



It's all About the Bugs: Significance of Pre- And Probiotics In Periodontics.

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ABSTRACT: The mouth consists of more than 700 bacterial species. These are commensals, that is, they are associated with health benefits. Remaining are pathogenic that cause various oral health problems including periodontal diseases. These bacteria contest among themselves to settle in the oral cavity and this is the basis for 'probiotic' therapy. Probiotic therapy aims at modulating the composition of microflora by administering living microorganisms that confer a health benefit to the host when administered in a sufficient amount. To prove probiotics beneficial more studies are required. Periodontal disease has proven very difficult to treat. The outcome of the animal and clinical periodontitis studies supported the use of probiotics in the treatment of periodontitis. Probiotics may offer a low-risk, inexpensive, easy to use prevention or treatment option for the management of periodontal disease. In the future, more independent studies are needed to look into specific probiotic strains, doses, delivery methods, treatment schedule, mechanisms of action, safety and how to maintain the results of the probiotic interventions.

KEYWORDS: Probiotics, periodontitis, microflora.

I. INTRODUCTION

The mouth consists of more than 700 bacterial species. These are commensals, that is, they are associated with health benefits. Remaining are pathogenic that cause various oral health problems including periodontal diseases. There are particular places in the oral cavity where these pathogenic bacterial species can be located. "Dental plaque", consists of plaque biofilm along with bacteria, that have a well-defined place and role. These bacteria contest among themselves to settle in the oral cavity and this is the basis for 'probiotic' therapy. Probiotic therapy aims at modulating the

composition of microflora by administering living microorganisms that confer a health benefit to the host when administered in a sufficient amount.¹ Prebiotics are compounds in food that bring about growth or activity of advantageous microorganisms like bacteria and fungi.²

There is a long-term history, that in certain areas of Europe and Asia, microbes or food products influences the intestinal microbiota, i.e., ingestion of probiotics and prebiotics has positive outcome. Probiotics are viable microorganisms that confer health benefit when applied in adequate doses. There are certain species of lactobacilli, bifidobacteria, and *Saccharomyces* spp., along with streptococci, enterococci and commensal *Escherichia coli* that have a beneficial effect in certain situations. So, these organisms are been used as probiotics.

Prebiotics (e.g., inulin-type fructans, maltodextrin, fructo-oligosaccharides and galacto-oligosaccharides) have been defined as non-digestible oligosaccharides that affect the proliferation of resident commensal bacteria that may then exert probiotic effects. In modern era, the definition has been purified to incorporate selectively fermented elements that permit particular changes in the configuration and/or activity of the inhabitant microflora that confer benefits upon host health and well-being. Prebiotics study of the oral cavity is still not clear. These studies mainly focus on gastrointestinal microbiota that have health benefits.

II. CRITERIA FOR PROBIOTICS

The criteria should be taken into account are as follows.

- It should be of human origin that are capable of influencing a useful effect on the host



organism, for example, increased growth or resistance to disease.

