ABSTRACT

With the respect to microbial flora, the oral cavity is one of the most densely populated sites of the human body. The environmental diversity of the oral cavity promotes the establishment of distinct microbial communities, such as supragingival plaque, subgingival plaque and tongue coating. The properties of the environment determine which microorganisms can occupy a site, while the metabolic activities of those microbial communities subsequently modify the properties of the environment. The mouth contains both distinct mucosal (lips, cheek, tongue, palate) and, uniquely, non-shedding surfaces (teeth) for microbial colonisation. Each surface harbours a diverse but characteristic microflora, the composition and metabolism of which is dictated by the biological properties of each site. The resident oral microflora develops in an orderly manner via waves of microbial succession (both autogenic and allogenic). The mouth contains both distinct mucosal (lips, cheek, tongue, palate) and, uniquely, non-shedding surfaces (teeth) for microbial colonisation. Each surface harbours a diverse but characteristic microflora, the composition and metabolism of which is dictated by the biological properties of each site. The resident oral microflora develops in an orderly manner via waves of microbial succession (both autogenic and allogenic).

KEY WORDS: Oral Micro-organisms, Dental caries, Dental plaque, Periodontal bacteria.

INTRODUCTION

The oral flora is one of the most ecologically diverse microbial populations known to man. From birth the oral cavity is exposed to many different micro-organisms present in the local & geographic environment. The diversity of microflora is due to the fact that the mouth is composed of a number of varied habitats supplied with a range of different nutrients. In dental plaque gradients develop, such as oxygen tension, pH, providing conditions suitable for the growth & coexistence of micro-organisms. In order to understand the etiology of many oral & dental diseases & to interpret the results of microbiological analyses of clinical specimens, a knowledge of the micro-organisms which comprise the resident flora of oral cavity is important.

Acquisition of the oral flora

Acquisition depends on the successive transmission of micro-organisms to the site of potential colonization. Initially, in the mouth, this is by passive contamination from the mother, from food, milk & water, & from the saliva of individuals in the close proximity to the baby.

By 24 hrs after birth the first species have become established. Streptococci, particularly S.salivarius, which binds to epithelial cells are usually the first to colonize. In first three days :- S.oralis, S.mitis. The diversity of the streptococcal microflora increases with time, so that all infants are colonized by more than one species of streptococcus by one month of age.

Table 1: Percentage viable count

<table>
<thead>
<tr>
<th>Streptococcus</th>
<th>1-3 days</th>
<th>2 weeks</th>
<th>1 month</th>
</tr>
</thead>
<tbody>
<tr>
<td>S.oralis</td>
<td>41</td>
<td>24</td>
<td>20</td>
</tr>
<tr>
<td>S.mitis</td>
<td>30</td>
<td>28</td>
<td>30</td>
</tr>
<tr>
<td>S.salivarius</td>
<td>10</td>
<td>30</td>
<td>28</td>
</tr>
<tr>
<td>S.sanguis</td>
<td>4</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>S.anginosus</td>
<td>3</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>S.gordonii</td>
<td>1</td>
<td>2</td>
<td>4</td>
</tr>
</tbody>
</table>
The diversity of the pioneer oral community increases during the first few months of life, & several gram-negative anaerobic species appear. P.melaninogenica – most frequently isolated anaerobe (76%) in infants with a mean age of 3 months. The dominant flora of the oral cavity in the child before eruption of teeth is mainly facultative in nature. With the eruption of teeth there is an increase in anaerobic forms, such as Leptotrichia, Spirochetes, Fusiform bacilli, Spiral forms & Vibrio. Complete loss of the dentition causes a reversion of the microflora to a predominantly aerobic facultative type. It has been demonstrated that microbial plaque, after removal from tooth surface, begins to regenerate with in minutes – 1 million organisms were reported to deposit on 1 cm² of clean tooth surface with in 5 minutes. There appears to be an upper limit to which the microflora may increase in the oral cavity.

