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REVIEW ARTICLE

Microbial Flora in Oral Diseases

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ABSTRACT

The oral cavity being the hub of gamut of microbes, promotes the establishment of distinct microbial communities, such as on the mucosa and teeth. Metabolism of these organisms facilitates the attachment and growth of the subsequent colonisers. A delicate balance is maintained in the microbial ecosystem, with these organisms contributing to normal development and defences. However, any change or disruption in the microbial profile due to either intrinsic or extrinsic factors can result in an unfavorable shift toward pathogenic organisms triggering various diseases like dental caries or periodontitis. Furthermore, recent findings also state that these microorganisms may lead to systemic diseases like diabetes or atherosclerosis. This article is an attempt to give an overview of the altered flora in diseased states.

Keywords: Oral disease, Ecosystem, Microbial flora.

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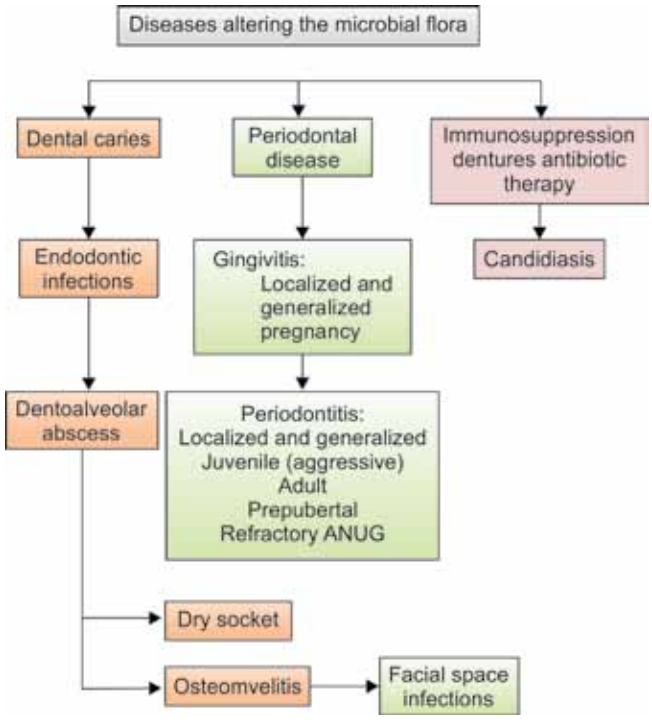
MICROBIAL ECOSYSTEM

The US National Institutes of Health, in 2007, launched a project termed 'Human Microbiome Project' to characterise the extent and diversity of microorganisms in the human body.[1] The oral cavity forms an indispensable part of this microbiome, as, of all the sites in the body, this is one of the most densely populated areas with microorganisms. With recently developed molecular methods 700 more species have been isolated.[2] The oral cavity is inhabited by a diverse microflora that may include bacteria, fungi, mycoplasma, protozoa and possibly viral flora of which bacteria are the predominant group.[3] Distinct microbial communities on mucosal surfaces of the tongue, buccal mucosa, tooth surfaces, gingival crevices and any artificial surfaces like prostheses and appliances, are established as a result of the environmental diversity in the oral cavity. The oral flora plays a very important role in the normal development of the host, rather than just having a passive relation. For example, it helps in contributing to host defences and prevents colonization by exogenous organisms.[4] The problem arises when there is a disruption of this resident flora leading to various diseases like dental caries or periodontitis. It is also possible that, members of the normal or the healthy flora, via many physiological processes, have the ability to change the environment to make it more conducive for the growth and proliferation of pathogenic organisms like *Streptococcus mutans* or *Porphyromonas gingivalis*. Hence, a change in this ecosystem, i.e. the microorganisms-environment interactions, increases the chances for pathogenicity leading to various oral diseases (Flow Chart 1). The understanding of this, is critical and of utmost importance in developing new preventive strategies.[5]

