

**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 demineralization 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<sup>5</sup>

**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.”<sup>1</sup>

The study of Microorganisms which inhabit 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 micro flora in the mouth are uncultivable.<sup>2</sup>

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.<sup>4,5</sup>

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.<sup>6</sup>

The baby may have an oral cavity which is sterile or it might contain different micro organisms 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<sup>7</sup>. Thus Mother can directly transfer S. salivarius to the infant.

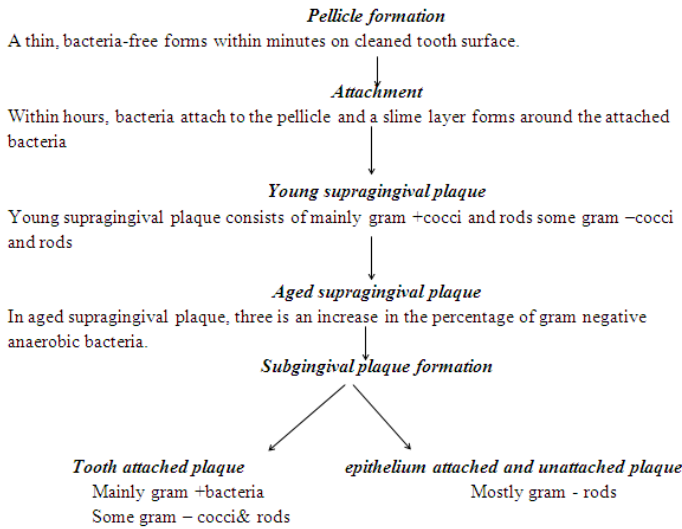
Only after the eruption of teeth does the appearance of S. sanguis was demonstrated, whereas S. mutans wasn't

isolated in the first year.<sup>8,9</sup> 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 pre-dental infants who have acrylic obturators for cleft palate and infants with primary incisors with Serotype being most common of S. mutans which were isolated.<sup>10,11</sup> 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 hygienic practices, the loss of the dentition, the degree of health & disease and the use of artificial dentures.

Biofilms have been defined by Costerton et al<sup>12</sup> 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 biofilm<sup>13</sup>. These biofilms are stubbornly resistant to the action of antimicrobials<sup>14</sup>. They are made of channels traversing depths and highly structured creating circulatory system which is primitive.<sup>15</sup>

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 areas<sup>12</sup>. 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 is 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 supplies nutrients in excess for the bacteria which form the plaque.<sup>16</sup>

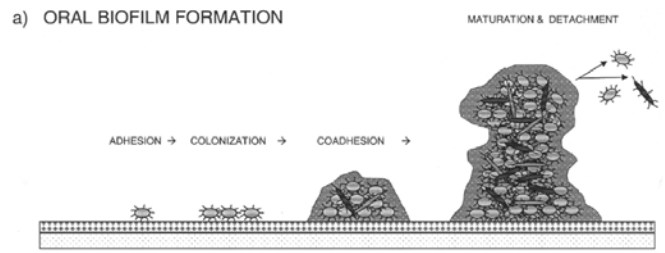
## Phases of plaque formation



Formation of biofilms is step by step process which commences by adhesion of plank tonic micro-organisms to a surface. Later steps involve co-adhesion, colonization, colonization an f growth and lastly detachment of few micro-organisms.<sup>17</sup>

### In the gingival crevice<sup>18</sup>

Group	Genera and/or species commonly found
Gram - positive facultative cocci (28.8 %)	Staphylococci Enterococci Streptococcus mutans Streptococcus sanguis Streptococcus mitis
Gram - positive anaerobic cocci (7.4% )	Peptostreptococcus
Gram - positive facultative rods (15.3% )	Corynebacterium Lactobacillus Nocardia Odontomycesviscosus Bacterionemamatruchotii



### b) PROSPECTS FOR FUTURE INTERVENTION

**TARGETS:**

Surface, Adhesion, Colonization, Coadhesion, Metabolism, Growth, Adaptation, Maturation, Climax community, Detachment

**STRATEGIES:**

Surface modification, Immunization, Replacement therapy, Two-component and quorum-sensing system interference

### Biofilm regulation of gene expression

They have vital roles to play during signal transduction and during adaptation to biofilm life they act as essential sensors. Two-component systems example for biofilm formation include the GacA/GacS in *P. aeruginosa* and the HK11/RR11 in *S. mutans*, as well as the ComD/ComE in *S. mutans* and *S. gordonii*. There are variations in microbial flora in different surfaces in the mouth. The various microfloras are:

Gram - positive anaerobic rods (20.2%)	Actinomycesbifidus
	Actinomycesisraelii
	Actinomycesnaeslundii
	Actinomycesodontolyticus
	Propionibacterium acnes
	Leptotrichiabuccalis
Gram -negative facultative cocci (0.4%)	Corynebacterium
	Neisseria
	Veillonellaalcalescens
Gram -negative anaerobic cocci (10.7%)	Veillonellaparvula
Gram -negative facultative rods (1.2%)	--
	Bacteroidesmelanogenicus
Gram -negative anaerobic rods (16.1%)	Bacteroidesoralis
	Vibrio sputorum
	Fusobacteriumnucleatum
	Selenomonassputigena
	Treponemadenticola
Spiral organisms (1 to 3)	Treponemaoralis
	Treponemamacrodentium
	Borelliaivinceti

