TPM Vol. 32, No. S5, 2025
ISSN: 1972-6325
https://www.tpmap.org/

Open Access

ORAL MICROBIOME ROLE IN ORAL HEALTH–AN INSIGHT – A REVIEW

DR.J. BHUVANESWARRI, MDS
PROFESSOR, DEPARTMENT OF PERIODONTOLOGY, SREE BALAJI DENTAL COLLEGE AND HOSPITAL.

DR JULIUS AMALDAS PHD
PROFESSOR & HEAD OF THE DEPARTMENT, DEPARTMENT OF BIOCHEMISTRY, SREE BALAJI DENTAL COLLEGE, CHENNAI. TAMIL NADU, INDIA

DR. PREETHI P, MDS
READER, DEPARTMENT OF PERIODONTOLOGY, SREE BALAJI DENTAL COLLEGE AND HOSPITAL.

DR. RAMYA.V MDS
PROFESSOR, RESEARCH SCHOLAR, DEPARTMENT OF PERIODONTOLOGY, SREE BALAJI DENTAL COLLEGE, CHENNAI.CHENNAI. TAMIL NADU, INDIA

DHEEPAK B
SAVEETHA MEDICAL COLLEGE, SAVEETHA INSTITUTE OF MEDICAL AND TECHNICAL SCIENCES

Abstract:
The oral microbiome is the main factor of human microbiome. It has the second largest microbial community after the bowel. With the newer advancement in coming- generation sequencing and bioinformatics, numerous studies have evolved describing the complexity of oral microbiome profile. By assessing the oral microbiome profile, we can identify the functional and chemical changes that can be related with the disease. It is also helpful for medicine development using molecular autographs and targeted remedy can be achieved rendering the substantiated and precised drug to the case. Hence, this review article gives a sapience about the oral microbiome profile in health and diseased state.

Keywords: Oral microbiome, socioeconomic status, stress, periodontal disease

INTRODUCTION

The entrance of the body to the external terrain is the mouth representing the most biologically complicated and important point. The first stage of the digestive process take place in the mouth and is plushly endowed with sensitive functions. Our human body composes of 10 ^{14} cells. Of these 10% represents mammalian. Other microorganisms comprise the resident microflora of the host. The community of oral microorganisms represents the association with the human host. There are numerous attestations proving the interrelation between the composition of the oral microbiome in healthy and diseased condition.

HISTORY OF ORAL MICROBIOLOGY :

The term " microorganism " simply refers to veritably small living things like archaea, bacteria, protozoa, fungi, and contagions, while " microbiome " is a collaborative and comprehensive term for microorganisms. Microbiome is the community of microbial residers, and recent exploration on microbiomes has demonstrated the significance of it in human homeostasis.[1,2] The discovery of the oral microbiome was first determined by the Dutchman Antony van Leeuwenhoek, who used microscope of his tone establishment. The American dentist W.D. Miller recognised the relation between oral microbial community and oral diseased condition in 19th century in Berlin. The term " microbiome " was established by Joshua Lederberg, [3] who said "to signify the ecological community of commensal, symbiotic, and pathogenic microorganisms that literally share our body space and have been all but ignored as determinants of health and disease"

In an healthy grown-up, one milliliter of saliva contains 100 million bacterial microorganisms. Saliva of 750 ml each day, has 8 × 10^{10} bacteria which are removed from the oral cavity every 24 hours. The human oral microbiome

community are drawn from the surfaces of mucosa of the palate, tonsils, cheeks and from the hard, non shedding areas of the teeth.[4] The saliva has a pH of 6.5 –7.0 and temperature of an normal of 37^{0}C, creating a good environment for the growth of microorganisms.[5] In 1676, Antonie van Leeuwenhoek identified bacterial cells with his microscope to estimate the " animacules " in plaque from the tooth surfaces.

PERIODONTITIS/ BACTERIAL RELATIONS :

Periodontitis has numerous etiologies. Periodontitis can occur has a result of interaction of microorganisms from the plaque present subgingivally on the tooth and a dysregulated response by the host in the periodontium.[6] This interaction can be obtruded by debridement and giving antibiotic remedy. It is the gold standard treatment. The sub-gingival microbial community has variable complex and makes it delicate for identification of the periodontal microorganisms even though there's a association between the quantity of the dental biofilm.[7] Numerous analysis on culture ways and DNA DNA hybridization ways, has honored periodontal pathogens as red complex bacteria. The virulence factors of the red complex bacteria, Porphyromonas gingivalis, Tannerella forsythia and Treponema denticola, doesn't give a exact association with the disease. They're able of inhibiting and escaping ingrain responses of the host. This shows that the relation of bacterial species to the diseased condition point is due to dismembering response of periodontal ingrain impunity that has negative effect on host relations with the dental biofilm.[8] The understanding of these relations may be assessed from environmental and endogenous factors that causes periodontitis. The factors are smoking, stress, oral hygiene, obesity and inheritable factors[9]. Normal host homeostasis can be disintegrated by numerous mechanisms, either altering defensive status of the host or the composition of microorganisms or both. In case of caries and periodontal disease, the composition of dental biofilm should be the distinct factor as these lesions are interrelated to dental plaque. The pathogens for caries and periodontal diseases are seen in areas of supragingival and subgingival plaque.[10] P. gingivalis is considered the keystone bacteria which gives the virulence of oral microbiome in periodontal disease rather acting directly on the periodontal tissue to cause destruction[11]. Removing the dental biofilm is the standard treatment for interfering the oral disease. The oral microbiome has the eventuality of regulating the virulence factors of bacteria in dental plaque. Still, further studies are demanded to describe the overall mechanisms of the microbial and the host connections.