Factors that control & limit the population of oral microflora

1) Flushing action of saliva : 1-2.5g of bacterial cells are swallowed each day in saliva.
2) Action of tongue, the lips, & mucous membrane of cheeks – helps to remove micro-organisms from tooth surface.
3) Desquamated exfoliated epithelial cells are shed, & with them the micro-organisms are removed & swallowed along with saliva.
4) Tissue fluids originating from sub-mucosal capillaries pass in to healthy gingival crevice & help remove micro-organisms from this area.

Micro-organisms in the oral cavity probably represent 2 phases of the growth curve: accelerated & stationary phase. After deposition, organism that become firmly attached to tooth enamel, should multiply & form communities of microcolonies. When the micro-organisms have increased to a certain population density, competition for nutrients between micro-colonies of the same & different organisms may result in retardation of cell division & in the death of some cells & thus ultimately cause decline of growth.

Distribution of oral microflora

The population making up the resident microflora community of the oral cavity are not uniformly distributed throughout the mouth.

Lip & palate
The lips form the border between the skin microflora (predominantly staph, micrococi, corny bacterium) & the mouth. Facultative anaerobic streptococci comprise a large part of the microflora on the lips. S.vestibularis is recovered most commonly from the gutter between lower cheeks. Desquamated exfoliated epithelial cells are shed, & with them the micro-organisms are removed & swallowed along with saliva. Micro-organisms in the oral cavity probably represent 2 phases of the growth curve: accelerated & stationary phase. After deposition, organism that become firmly attached to tooth enamel, should multiply & form communities of microcolonies. When the micro-organisms have increased to a certain population density, competition for nutrients between micro-colonies of the same & different organisms may result in retardation of cell division & in the death of some cells & thus ultimately cause decline of growth.

Distribution of oral microflora

The population making up the resident microflora community of the oral cavity are not uniformly distributed throughout the mouth.

Lip & palate

The lips form the border between the skin microflora (predominantly staph, micrococi, corny bacterium) & the mouth. Facultative anaerobic streptococci comprise a large part of the microflora on the lips. S.vestibularis is recovered most commonly from the gutter between lower lip & gums. Candida albicans can colonize damaged lip mucosal surfaces in the corner of the mouth (angular cheilitis).

Palate: majority of bacteria are streptococci, actinomyces, neisseria, veillonella.

Candida are not regularly isolated from the normal palate although their prevalence does increase if dentures are worn, & the mucosa is infected with C.albicans.

Cheek
Streptococci are the predominant bacteria from the cheek, especially S.mitis. Spirochaetes & other motile organisms have been occasionally attached to buccal mucosa. Haemophili are commonly isolated in relatively large numbers.

### Table 2: Other isolated species were

<table>
<thead>
<tr>
<th>Micro-organism</th>
<th>Occasionally found</th>
<th>Usually found</th>
</tr>
</thead>
<tbody>
<tr>
<td>Veillonella</td>
<td>&lt; 2 days</td>
<td>After 1 week</td>
</tr>
<tr>
<td>Fusiform bacilli</td>
<td>&lt; 2 months</td>
<td>Before eruption of incisors, appear to increase during 4th &amp; 8th month</td>
</tr>
<tr>
<td>Peptostreptococcus anaerobicus</td>
<td>---</td>
<td>&gt; 5 months</td>
</tr>
</tbody>
</table>

Anaerobic forms generally reappear with the wearing of dentures.

e.g.–S.sanguis & S.mutans disappear in edentulous mouth but reappear with wearing of dentures.

