitis</td>
<td>8</td>
<td>15</td>
<td>8</td>
<td>60</td>
<td>20</td>
</tr>
<tr>
<td>Streptococcus sanguis</td>
<td>8</td>
<td>15</td>
<td>4</td>
<td>11</td>
<td>8</td>
</tr>
<tr>
<td>Streptococcus mutans</td>
<td>?</td>
<td>0-50</td>
<td>&lt;1</td>
<td>&lt;1</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Enterococci</td>
<td>0-10</td>
<td>&lt;0.1</td>
<td>&lt;0.01</td>
<td>&lt;0.1</td>
<td>&lt;0.1</td>
</tr>
<tr>
<td>Gram positive filaments</td>
<td>35</td>
<td>42</td>
<td>20</td>
<td>?</td>
<td>15</td>
</tr>
<tr>
<td>Lactobacilli</td>
<td>&lt;1</td>
<td>&lt;0.005</td>
<td>&lt;0.1</td>
<td>&lt;0.1</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Veillonella spp.</td>
<td>10</td>
<td>2</td>
<td>12</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>Neisseria spp.</td>
<td>&lt;0.5</td>
<td>&lt;0.5</td>
<td>&lt;0.5</td>
<td>&lt;0.5</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Bacteroidesoralis</td>
<td>5</td>
<td>5</td>
<td>4</td>
<td>?</td>
<td>?</td>
</tr>
<tr>
<td>Bacteroidesmelanogenicus</td>
<td>6</td>
<td>&lt;1</td>
<td>&lt;1</td>
<td>&lt;1</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Vibrio sputorum</td>
<td>5</td>
<td>1</td>
<td>&lt;0.5</td>
<td>&lt;0.5</td>
<td>?</td>
</tr>
<tr>
<td>Spirochetes</td>
<td>2</td>
<td>&lt;0.1</td>
<td>&lt;0.1</td>
<td>&lt;0.1</td>
<td>&lt;0.1</td>
</tr>
<tr>
<td>Fusobacterium spp.</td>
<td>3</td>
<td>4</td>
<td>1</td>
<td>?</td>
<td>&lt;1</td>
</tr>
</tbody>
</table>

Changes in microbial flora in various conditions

During Menstrual Cycle: Changes in the levels of female sex hormones during the menstrual cycle may cause cyclic differences in subgingival bacterial colonization patterns.

During Menstrual Cycle: Cyclic differences could be seen in subgingival bacterial colonization patterns due to change in levels of the sex hormones while active menstrual cycle.

Orodental Infections:
In Orodental infections like dental caries, gingivitis and periodontitis anaerobic lactobacilli and Streptococcus mutans were high in dental caries, Peptostreptococcus and Actinomyces common in gingivitis, Porphyromonas gingivalis and Actinobacillus actinomycetem comitans were common in periodontitis. Streptococcus sobrinus and Streptococcus mutans and lactobacilli were main etiologic agents of dental caries in supragingival area, but during root-surface caries, Actinomyces were involved. Low lactobacilli levels in saliva, high plaque levels of Streptococcus sanguinis and reduced numbers of streptococci mutans in plaque were seen in Oral cavities with healthy dentitions.

Periodontal Disease: The chronic bacterial infections namely periodontitis and gingivitis have host bacterial
interactions similar to other infections which determine
the extent and the nature of the resultant disease, indirect
disease may be caused by pathogenic organisms by
producing toxins and tissue invasion or host response
stimulation.

**Changes Seen With Malignancy:** There may be systemic
as well as local infections when changes of microflora on
the surface of oral canicomas occur leading to
complications in the morbidity of patients having
malignant neoplasms in the mouth.

Anticancer treatment, chemotherapy, radiotherapy or surgery
can impair defence mechanism of oral mucosa along with
mucosal biofilm proliferation and yeast and bacterial
overgrowth.²¹

**Discussion and Conclusion**

There is omnipresence of microbial communities in the
nature specially present on surfaces as organized biofilms.
There is a diversity in phenotypes and genotypes (even in
mono species biofilms of P. aeruginosa) within the
biofilms produced by heterogeneity in environment
within biofilms. In case of any adverse conditions faced
these diversities act as biological guards that gives safety
to the community of microorganisms. These diversities
can hinder several important properties found in the cell
like nutritional requirements, detachment, secretion of
products and formation of biofilms and enhances the
survival of community or organisms during environmental
pressure and stress.

Dental plaque shows properties more than the sum of
properties shown by its constituent members and sets up a
good example for microbial community and biofilm. The
functions like horizontal gene transfer, reduced
susceptibility to antimicrobial agents, organism
communication via cell–cell signaling strategies and gene
expressions may be indirectly or directly effected by
biofilm. Approaches which are independent of culture are
showing the diversity of micro flora from sites of disease
and health and proving that even fastidious
microorganisms can grow in environment which is
heterogenous provided by the biofilms. These studies
provide a huge boost in defining and challenging the
current practices of diagnosis and treatment by trying to
enlighten the etiological importance of microbial in
diseases which are plaque mediated. Newer specifications
and prospectus would be required to be defined which is
away from the older concepts of conventional infections
with specific and simple etiology if we wholly understand
relationship between host and plaque bacteria in disease
and health and develop more stronger control strategies.¹²

**References**

1. International Health Conference, New York, 19-22
   June, 1946 (Official Records of the World Health
   Organization, no. 2, p. 100).
2. Ruby J and Goldner M. Nature of Symbiosis in Oral
3. McCarthy C, Snyder M.L. and Parker R.B. The
   indigenous oral flora of man-The newborn to the 1-
   Specificity of utilization of human salivary proteins
   in saliva from caries active and caries free subjects as
6. Long SS and Swenson RM. Determinants of the
developing oral flora in normal newborns. Applied