SOCIOECONOMIC STATUS :

One of the major threat factor of periodontium are the lifestyle mainly smoking habit. Numerous studies assess that persons who smoke greater than 10 cigarettes each day has an increased threat of developing periodontitis than compared with the average risk of the population [12]. Several studies has shown that the difference in socioeconomic status has a huge impact on periodontal conditions [13] whereas other studies has not verified this risk[14]. Studies have shown that people with low socioeconomic status have a low health profile because of health deteriorating habits and behaviours and also negligible response on their oral health. They are further susceptible to consume unhealthy foods, tobacco and alcohol.

The distinct features of their behavior, their knowledge and health approach of these people is associated to their education.[15] People with lower educational position have an increased smoking habit than people with higher education level[16]. In a study conducted over a decade, the effectiveness of education on periodontal disease was set up to be significant in never smokers[17]

STRESS AND PERIODONTAL DISEASE:

Stress has a huge influence on the immune system and changes a person's behaviour , which can be used to assess progression and forms of periodontal disease. In 1969 Ringsdorf and Cheraskin[18] stated that stress can have a major impact on life- style and oral hygiene practices. It causes a negative effect on the oral hygiene habits and increase the frequence of consumption of alcohol and tobacco which can deteriorate general health. Suchday et al[19] conducted a study which verified the same. When the person has a poor oral hygiene, has a weaker immune response and increases the threat of periodontal disease.

GINGIVITIS :

Stress can alter the salivary inflow rate and increase the dental plaque formation.[20] The composition of the IgA secretion and salivary pH is altered[21].

NECROTIC PERIODONTITIS :

Psychological and social factors is a susceptible element for developing the necrotic periodontitis. In 1983, Cohen-Cole stated the effect of psychosocial factors[22]. The following are the threat factors of the disease: inadequate sleep, poor oral hygiene, stress, tobacco and alcohol consumption.

AGGRESSIVE PERIODONTITIS :

In 1983, Page et al [23] stated that the aggressive periodontitis showed the interaction between psycho-social factors and appetite loss. In 1996, Monteira da Silva [24] stated that aggressive periodontitis cases are known to have an increased depression and people who are isolated socially than people with chronic periodontitis. Kamma and Baehni[25] stated

that supportive periodontal care has a positive impact in some cases and in certain spots is still adding . The following factors were associated to the development of the disease. P. gingivalis, T. denticola, total bacterial count, number of pivotal occurrences, total teeth lost, smoking and stress.

CHRONIC PERIODONTITIS :

Linden et al[26] stated that the future aspect of loss of attachment can be assessed based on age, socio- economic status, unsatisfactory professional life and dependant character. In 1998, Axtelius[27] has stated that people with psychosocial strain have low response than people with lower psychosocial stress to periodontal treatment. Financial problems and distress are other factors which impact the development of periodontal disease [28].

CONCLUSION

Numerous attestations showed the positive impact among psychosocial status, stress, quality of life and periodontal status conducted in other countries. Variable studies were conducted in South Indian population. This study measures the significance of psychosocial factors in assessing the risk probability and oral microbiome profile in high risk periodontal disease patients of South Indian Population. People at low socioeconomic status, feeling depressed and socially isolated are at greater threat of periodontal conditions. Hence the dentists can provide periodontal treatment and also people can be appertained to a psychologist if demanded therefore enhancing the quality of life. Further longitudinal studies are demanded with a larger population.