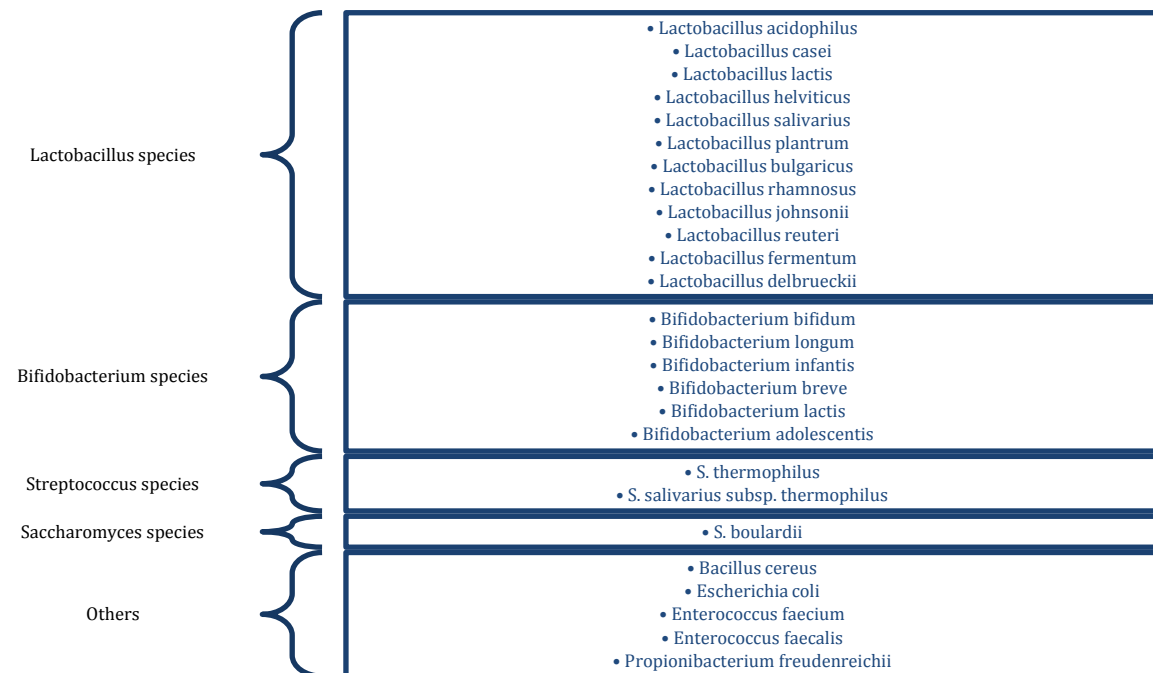
- It should be non-pathogenic and nontoxic having high cell viability.
- It should be able to interact or to send signals to immune cells. Thus, the capacity to influence the local metabolic activity is enhanced.
- It should be stable i.e., capable of remaining viable for periods under storage and does not metabolize in the gut, for example, resistance to low pH and organic acids.

III. RATIONALE FOR THE USE OF PROBIOTICS

- Conventional antibiotic therapy provides resistance to bacteria and an alternative is required to treat the acute and chronic form of disease.
- To raise the profile of disease prevention rather than disease management.
- Changing paradigms in understanding of the pathogenesis of disease.
- Probiotics prevent colonization, overgrowth, and translocation of potential pathogens.

IV. COMPOSITION OF PROBIOTICS

Probiotics can be varied. They can be yeast, bacteria or moulds. But most frequently, bacterial species are predominant.



The lactobacillus species help in manufacturing of enzymes to digest & metabolize proteins and carbohydrates. Important probiotics microbial species useful in oral cavity are L. acidophilus, L. casei, L. rhamnosus GC, L. sporogens, L. bulgaricus, L. johnsonii, L. thermophilus, L. bifidum, L. reuteri, L. salivarius,

V. REPLACEMENT THERAPY AND PROBIOTIC THERAPY

The term replacement therapy (also called bacteriotherapy or bacterial interference) is sometimes used correspondently with probiotics. For

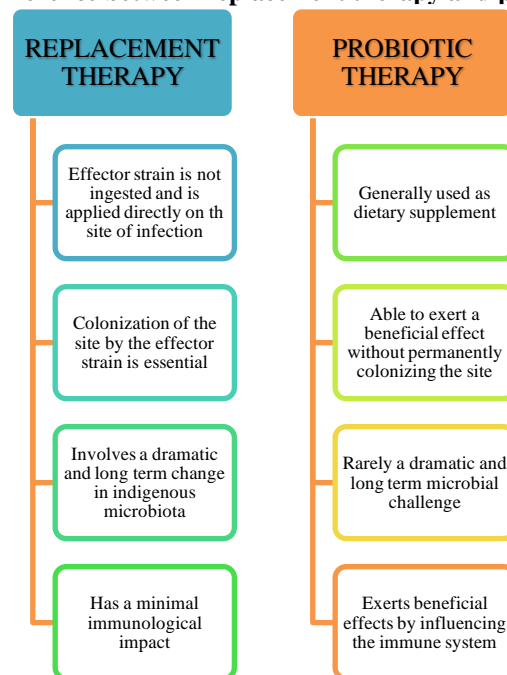
L. paracasei, S. thermophilus, S. salivarius, W. cibaria and bifidobacterium. They assist in the production of vitamin B, and vitamin K and promote breakdown of bile salts. Probiotics may be in gel, paste, powder or liquid forms. Most commonly probiotics are ingested in lozenges, tablets, yoghurt, cheese, milk or mouth rinse. some decades now, bacteria known as probiotics have been added to various foods because of their advantageous effects for human health. Probiotics are designed to help the body's naturally existing gut microbiota. The substances which abandons the viability of microorganisms or excludes the



survival of the microbes and their effects on the indigenous microbiota is called probiotic. The term 'replacement therapy' is sometimes used interchangeably with probiotics. These two approaches use live bacteria for prevention or treatment of infectious diseases. But both are not same. There are some dissimilarities which are listed below (Table 1).