Flow Chart 1: Diseases altering the microbial flora



ROLE OF DENTAL PLAQUE

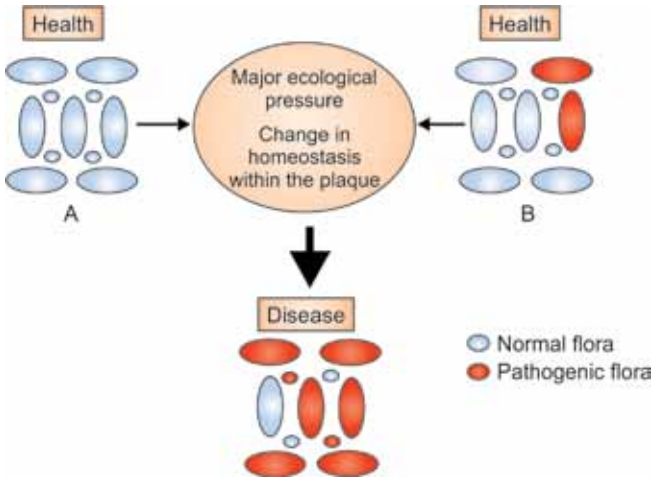
The oral cavity houses various habitats for microorganisms like, the mucosal surfaces (such as the lips, cheek, palate and tongue) and teeth which support the growth of microbial communities.[6] Dental plaque is the community of microorganisms found on a tooth surface as a biofilm. Plaque formation is a normal phenomenon which contributes to the host's normal development and defences. Problems arise as a result of disruption in the homeostasis existing between microbial communities in the plaque. Two common examples explaining this imbalance are dental caries and periodontal disease. In the former a change in the nutrient status like excess carbohydrates (increased supragingival plaque) results in the balance tilting toward more aciduric and acidogenic bacteria (e.g. *Streptococcus mutans*) to thrive, resulting in the disease. In the latter, accumulation of plaque, due to poor oral hygiene can result in deepening of pockets, creating anaerobic environments, thus favoring the proliferation of pathogenic bacteria (anaerobic species like *Fusobacterium, Prevotella*) leading to periodontal disease. Thus any change in the dynamics within this ecosystem can alter the flora, increase its potential pathogenicity and subsequently initiate and promote oral diseases (Fig. 1A and B).[2,7]

DENTAL CARIES

Dental caries is a chronic disease that progresses slowly in most individuals and is characterized by localized destruction of the tooth following long contact/interaction with acidic products that result from the bacterial fermentation of dietary carbohydrates.[8]

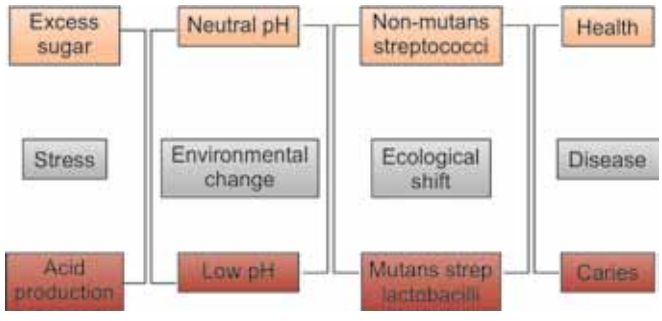
Dental caries is a very good example of how an alteration in the microflora can cause a disease. The normal microflora usually consists of nonmutans streptococci like the salivarius group (e.g. *Streptococcus salivarius*) on the root surface, mitis group (e.g. *S.sanguis*) in the pit and fissures and also a small number of microbes of the mutans group (not enough to induce caries). A situation may arise wherein the microbial ecosystem is disturbed and may result in increase in the number of caries inducing organisms like *Streptococcus mutans* and *Lactobacillus* species. It is possible that members of the healthy microbial flora have the potential to change the environment through physiological processes such as metabolic activities, subsequently facilitating the introduction of more pathogenic microorganisms like mutans streptococci. Nonmutans *Streptococcus* and *Actinomyces* are present in the supragingival area where there is a continuous supply of nutrients by the saliva and also by carbohydrates derived from food.