REFERENCES

1.Segata N, Haake SK, Mannon P, Lemon KP, Waldron L, Gevers D et al. (2012) Composition of the adult digestive tract bacterial microbiome based on seven mouth surfaces, tonsils, throat and stool samples. Genome Biol 13, R42.
2. Zhou Y, Gao H, Mihindukulasuriya KA, La Rosa PS, Wylie KM, Vishnivetskaya T et al. (2013) Biogeography of the ecosystems of the healthy human body. Genome Biol 14
3. Kilian M, Chapple IL, Hannig M, Marsh PD, Meuric V, Pedersen AM, et al. The oral microbiome – An update for oral healthcare professionals. Br Dent J. 2016;221:657–66
4.Dewhirst FE, Chen T, Izard J, Paster BJ, Tanner AC, Yu WH, et al. The human oral microbiome. J Bacteriol. 2010;192:5002–17
5. Lim Y, Totsika M, Morrison M, Punyadeera C. Oral microbiome: A New biomarker reservoir for oral and oropharyngeal cancers. Theranostics. 2017;7:4313–21.
6. Page RC, Kornman KS. The pathogenesis of human periodontitis: an introduction. Periodontology 2000. 1997 Jun;14(1):9-11.
7. Socransky SS, Haffajee AD, Cugini MA, Smith CK, Kent Jr RL. Microbial complexes in subgingival plaque. Journal of clinical periodontology. 1998 Feb;25(2):134-44.
8. Darveau RP. Periodontitis: a polymicrobial disruption of host homeostasis. Nature Reviews Microbiology. 2010 Jul;8(7):481-90.
9. Stabholz A, Soskolne WA, Shapira L. Genetic and environmental risk factors for chronic periodontitis and aggressive periodontitis. Periodontology 2000. 2010 Jun;53(1):138-53.
10. Ximenez-Fyvie LA, Haffajee AD, Socransky SS (2000) Comparison of the microbiota of supra- and subgingival plaque in health and periodontitis. J Clin Periodontol 27, 648-657
11. Hajishengallis G, Liang S, Payne MA, Hashim A, Jotwani R, Eskan MA et al. (2011) Low-abundance biofilm species orchestrates inflammatory periodontal disease through the commensal microbiota and complement. Cell Host Microbe 10, 497-506
12.Silva AM, Newman HN, Oakley DA, O'Leary R. Psychosocial factors, dental plaque levels and smoking in periodontitis patients. Journal of clinical periodontology. 1998 Jun;25(6):517-23.
13. Halling & Bengtsson 1984, Brown et al. 1994, Elter et al. 1999), Klinge B, Norlund A. A socio-economic perspective on periodontal diseases: a systematic review. Journal of clinical periodontology. 2005 Oct;32:314-25.
14. Klinge B, Norlund A. A socio-economic perspective on periodontal diseases: a systematic review. Journal of clinical periodontology. 2005 Oct;32:314-25.)
15. Peterson MW, Spencer MG. Understanding academic culture and climate. New directions for institutional research. 1990 Dec;1990(68):3-18.
16. Tillgren et al 1996). Tillgren P, Haglund BJ, Lundberg M, Romelsjö A. The sociodemographic pattern of tobacco cessation in the 1980s: results from a panel study of living condition surveys in Sweden. Journal of Epidemiology & Community Health. 1996 Dec 1;50(6):625-30

17. Paulander et al 2004). Paulander J, Axelsson P, Lindhe J, Wennström JL. Intra-oral pattern of tooth and periodontal bone loss between the age of 50 and 60 years. A longitudinal prospective study. Acta Odontologica Scandinavica. 2004 Jan 1;62(4):214-22.
18. Ringsdorf WM, Cheraskin E. Emotional status and the periodontium. J Tenn State Dent Assoc 1969; 49: 5–18.
19. Suchday S, Kapur S, Ewart CK, Friedberg JP. Urban stress and health in developing countries: development and validation of a neighbourhood stress index for India. Behav Med 2006; 32: 77–86
20.Somer E, Ben-Aryeh H, Laufer D. Salivary composition gender and psychosocial stress. Int J Psychosom 1993; 40: 17–21.
21. Fournier C, Mascres C. L'influence du stress sur les tissus buccodentaires. J Dent Que 1988; 25: 701–706
22. Cohen-Cole SA, Cogen RB, Stevens AW Jr. Psychiatric psychosocial and endocrine correlates of acute necrotizing ulcerative gingivitis. Psychiatr Med 1983; 1: 215–225.
23. Page RC, Altman LC, Ebersole JL et al. Rapidly progressive periodontitis. A distinct clinical condition. J Periodontol 1983; 54: 197–209.
24. Monteira da silva A, Oakley D, Newmann H, Nohl F, Lloyd H. Psychosocial factors and adult onset rapidly progressive periodontitis. J Clin Periodontol 1996; 23: 789–794.
25. Kamma JJ, Baehni PC. Five-year maintenance follow-up of earlyonset periodontitis patients. J Clin Periodontol 2003
26. Linden G, Mullaly B, Freeman R. Stress and the progression of periodontal disease. J Clin Periodontol 1996; 23: 675–680.
27. Axtelius B, So¨drefeldt B, Nilson A, Edwardson S, Attstro¨m R. Therapy-resistant periodontitis psychosocial characteristics. J Clin Periodontol 1998; 25: 482–491
28. Genco RJ. Relationship of stress, distress and inadequate coping behaviors to periodontal disease. J Periodontol 1999; 70: 711–723.