The possibility of usage of antagonistic organisms to control pathogens and prevent disease has been termed as "Replacement Therapy". This approach has the possible advantage that it furnishes lifelong shield with minimal cost and compliance on behalf of recipients, once colonization by effectors strain has been achieved.³

Table 1: Difference between replacement therapy and probiotic therapy



There are two main perspectives by which replacement therapy are being considered as a means of enhancing colonization resistance in plaque to prevent periodontal disease. These are pre-emptive colonization and competitive displacement (Figure 1).

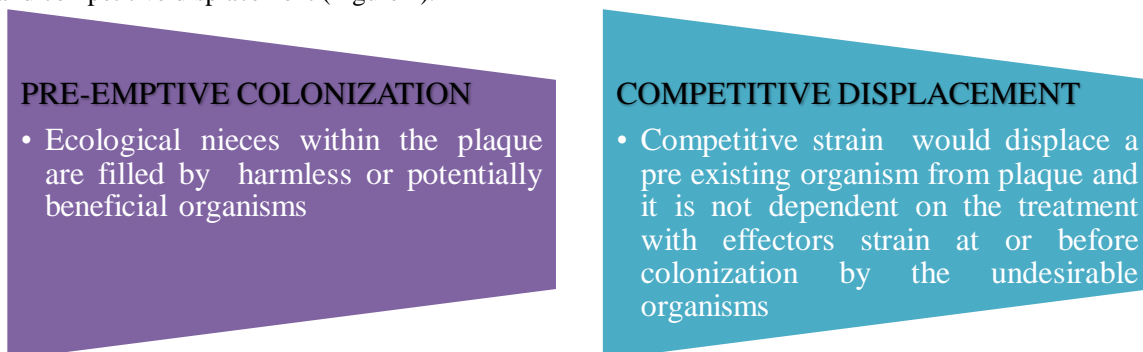


Figure 1: Pre-emptive colonization and Competitive displacement

VI. MECHANISM OF ACTION OF PROBIOTICS

Major probiotic mechanisms of action include enhancement of the epithelial barrier, increased adhesion to intestinal mucosa, and

concomitant inhibition of pathogen adhesion, competitive exclusion of pathogenic microorganisms, production of anti-microorganism substances and modulation of the immune system.⁴