As the supragingival plaque accumulates or if there is an increased supply of carbohydrates, an acidic and anaerobic environment is created. The normal flora adapts to this change in the environment and can become aciduric (Flow Chart 2 and 3). These conditions now become more favorable

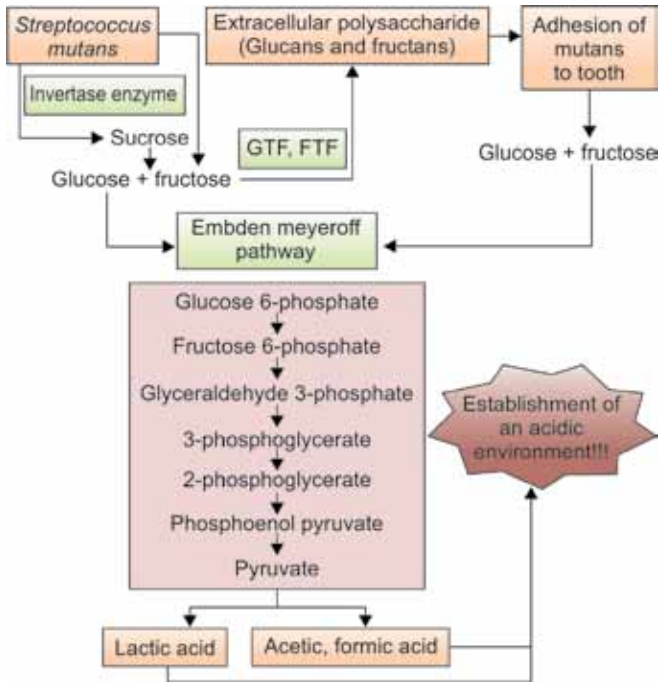


Figs 1A and B: Schematic representation: The inhabitants of the oral cavity maybe either normal (A) or minimally altered flora having micro-population of pathogens (B) The microbial ecosystem may tilt toward pathogenic organisms owing to homeostatic imbalance within the plaque

Flow Chart 2: Ecological plaque hypothesis



Flow Chart 3: Metabolism of *Streptococcus mutans*: (GTF: Gluocosyl transferase; FTF: Fructosyl transferase)



for aciduric organisms like mutans streptococci thus demineralizing the tooth surface. Mutans streptococci that colonise acidic sites, considered to be successors to the normal flora which have established an acidic environment, enhance the cariogenicity through a microbial shift (Table 1).[2,9,10]

PERIODONTAL DISEASE

Establishment of Flora in Periodontal Lesions

The subgingival crevice is flooded with gingival crevicular fluid (GCF) which creates a neutral/alkaline environment due to the presence of nitrogenous compounds, such as amino acids, peptides and proteins. As the gingival sulcus deepens, this environment is established and under these conditions, asaccharolytic and anaerobic and/or proteolytic bacteria, such as *Fusobacterium, Eubacterium, Campylobacter, Prevotella* and *Porphyromonas* are found. Proteolytic bacteria can degrade nitrogenous compounds into small peptides and amino acids by cell membrane-bound and/or extracellularly secreted proteases, for subsequent use as metabolic substrates. However, these enzymes secreted by the micro-organisms for degrading the nitrogenous compounds, induce inflammation and immunoreactions.

Flow Chart 4: Schematic representation of periodontal disease initiation (GCF: Gingival crevicular fluid)

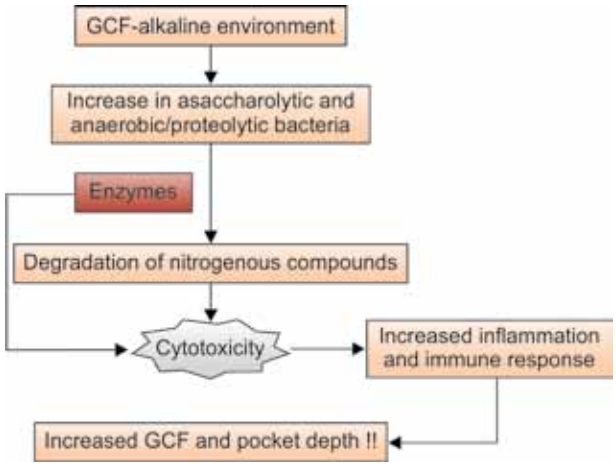


Table 1: Microorganisms implicated in dental caries[10]