### Tongue microflora

Facultative streptococci	38.3%
Facultative diphtheroids	13.0%
Micrococci-staphylococci	6.5%
Fusobacterium	0.8%
Vibrio	2.1%
Bacteroides	5.3%
Unidentifiable gram-negative cocci	2.6%

Anaerobic diphtheroids	7.4%
Veillonella	14.5%
Peptostreptococcus-peptococcus	4.2%
Gram-negative rods Unidentifiable	3.2%
Neisseria	2.3%

**Approximate proportional distribution of bacteria on various oral surfaces<sup>19</sup>**

Bacteria	Gingival crevice	Coronal plaque	Tongue dorsum	Buccal mucosa	saliva
Streptococcus salivarius	<0.5	<0.5	20	11	20
Streptococcus mitis	8	15	8	60	20
Streptococcus sanguis	8	15	4	11	8
Streptococcus mutans	?	0-50	<1	<1	<1
Enterococci	0-10	<0.1	<0.01	<0.1	<0.1
Gram positive filaments	35	42	20	?	15
Lactobacilli	<1	<0.005	<0.1	<0.1	<1
Veillonella spp.	10	2	12	1	10
Neisseria spp.	<0.5	<0.5	<0.5	<0.5	<1
Bacteroides oralis	5	5	4	?	?
Bacteroides melanogenicus	6	<1	<1	<1	<1
Vibrio sputorum	5	1	<0.5	<0.5	?
Spirochetes	2	<0.1	<0.1	<0.1	<0.1
Fusobacterium spp.	3	4	1	?	<1

**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 sub gingival bacterial colonization patterns.

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

**Oro-dental Infections:**

In Oro-dental 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<sup>137</sup>. 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.<sup>21</sup>

#### **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.<sup>12</sup>

#### **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 Disease. *J Dent Res.* 2007; 86:8-11.
3. McCarthy C, Snyder M.L. and Parker R.B. The indigenous oral flora of man-The newborn to the 1-year-old infant. *Archives of Oral Biology.* 1965;10:61-70.
4. Cowman R.A., Schaefer S.J. and Fitzgerald R.J. Specificity of utilization of human salivary proteins for growth of oral streptococci. *Caries Res.* 1979;13:181.
5. Cowman R.A. et al. Differential utilization of proteins in saliva from caries active and caries free subjects as growth substrates by plaque forming streptococci. *J.Dent.Res.* 1979;58:2019.
6. Long SS and Swenson RM. Determinants of the developing oral flora in normal newborns. *Applied and Environmental Microbiology.* Oct.1976;32:494-497.

7. Ofek I. et al. Postnatal development of binding of streptococci and lipoteichoic acid by oral mucosal cells of humans. *J. Infec. Dis.* 1977;135:268.
8. Carlsson J. Dental plaque as a source of salivary streptococci. *Odontology. Revy* 1967;18: 173.
9. Carlsson J. et al. Establishment of *Streptococcus sanguis* in the mouth of infants. *Arch. Oral Biol.* 1970;15:1413.
10. Catalanatto F.A. et al. Prevalence and location of *Streptococcus mutans* in infants and children. *International Association for Dental Research, 52<sup>nd</sup> general meeting (abstract 563), 1974.*
11. Berkovitz R.J. Jordan H.V. and White G. The early establishment of *Streptococcus mutans* in the mouth of infants. *Arch. Oral Bio.* 1975;20:171.
12. Marsh PD. Dental plaque: biological significance of a biofilm and community life-style. *JClinPeriodontol.* 2005;32:7-15.
13. Wilson M. Susceptibility of oral bacterial biofilms to antimicrobial agents. *J Med Microbiol.* 1996;44:79-87.
14. Nichols WW. Sensitivity of bacteria in biofilms to antibacterial agents. *Formation and control.* Oxford: Blackwell Scientific Publications Ltd. 1993:187-200.
15. John F. O'Neill, Christopher K. Hope, and Michael Wilson. Oral Bacteria in Multi-Species Biofilms Can be Killed by Red light in the Presence of Toluidine Blue. *Lasers in Surgery and Medicine.* 2002;31:86-90.
16. Paster B.J. et al. Bacterial diversity in human subgingival plaque. *J. Bacteriol.* 2001;183: 3770-3783.
17. Jenkinson HF and Lamont RJ. Oral microbial communities in health and disease. *Trends in Microbiology.* 2005;13:589-95.
18. Nolte WA. Oral microbiology with basic microbiology and immunology. C.V. Mosby, St. Louis, 2<sup>nd</sup> edition. 1973: chapter-3:193-200.
19. Hammond BF. Dextran production by a human oral strain of *Lactobacillus casei*. *Arch Oral Biol.* 1969;14:879-890.
20. Saini S, Gupta AN, Mahajan A, Arora DR. Microbial flora in orodental infections. *Indian Journal of Medical Microbiology.* 1993;21:111-114.
21. G. H. W. Bowden. *The Microbial Ecology of Dental Caries. Microbial Ecology in Health and Disease.* 2000;12:138-148