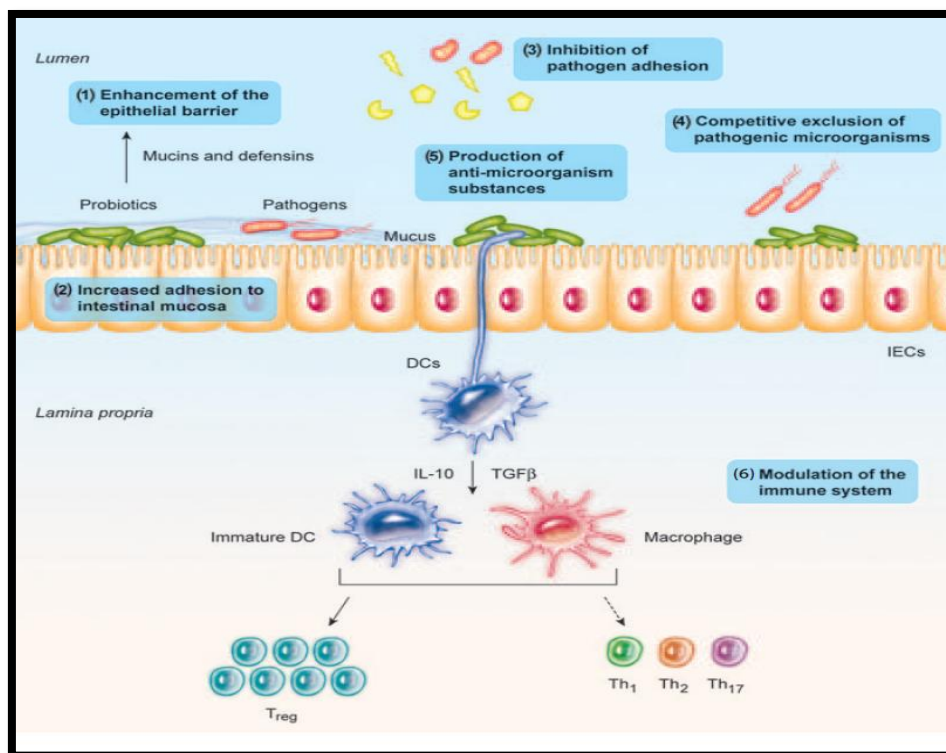


Figure 2: Mechanism of action of probiotics

VII. ROLE OF PROBIOTICS IN PERIODONTAL HEALTH AND DISEASE

PROBIOTICS AND PERIODONTITIS – Probiotics animal study done by Teughles et al., 2007, Nackaert et al., 2008, Messori et al., 2013, and Foureaux et al. 2014 with sample sizes 8, 8, 32 and 64 of beagle dogs, beagle dogs, wistar rats and

wistar rats, with pellets, pellets, water and water respectively. The conditions used were artificially created periodontitis. Different parameters which had been used in these studies, bleeding on probing, plaque and gingival indices, attachment loss and alveolar bone level, have shown positive results towards the concerned treatment modalities.

Table 2: Animal probiotics studies

Study	Type of participants, number	Condition	Probiotic strains, vehicle, time	Results
Teughles et al., 2007 (Teughels et al., 2007b)	Beagle dogs, 8	Artificially created periodontal pockets	Streptococcus salivarius, S. sanguinis, S. mitis, pellets, 12 weeks	Reduction in periodontal pathogens and BOP when probiotics were used in adjunction to mechanical debridement



Nackaerts et al., 2008	Beagle dogs, 8	Artificially created periodontal pockets	<i>S. salivarius</i> , <i>S. sanguinis</i> , <i>S. mitis</i> , pellets, 12 weeks	Significant increase in bone levels and bone density in probiotic group when compared with placebo
Messora et al., 2013	Wistar rats, 32	Ligature-induced periodontitis	Product based on <i>Bacillus subtilis</i> , in water, 44 days	Mean values of AL and ABL were significantly higher in the induced periodontitis group compared with the treatment group
Foureaux et al., 2014	Wistar rats, 64	Ligature-induced periodontitis associated with restraint stress	Product based on <i>B. subtilis</i> , in water, 44 days	Bone loss was prevented in the probiotic treated induced periodontitis unstressed group
BOP, bleeding on probing; AL, attachment loss; ABL, alveolar bone level				

Probiotics human study done by Vivekananda et al., 2010, Teughles et al., 2013, Vicario et al., 2013, Szkaradewicz et al., 2014 and Ince et al., 2015 with sample sizes 30, 30, 20, 38 and 30 of all adults having periodontitis with lozenges, lozenges, tablets, tablets, lozenges respectively. Different parameters which had been used in these studies, bleeding on probing, plaque and gingival indices, clinical attachment loss, have shown positive results towards the concerned treatment modalities.

Probiotics human study done by Riccia et al., 2007 and Shimauchi et al., 2008 with sample sizes 29 and 66 of all adults having periodontitis with lozenges and tablets respectively. Different parameters which had been used in these studies, bleeding on probing, plaque and gingival indices, clinical attachment loss, have shown neutral results towards the concerned treatment modalities. Although such studies are fewer in number in compare to studies which have been shown positive implications.

Table 3: Clinical probiotics-chronic periodontitis studies.

Study	Type of participants, number, age	Probiotic strains, vehicle, time	Result
Riccia et al., 2007	Adults, 29, 24-51	<i>Lactobacillus brevis</i> , lozenges, 4 days	Decreased clinical parameters in treated periodontitis patients when compared with controls (gingival inflammation, BOP, plaque, calculus, temperature sensitivity) Decreased levels of PGE ₂ , MMP and INF-γ in saliva samples of treated periodontitis patients
Shimauchi et al., 2008	Adults, 66, 32-61	<i>L. salivarius</i> , tablets, 8 weeks	Current smokers in the probiotic group showed a significantly greater improvement of plaque index and probing depth from baseline when compared with those in the placebo group
Vivekananda et	Adults, 30, 34-50	<i>L. reuteri</i> ,	PPD, CAL, GI, GBI and PPD