Type of caries	*Microorganism*
Pit and fissure	*Streptococcus mutans*
	Lactobacillus sp.
Smooth surface (enamel)	*Streptococcus mutans*
Deep dentinal caries	*Lactobacillus sp.*
	Actinomyces naeslundii
	Actinomyces viscosus
	Other Filamentous rods
Root surface	*Actinomyces viscosus*
	Actinomyces naeslundii
	Other Filamentous rods
	Streptococcus mutans

P. intermedia and *F. nucleatum* are capable of growth at acidic and neutral pH, and are frequently found in supragingival plaque. In addition, *P. intermedia* and *F. nucleatum* are capable of neutralizing the acidic environmental pH by changing the acid-base balance through amino acid metabolism. *F. nucleatum* and *P. Intermedia* colonize a shallow gingival pocket (where the pH is variable and sometimes becomes acidic) and then promote the establishment of a neutral pH environment. This induces inflammation and an increase in GCF, inducing and promoting the growth of more proteolytic bacteria like *P. gingivalis*, and enhance the pathogenicity of *P. intermedia* through the increase in proteolytic activity and cytotoxic end products (Flow Chart 4 and Table 2).[2,11,12]

Table 2: Organisms associated with various periodontal diseases[3,5,16]

Periodontal disease	*Microorganisms*
Gingivitis	*Streptococcus sanguis*
	Streptococcus milleri
	Actinomyces israelii
	Actinomyces naeslundii
	Prevotella intermedia
	Capnocytophaga spp.
	Fusobacterium nucleatum
	Veillonella spp.
Pregnancy gingivitis	*Prevotella intermedia*
Adult periodontitis	*Porphyromonas gingivalis*
	Prevotella intermedia
	Fusobacterium nucleatum
	Tannerella forsythia
	Treponema denticola
	Aggregatibacter actinomycetemcomitans
Aggressive periodontitis — Localized chronic	*Aggregatibacter actinomycetemcomitans*
	Porphyromonas gingivalis
	Prevotella intermedia
	Capnocytophaga spp.
	Eikenella corrodens
	Neisseria spp.
	Aggregatibacter actinomycetemcomitans
Prepubertal periodontitis	*Fusobacterium spp.*
	Selenomonas spp.
	Campylobacter spp.
	Prevotella spp.
	Capnocytophaga spp.
Refractory periodontitis	*Tannerella forsythus*
	Porphyromonas gingivalis
	Campylobacter rectus
	Prevotella intermedia
Acute necrotizing ulcerative periodontitis (ANUG)	*Prevotella intermedia*
	Treponema spp.

IMPORTANT PERIODONTAL PATHOGENS

Porphyromonas Gingivalis

P. gingivalis, one of the major periodontal pathogens, is strictly anaerobic and a Gram-negative rod. It possesses several putative virulence factors (including proteases which degrade immunoglobulin, complement, collagen fibers, hyaluronic acid; adhesins, endotoxins and cytotoxins) that can cause periodontal disease directly or elicit host response resulting in gingival tissue and bone damage.[13]

Prevotella Intermedia

It is a black pigmented Gram-negative microorganism, resists phagocytosis by virtue of its capsule. It is also an important organism implicated in periodontal disease in association with *P. gingivalis* and *Aggregatibacter actinomycetemcomitans*.[13]

Aggregatibacter Actinomycetemcomitans

Implicated in aggressive periodontitis, it is a Gram-negative facultative nonmotile bacillus. *A. actinomycetemcomitans* secretes a protein toxin, leukotoxin (LtxA), which helps the bacterium evade the host immune response during infection.[14]

Fusobacterium Nucleatum

This organism plays a crucial role in the beginning of periodontal disease by producing proinflammatory cytokines and up-regulating the inflammatory response. They also induce the epithelial cells to secrete various proteolytic enzymes like matrix metalloproteases MMPs.[15]

