Review Article

Normal Microbial Flora of Oral Cavity

Swapan Majumdar[1], Ashok Bhagat Singh[2]

[1]Professor, Department of Prosthodontics
[2]Professor, Department of Oral and Maxillofacial Surgery, Mithila Minority Dental College, Darbhanga

Abstract:
Oral microbiology is the study of the microorganisms of the oral cavity and the interactions between the oral microorganisms with each other and with the host &refers to the population of microorganisms that inhabit skin and mucous membranes of normal healthy oral cavity and GIT. The mouth is inhabited by an indigenous "normal" micro flora that is composed of over 500 species—the majority still uncultivable. Certain microbial types are constantly found in the specific oral areas. These microbial types are referred to collectively as the normal, indigenous, or resident floras and constitute the oral ecosystems.

Keyword: Microorganisms, Ecosystem, Environment

Corresponding Author: Dr. Swapan Majumdar, Professor, Department of Prosthodontics, Mithila Minority Dental College, Darbhanga, India.

This article may be cited as: Majumdar S, Singh AB. Normal Microbial Flora of Oral Cavity. J Adv Med Dent Scie Res 2014;2(4):62-66.

INTRODUCTION

The normal flora can be divided into two groups Resident flora and Transient. Some organisms are almost always present in a normal oral cavity and constitute constant/normal microbial flora. It is further divided into two types: indigenous & supplemental. Comprise of those species that are almost always present in high numbers (> 1%) in a particular site, such as the supra gingival plaque, the surface of the tongue. There numerical dominance implies that they are compatible with the host and have entered into a stable relationship with the host.[1]The supplemental flora comprises the species that are nearly present but are in low numbers (<1%). These organisms may become indigenous if the environment changes.Certain microbes tend to stay for a short time in the oral cavity of a few individuals. If the resident flora is intact, there is very little significance of the transient flora. However if the normal flora is disturbed, the transient flora may proliferate and produce disease. In addition to the normal & transient micro floras, a third or intermediate group, the supplemental (normal) flora has been designated it represents those microorganisms that may be detected in a significant percentage of individuals. Persons harboring supplemental normal flora generally possess them in moderate numbers & for long periods of time.[2]

SALIVARY MICROFLORA

Dislodgment of microorganisms from colonizing aggregations in various locations of oral cavity(the teeth, tongue, cheek, & pharyngeal mucous membrane) contributes to the microflora of the saliva.[4]Adult human saliva is reported to contain approximately 6 billion ($6x10^9$) microorganisms per mililitre including streptococci, peptostreptococci, Veillonella, Corynebacterium, Neisseria, Nocardia, Fusobacterium, Bacteroides, lactobacilli, Actinomyces, spirochaetes.

Table 1: Microbial flora varies on the different surfaces of the oral cavity.

In the gingival crevice[3]

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 Odontomyces viscosus Bacterionema matruchotii
Gram - positive anaerobic rods (20.2%)	Actinomyces bifidus Actinomyces israelii Actinomyces naeslundii Actinomyces odontolyticus Propionibacterium acnes Leptotrichia buccalis Corynebacterium
Gram -negative facultative cocci (0.4%)	Neisseria
Gram -negative anaerobic cocci (10.7%)	Veillonella alcalescens Veillonella parvula
Gram -negative facultative rods (1.2%)	--
Gram -negative anaerobic rods (16.1%)	Bacteroides melanogenicus Bacteroides oralis Vibrio sputorum Fusobacterium nucleatum Selenomonas sputigena
Spiral organisms (1 to 3)	Treponema denticola Treponema oralis Treponema macrodentium Borellia vincenti

Tongue Microflora

Facultative streptococci	38.3%
Veilonella	14.5%
Facultative diphtheroids	13.0%
Anaerobic diphtheroids	7.4%
Micrococci-staphylococci	6.5%
Bacteroides	5.3%
Peptostreptococcus-peptococcus	4.2%
Neisseria	2.3%
Vibrio	2.1%
Fusobacterium	0.8%
Unidentifiable gram-negatetive rods	3.2%
Unidentifiable gram-negetative cocci	2.6%

Table 2: Approximate proportional distribution of bacteria on various oral surfaces

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	6	<1	<1	<1	<1
melanogenicus	5	1	<0.5	<0.5	?
Vibrio sputorum	2	<0.1	<0.1	<0.1	<0.1
Spirochetes	3	4	1	?	<1
Fusobacterium spp.					

Yeasts, protozoa, and others. Investigations of the possible source of salivary bacteria indicates that 47% of the facultative streptococci present in saliva, 21% to 55 % of the facultative streptococci on the tongue, and 10% of the facultative streptococci on the cheek. Dental plaque is not considered to be the source of S. salivarius found in saliva. Although S.sanguis is reported to be the dominant streptococci in early plaque from teeth. It constitutes only a minor portion of the flora of other sites in the oral cavity.[4]So, dental plaque is not the major contributor to the salivary microflora. Major source appears to be the tongue.