al., 2010		lozenges, 42 days	significantly reduced in the SRP plus probiotic group compared with SRP alone or placebo
Teughles et al., 2013	Adults, 30, older than 35	L. reuteri, lozenges, 12 weeks	Significantly more pocket depth reduction and attachment gain in the moderate and deep pockets and also reduction in P. gingivalis numbers in the test group when compared with controls
Vicario et al., 2013	Adults, 20, 44-65	L. reuteri, tablets, 30 days	Improved short-term clinical outcomes (PI, BOP, and PPD) in non-smoking patients with initial to moderate chronic periodontitis.
Szkaradiewicz et al., 2014	Adults, 38, 31-46	L. reuteri, tablets, 2 weeks	Significant improvement in SBI, periodontal probing depth and clinical attachment level and also decreased levels of pro-inflammatory cytokines TNF- α , IL-1 β , IL-17 in treated patients when compared with the control group
Ince et al., 2015	Adults, 30, 35-50	L. reuteri, lozenges, 3 weeks	Significant differences in PI, GI, BOP and PPD and significant mean values of attachment gain in favour of the test group compared with control tools. Significant decreased levels of MMP-8 and increased levels of TIMP-1 were found in GCF for the group up to day 180
Tekce et al., 2015	Adults, 30, 35-50	L. reuteri, lozenges, 3 weeks	1 year follow-up study from the previous Ince et al.; 2015. PI, GI and BOP significantly lower in the test group compared with controls; difference in the total viable count and the proportion of obligate anaerobes were decreased in the test group up to day 180
<p>PGE₂, prostaglandin E₂; MMP, metalloproteinase; TIMP-1, tissue inhibitor of metalloproteinase; INF-γ, interferon γ; PI, plaque index; BOP, bleeding of probing; PPD, pocket probing depth; CAL, clinical attachment loss; GI, gingival index; GBI, gingival bleeding index; SRP, scaling and root planning; SBI, sulcus bleeding index</p>			

PROBIOTICS AND HALITOSIS- Halitosis is a very familial oral health issue among general population. It is fundamentally produced by various anaerobic bacteria found in various niches in the oral cavity. Various periodontopathogens have been located to be correlated with the manufacture of several compounds that are correlated with the formation of oral malodor. Cultivable oral cavity bacteria associated with halitosis consists mainly of Porphyromonas gingivalis, Treponema denticola and Tenerella forsythia. Halitosis is caused by the generation of volatile sulfur compounds due to degradation of S-containing amino acids by bacteria in the oral cavity. One effective way of governing halitosis is decreasing the number of

anaerobic bacterial species in the oral cavity by a deduction in their total counts by settling their niches with probiotic bacterial strains.⁵

Only a few studies have evaluated the effect of using probiotics on halitosis. In a study, Kang et al. (2006) have reported that various strains of W. cibaria could restrict the generation of various volatile sulfur compounds (VSCs) produced by F. nucleatum. The reason proposed for this inhibition was the generation of hydrogen peroxide by W. cibaria, which restricted the amplification of F. nucleatum. In another study, Kazor et al. (2003) compared the microbial species isolated from the tongue of halitosis patients to that isolated from healthy patients. The authors reported



that most commonly linked bacterial species with halitosis patient included *Atopobium parvulum*, *Eubacterium sulci*, and *Fusobacterium periodonticum* whereas *Streptococcus salivarius* was most commonly isolated from healthy patients. The authors stated that *S. salivarius* being competent of producing bacteriocins contributes to the reduction of bacterial species producing VSCs that produces a foul smell. Thus, it can be used as an effective treatment strategy to control halitosis. These results are supported by another study done by Burton et al. (2006) who reported a reduction in halitosis after consumption of gum or lozenges containing *S. salivarius* K12 by patients diagnosed with halitosis. The inhibitory effect of bacteriocins produced by *S. salivarius* K12 on halitosis associated bacteria has also been confirmed another study done by Masdea et al. (2012).

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VIII. CONCLUSION

The concept of probiotics give rise to a new scope between diet and oral health. Probiotics are very useful in oral care applications. To prove probiotics beneficial more studies are required. Periodontal disease has proven very difficult to treat. The outcome of the animal and clinical periodontitis studies supported the use of probiotics in the treatment of periodontitis. Probiotics may offer a low-risk, inexpensive, easy to use prevention or treatment option for the management of periodontal disease. In the future, more independent studies are needed to look into specific probiotic strains, doses, delivery methods, treatment schedule, mechanisms of action, safety and how to maintain the results of the probiotic interventions.

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