Capnocytophaga Species

These are microaerophilic Gram-negative rods, implicated in the beginning of a juvenile periodontal disease and adult periodontal disease. This bacterium produces lipopolysaccharide with activity on alveolar bone, extracellular proteases which could damage immunoglobulins.[13]

ENDODONTIC INFECTIONS

The microbial flora of root canals have been studied extensively over the years, by using different sampling techniques and identification methods.[5] Current concepts suggest that the number of bacterial species in an infected root canal may vary from one to more than 12 (around 7-20) and the number of bacterial cells from $<10^2$ to $>10^8$ per sample.[17]

Microflora in Primary Endodontic Infections

The microbiota of the root canal is very similar to the microbiota of the periodontal pocket. Microbes usually present are *B. forsythus, F. nucleatum, P. gingivalis, P. intermedia, C. rectus, T. socranskii and T. denticola*.[18] Generally, *Prevotella* and *Porphyromonas* species are discussed when talking about participation of black pigmented bacteria in pathogenesis in primary endodontic pathology.[17] Gram- positive cocci, anaerobic rods, enteric bacteria and *P. aeruginosa* are also found.

Prevotella species, such as *P. intermedia* and *P. nigrescens* were more often found in infected root canals. These two species have been cultured from 26 to 40% of root canals of teeth with apical periodontitis, although during one study they were detected in only 13% of infected root canals. It was shown that *P. nigrescens* is more common in endodontic infections than *P. intermedia*.[17]

MICROFLORA IN ROOT CANAL FILLED TEETH

The persistence of microorganisms in the apical part of the root canal of filled teeth is believed to be the major cause of post-treatment disease after root canal treatment. Results of studies in which the microflora of teeth, with persistent disease, has showed a high prevalence of enterococci and streptococci followed by *Lactobacilli, Actinomyces* species, *Peptostreptococci, Candida, Eubacterium al actolyticus, Propionibacterium propionicum, Dialister pneumosintes* and Filifactor alocis.[17] *E. faecalis* came under focus as it was thought to be associated with endodontic failures, but it has now been proved otherwise and is known to be present in necrotic pulps prior to treatment as well as after any failed treatment.[5] Number of studies showed another extremely important characteristic of this microorganism, capacity to withstand a wide pH range up to around 11.5 of intracanal medicaments such as calcium hydroxide which is generally a highly potent antimicrobial dressing. These studies reveal

Table 3: Pathogens involved in endodontic infection

Species	*Prevalence (%)*
Peptostreptococcus	16
Streptococcus	14.2
Porphyromonas	12.2
Enterococcus faecalis	9.6
Staphylococcus salivarius	8.6
Prevotella spp.	8.1
Lactobacillus spp.	7.1
Actinomyces spp.	7.1
Candida albicans	3.6
Veillonella spp.	2.5
Eubacterium spp.	2.5
Bacillus spp.	2
Escherichia coli	1.6

a polymicrobial community within endodontic infections. Table 3 Lists out the prevalence of endodontic pathogens according to a study by Balaei Gajan et al.[19]

Dentoalveolar Abscess

Acute dentoalveolar abscess, a sequel of dental caries, trauma or failed root treatment, is the most commonly occurring orofacial bacterial infection. It develops by the extension of the initial carious lesion and spread of bacteria to the pulp. The bacteria and their toxic products cause pus formation by inducing acute inflammation via the apical foramen.

Culture and molecular methods have confirmed its polymicrobial nature, which is a mixture of obligate and facultative anaerobes (Table 4). In mixed infections, strict anaerobes outnumber facultatives by a ratio which varies between 1.5 and 3.1.

Facultative Anaerobes

The viridans group streptococci comprises of mitis group, oralisgroup, salivarius group, sanguinis group and the mutans group (Facklam, 2002). The anginosus group (formerly referred to as '*Streptococcus milleri*' or *Streptococcus anginosus*) is also identified and reported with varying degrees of accuracy. *Staphylococcus aureus* has been reported to occur more frequently in severe dental abscesses in children.