### Table 3

<table>
<thead>
<tr>
<th>After eruption of teeth</th>
<th>S.sanguis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary dentition, when molars erupt</td>
<td>S.mutans</td>
</tr>
<tr>
<td>Predentate infants with acrylate cleft palate obturators</td>
<td>S.Mutans</td>
</tr>
</tbody>
</table>

### Table 4:

<table>
<thead>
<tr>
<th>Bacterial type present</th>
<th>Before eruption of teeth</th>
<th>After eruption of teeth</th>
<th>Edentulous mouth</th>
<th>On wearing dentures</th>
<th>Neglected/diseased mouth</th>
<th>Well kept mouth</th>
<th>Healthy individual age 70 yrs/over</th>
<th>Healthy individual age 80 yrs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Facultative</td>
<td></td>
<td>Increase in anaerobic forms</td>
<td>Aerobic facultative (reversion of the microflora)</td>
<td>Anaerobic forms reappear</td>
<td>Anaerobic &amp; proteolytic</td>
<td>Aerobic &amp; facultative</td>
<td>Lactobacilli ; Staphlycocci</td>
<td>Yeast</td>
</tr>
</tbody>
</table>
Tongue
The dorsum of tongue, with its highly papillated surface, has a large surface area & therefore supports a higher bacterial density than other mucosal surfaces (100 bacteria / tongue epithelial cell). Streptococci are the predominant bacteria – 40% of the cultivable microflora. (S.salivarius & mitis group predominating). Stomatococcus mucilaginous – found exclusively on tongue. Veillonella : 16% Gram-positive rods : 16% Neissera : 20%

Saliva
Although saliva contains up to $10^8$ micro-organisms, it is not considered to have a resident microflora. Swallowing ensures that bacteria cannot be maintained in the mouth by multiplication in the mouth. The organisms found are derived from GCF flow, chewing, & oral hygiene but the tongue is the major source. Levels of streptococci / lactobacilli – is an indicator of the caries susceptibility of an individual.

Teeth
The microbial community associated with teeth varies in composition at each tooth surface due to differences in the local environmental conditions. S.mutans, S.mitis, S.anginosus – found in highest number on teeth as have strong affinity for hard surfaces. Gingival crevice debris develops in an environment (free from saliva). Supragingival plaque growing at the gingival margin of gingival fluid, epithelial cells & blocks entrance of saliva into the crevice. This environment would tend to encourage the establishment of anaerobic bacteria & the formation of the calculus.

Factors influencing bacterial adherence in oral cavity

Host factors
1) Mucoosal cell surfaces contain salic acid which binds to streptococci mitis.
2) Enamel pellicle consists of albumin, lysozyme, immunoglobulins, proline -rich peptides & mucins. These act as receptors for oral bacteria.
3) Acidic proline-rich proteins: These promote the adherence of Actinomyces spp, S.mutans & black pigmented anaerobes.
4) Minerals, such as Calcium & phosphate, may influence the rate of calculus formation & provide larger surface areas for plaque to accumulate.
5) Many bacterial adhesins are lectins which bind to carbohydrate receptors on a surface.

Saliva
1) Mechanical washing action helps prevent microbial overgrowth
2) Salivary enzymes may inhibit bacteria. Example: Lysozyme – destroys bacterial cell walls.
3) Buffering capacity stabilizes pH to about 6.7. This prevents overgrowth of micro-organisms which require high/low pH for maximal growth.
4) Salivary immunoglobulins, especially secretory IgA, may obstruct microbial adherence.

Bacterial factors
1) Adhesins are proteins associated with surface firls & fimbrae.
2) Lipoteichoic acid (in bacterial cell walls) may help attachment to glycoproteins in tooth pellicle.
3) Extracellular polymers, example: Glucosyl transferase enzymes produce glucans which play a primary role in adhesion & support adherence of other microbial species.
4) Some bacteria possess IgA proteases – allowing them to overcome the blocking action of secretory IgA at mucosal surfaces.
5) Bacterial enzymes may expose binding site. example: Neuraminidase

CONCLUSION
Clinicians need to be aware of the beneficial properties of the resident microflora, and their treatment strategies should be focussed on the control rather than the elimination of these organisms, especially in dental plaque. In the future, it may be feasible to target treatment more specifically at particular ‘pathogens’ (e.g. immunotherapy (53)), or more imaginative approaches could be used to prevent disease.

REFERENCES