MICROBIAL ADHERENCE AND AGGREGATION IN THE ORAL CAVITY

Theecologic relationship of microorganisms with the oral cavity is perhaps more dependent on the ability of the organisms to attach to specific oral tissues than on their association with the nutritional factors of the specific tissue or the area. Organisms that cannot attach to specific oral surfaces would be expected to be more easily removed by salivary flow and the flow of oral secretions. [3]

Recently, considerable research has been concerned with the ability of oral micro-organisms to adhere to different oral surfaces. Early work has demonstrated that S. sanguis appears to have a preference for colonizing on tooth surfaces, whereas S. salivarius has a marked affinity for epithelial surfaces and shows little affinity to adhere to teeth. It has been observed that plaque bacteria, S. sanguis, S. mitis, and Actinomyces species, but not S. mutans or S. salivarius aggregate in presence of human saliva. Also, S. sanguis and Actinomyces have been observed to adhere to saliva coated enamel, whereas lactobacilli, S. mutans and S. salivarius appear to be unaffected or demonstrate little ability to adhere to saliva coated enamel.[5] Bacteria adsorbed to saliva-coated enamel also are agglutinated by cell free saliva. This affinity between host and saliva explains a method of adherence to teeth in the oral cavity. Extracellular dextran polymers,[6,7] an outer capsular slime formed by certain microorganisms, has demonstrated a high affinity for adherence to dental enamel. Dextrans, mainly of the alpha 1.3 linkage, are insoluble and appear to be involved in initiating plaque formation and caries by localizing acidogenic-aciduric bacteria on teeth. The synthesis of extracellular polysaccharides before sorption has been shown to reduce desorption and foster a firm attachment to tooth substance.[8] Completely unrelated strains of bacteria can aggregate by cell to cell interaction.

Such interaction also explains adherence mechanisms in developing dental plaque. Salivary polymers on tooth surface mediate the attachment of S. sanguis, whereas the attachment of S. mutans is mediated mainly by its own extracellular glucans to tooth surfaces. On the other hand, S. salivarius does not appear to attach to saliva coated teeth. Recent studies have demonstrated that S. salivarius and S. mitior (mitis) exhibit morphologically different fibrillar fuzzy surface coatings, whereas S. sanguis, S. mutans and lactobacilli have less fibrillar surfaces.[8] Fibril hair like structures were observed with the electron microscope for A. naeslundii and A. viscosus. These structures appear on the outer surface, and it is suggested that they provide a mechanism or a structure for adherence of these organisms with one another and with the tooth surface.[9]

Teichoic acid has the potencial to adsorb to tooth enamel. These acids have been found in culture broth of strains of S. mutans, S. sanguis, S. mitis, and S.salivarius; in the cell wall and cell surface membrane of these streptococci; and in the strains of L. casei, L. plantarum, and Lactobacillus fermentum. Cell surface teichoic acids can aid these organisms in the adherence to tooth surfaces and in the formation of dental plaque. The adhesiveness of enamel shown by the capsular coat of S. mutans may in part be the effect of teichoic acid in this capsular coat.**[10]**

The teichoic acids at the bacterial cell surfacesmay be involved in the adsorption of the oral organisms S. pyogenes, S. salivarius, and S. mutans to oral epithelial cells and may also explain their feeble affinity for saliva treated hydroxyapatite.[11]

Many plaque bacteria are capable of hydrolyzing the extracellular levan(fructan) in vitro.**[12]**

It is suspected that this happens also in vivo because the fructan content of dental plaque from humans varies widely and may drop to levels that cannot be detected.[12] It has been observed that cariogenic S. mutans has not been readily implanted into the human mouth and also is not readily passed between members of families. Although it forms an extra cellular dextran in the presence of dietary sucrose and has an affinity to adhere to tooth enamel, it does not colonize uniformly over surfaces of teeth, nor does it appear to be easily transmitted from one tooth surface to another in the same individual. Recent studies have demonstrated that significant proportion of the human dental plaque bacterial flora produce glucan (dextran)-degradating enzymes.**[13]**

REFERENCES:

1. Ahrne S, Nobaek S, Jeppsson B, Adlerberth I, Wold AE, Molin G. The normal Lactobacillus flora of healthy human rectal and oral mucosa. J Appl Microbiol.1998; 85: 88-94.
2. Ahumada M del C, Bru E, Colloca ME, Lopez ME, Nader- MaciasME. Characterization of lactobacilli isolated from the tongue and gum. Anaerobe. 1999;5:129-135.
3. Nolte WA. Oral microbiology with basic microbiology and immunology. C.V. Mosby, St. Louis, 2nd edition. 1973: chapter-3:193-200.
4. Hamada S. and H. D. Slade. Biology, immunology, and cariogenicity of Streptococcus mutans. Microbiol. Rev.1980;44:331–384.
5. Hillman J.D., Van Houte J. and Gibbons R.J. Sorption of bacteria to human enamel powder.Arch.Oral Bio.1970;15:899.
6.
7. Gibbons R.J., and Spinell D.M. Salivary induced aggregation of plaque bacteria, Symposium on Dental Plaque

(McHough, W.D.,editor), Dendee, Scotland.1969.

8. Skobe Z., Gibbons R.J. and van Houte J. Ultrastructural differences of surface components of oral streptococci.J. Dent. Res.973; 53:242.
9. Gibbons R.J., Van Houte J. and LiljemarkW.F. Parameters that affect the adherence of Streptococcus salivarius to oral epithelial surfaces.J. Dent. Res.1972;51: 424.
10. Hamada S. and H. D. Slade. Biology, immunology, and cariogenicity of Streptococcus mutans. Microbiol. Rev.1980;44:331–384.
11. Rolla G. and Killiam M. Haemagglutination activity of plaque forming bacteria.Caries Res.1977;11:85.
12. Clark W.R.,Bamnann L. and Gibbons R.J. Comparative estimates of bacterial affinities and absorption sites on hydroxyl apatite surfaces. Infect.Immun. 1978;19:846.
13. CritchleyP. The breakdown of the carbohydrate and protein matrix of dental plaque.Caries Res.1969;3:249.
14. StaatR.H. and Schachtele C.F. Dextranase producing bacteri from human dental plaque (abstract 211).J.Dent.Res.1974; 53:107.

Source of Support: Nil
Conflict of Interest: None declared