Obligate Anaerobes

Common species isolated are *Prevotella*, *Porphyromonas* and *Fusobacterium* spp. Among the bacteria belonging to genus *Prevotella* most frequently reported are *P. intermedia, P. nigrescens* and *P. pallens*. Among the *Porphyromonas* genus, *P. endodontalis*, *P. gingivalis* and *F. nucleatum* in the *Fusobacterium* genus have been isolated.

Improvements in sampling, culture and identification have resulted in the discovery of microorganisms which are 'unfamiliar'. These include members of the genus *Atopobium* (Gram-positive strictly anaerobic coccobacilli), e.g. *Atopobium parvulum* and *Atopobium rimae*. Anaerobic Gram-positive rods include *Bulleidia extructa, Cryptobacterium curtum, Eubacterium sulci, Mogibacterium timidum and Mogibacterium vescum* (Sakamoto et al, 2006), *Pseudoramibactera lactolyticus* and *Slakia exigua* (Siqueira and Rocas, 2003c).[3,5,20]

Table 4: Most common bacteria isolated in dentoalveolar abscess[3,5,20]

Facultative anaerobes	*Obligate anaerobes*
Streptococcus milleri	*Peptostreptococcus* species
Streptococcus sanguis	*Porphyromonas gingivalis*
Actinomyces spp.	*Prevotella intermedia*
	Fusobacterium nucleatum

Table 5: Principle of candida species[30]

Candida albicans
Candida dubliniensis
Candida famata
Candida glabrata
Candida guilliermondii
Candida inconspicua
Candida kefyr
Candida krusei
Candida lusitaniae
Candida norvegensis
Candida parapsilosis
Candida rugosa
Candida tropicalis

OSTEOMYELITIS

Osteomyelitis is an inflammation of the medullary cavity within the maxilla or mandible with possible extension of infection into the cortical bone and the overlying periosteum.[3,5] Chronic osteomyelitis of the jaws are common in the developing countries, where these diseases are associated with trauma, surgical procedures and previous infections, such as endodontic and periapical infections.[21]

As majority of osteomyelitis cases begin as dentoalveolar abscess, they share the same microbiota. However it is possible that pathogens different from those of the oral microbiota may reach the bone tissues through transient bacteremia, which are common after surgical procedures or traumas.[22]

Staphylococcus aureusis implicated in 90% of osteomyelitis cases. *S. aureus, S. epidermidis, Pseudomonas aeruginosa, Serratia marcescens* and *Escherichia* coli are also isolated in cases of chronic osteomyelitis.[23]

In the chronic osteomyelitis associated with previous odontogenic infections, the etiology of the osteomyelitis will depend on the microbiota of the previous infectious process and usually comprises of a mixed microbial population like *Tannerella, Prevotella, Porphyromonas* and *Fusobacterium, Parvimonas and Eikenella* mostly *Actinomycetes and Staphylococci*.[3,24]

Osteomyelitis
Staphylococcus aureus
Staphylococcus epidermidis
Pseudomonas aeruginosa
Serratia marcescens
Escherichia coli
Tannerella species
Prevotella species
Porphyromonas species
Fusobacterium parvimonas
Eikenella species
Actinomycetes species

FACIAL SPACE INFECTIONS

A dentoalveolar infection, if not properly treated, can rapidly spread bilaterally to the tissue spaces (submandibular, sublingual and submental) in the head and neck. It is a serious, life-threatening infection that needs immediate intervention.

It usually arises as a result of dental or postextraction infection, mandibular fractures, sialadenitis, peritonsillar abscess, oral soft tissue lacerations and puncture wounds of the floor of the mouth.

Common Microorganisms

The organisms most often isolated in patients with this infection are *Streptococcus viridians* and *Staphylococcus aureus*. Anaerobes also are frequently involved, including bacteroides, peptostreptococci and peptococci. Other Gram-positive bacteria that have been isolated, including *Fusobacterium nucleatum, Aerobacter aeruginosa, Spirochetes* and *Veillonella, Candida, Eubacteria* and *Clostridium* species. Gram-negative organisms that have been isolated include *Neisseria* species, *Escherichia coli, Pseudomonas species, Haemophilus influenza* and *Klebsiella* species, *Prevotella spp*. and *Porphyromonas spp*. have also been isolated.[3,5,25,26]

Facial space infections
Streptococcus viridans
Staphylococcus aureus
Fusobacterium nucleatum
Aerobacter aeruginosa
Spirochetes
Veillonella species
Candida species
Eubacteria species
Clostridium species
Neisseria species
Escherichia coli
Pseudomonas species
Haemophilus influenzae
Klebsiella species
Prevotella species
Porphyromonas species

DRY SOCKET

Alveolar osteitis or dry socket is one of the most common postextraction complication, 2 to 4 days postsurgery.[27] A localized fibrinolysis (resulting from conversion of plasminogen to plasmin, which dissolves fibrin crosslinks) occurring within the socket and subsequently leading to loss of the blood clot is believed to underlie the pathogenesis of alveolar osteitis. There is an increased incidence of dry socket being reported in patients with poor oral hygiene, higher pre and postoperative microbial counts, pericoronitis and periapical infection. Hence, bacteria are suspected of causing dry socket.[28]

Nitzan et al (1983) proposed, in particular, the role of anaerobic bacteria, especially *Treponemma denticola*, which showed plasminlike fibrinolytic activity *in vitro*.

A series of bacteria, which included *Enterococcus, Streptococcus viridians, Bacillus coryneform, Proteus vulgaris*, *Pseudomonas aeruginosa*, *Citro bacterfreundii,* and *Escherichia coli* were identified in the biologic material within the alveolus in experimental dry socket models. Also, in accordance with the potential role of bacteria in dry socket development, the inoculation of *Actinomycesviscosus* and *Streptococcus mutans* in animal sockets was reported to delay the sequence of alveolar repair.[27-29]

Dry socket
Treponemma denticola

CANDIDIASIS

Candidiasis is caused by a yeast like fungus, *Candida albicans*. Other species like *C. tropicalis, C. parapsilosis, C. stellatoidea* and *C. krusei* may also be involved (Table 5). *Candida* is a component of the normal oral microflora, with 30 to 50% of people carrying it in their oral cavity and is kept under control by means of specific and nonspecific defencse mechanisms and also by the competition of the microbes in the normal flora. Colonization in the newborn occurs from the mother's vaginal flora or other exogenous sources. Most people usually carry a distinct strain of *Candida* and the colonizer is usually the culprit (infecting strain) if at all infection occurs.[30] *C. albicans* exists in two forms- a trait known as dimorphism (yeast and hyphal form). Candidiasis is the most common fungal infection in humans. However, the mere presence of the microorganism is not enough to cause the disease.

Candidiasis is an opportunistic infection which flares up during immune suppression or a drastic change in the oral flora. The ability of Candida to adhere to the mucosa and dentures plays an important role in the pathogenesis of oral yeast infections. Adherence is achieved by specific and nonspecific mechanisms. However, the mechanisms are still not fully understood.

Predisposing factors include the following:

1. Acute and chronic diseases like tuberculosis, diabetes mellitus and anemia.
2. AIDS
3. Nutritional deficiencies
4. Prolonged hospitalization for debilitating diseases
5. Prolonged use of antibiotics and corticosteroids
6. Radiation therapy
7. Old age

Candidiasis incidence has resurfaced over the recent years with the advent of HIV. It is reported that more than 90% of HIV infected patients develop candidiasis.[29-31]

CONCLUSION

Oral microbial flora, beyond doubt, have a very important role to play in maintenance of homeostasis of the ecosystem in the oral cavity. It is crucial for clinicians to be aware of this fact and they should focus their treatment toward control of this flora rather than eliminating it. A thorough knowledge of the normal and altered flora and the mechanics behind how the change can happen and what it might lead to would give us a fair idea of how various oral diseases could be controlled and preventive strategies be developed.

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