

Microbial Symbiosis and Dysbiosis-an Overview of Dental Plaque

Nanditha Chandran¹, Arjun MR^{2*}, Subair K¹, Mahesh Raj VV¹, Priscilla Mercy B³, Nikitha³ and Ramana R³

¹Department of Periodontics, Mahe Institute of Dental Sciences & Hospital, Kerala, India

²Department of Periodontics, Mahe Institute of Dental Sciences & Hospital, Kerala, India

³BDS Student, Mahe Institute of Dental Sciences & Hospital, Kerala, India

Abstract

We clinicians assume that the clinical picture of dental disease is a net result of an interaction between the pathogenic dental plaque and host tissue response. Dental plaque biofilm cannot be eliminated. However, the pathogenic nature of the dental plaque biofilm can be reduced by reducing the bio-burden (total microbial load and different pathogenic isolates within that dental plaque biofilm) and maintaining a normal flora with appropriate oral hygiene methods that include daily brushing, flossing and rinsing with antimicrobial mouth rinse. This review is a M Dental Sciences & Hospital, Kerala, India, E-mail: arjunjai2002@gmail.com

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Received: 26-Mar-2022, Manuscript No: johh-22-58657; **Editor assigned:** 31-

Mar-2022, PreQC No. johh-22-58657(PQ); **Reviewed:** 14-Apr-2022, QC No. johh-

Keywords: Symbiosis; Dysbiosis; Plaque; Microorganisms; Bio-film

22-58657; **Revised:** 19-Apr-2022, Manuscript No: Johh-22-58657 (R); **Published:**

Introduction

26-Apr-2022, DOI: 10.4172/2332-0702.1000311

Citation: Chandran N, Arjun MR, Subair K, Mahesh Raj VV, Priscilla Mercy B, Nikitha, Ramana R (2022) Microbial Symbiosis and Dysbiosis-an Overview of Dental Plaque. *J Oral Hyg Health* 10: 311.

The oral cavity is a portal for entry of microorganism which alters the immunity of an individual. Oral bacteria are commensals, which become pathogenic in adverse conditions and cause infections [1]. Dental plaque is a structurally and functionally organized biofilm of diverse microflora [2]. It is a matrix of polymers of bacteria and salivary origin which forms naturally on tooth and defends host by prevention of colonisation of exogenous pathogens [3]. Plaque is natural and contributes to the normal development of the physiology and defences [4].

Definition

A specific but highly variable structural entity resulting from colonisation of microorganisms on the tooth, restorations or other parts of the oral cavity composed of mucin, microorganisms, desquamated epithelial cells and debris all embedded in a gelatinous extracellular matrix.

Classification

Dental Plaque is classified into two categories [5].

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microbiota is found after the emergence, with loss of tooth in old age, some ecological niches such as tooth surfaces and gingival sulcus that favours retention of certain species [6].

Steps in plaque formation

A certain sequence of events is observed in the formation of dental plaque biofilm [3, 5, 11, 12].

Phase 1: Acquired pellicle formation and Transport to the surface

Phase 2: Initial adhesion

Phase 3: Attachment

Phase 4: Colonization and plaque maturation

Acquired pellicle formation and transport to the surface: In the first phase, acquired pellicle formation is seen when bacterial and host products present in the saliva and gingival crevicular fluid come in contact with the tooth surface. In supra-gingival areas, this layer is covered with molecules like salivary glycoproteins, histatin, proline-rich proteins and alpha-amylase. Glucosyl transferases and glycan are also found in the acquired pellicle. Bacteria are transported to the surface of the surface by Random contact– Brownian movement, Sedimentation or active bacterial movement.

unit. In the dental arch, more difference in plaque growth rate can be detected. Plaque formation generally occurs faster in lower jaw when compared with upper Jaw [3].

as well as modulation of the host. In terms of host modulation, *P. gingivalis* facilitates the colonization and growth of other organisms, for example, *S. aureus*, by delaying neutrophil recruitment by transiently inhibiting the initiation of chemokine's like gingival IL8 and T-cell chemokine-like IP 10. It has also shown to affect the function of neutrophils by activating toll-like receptor (TLR) 2 and C5aR. However, the persistence of *P. gingivalis* in the periodontium is dependent on the instigation of incendiary crosstalk seen between receptor of complement C5a and TLR 2 and also the ability of its gingipains to produce C5 convertase activity, which has shown to retard the annihilating ability of leukocytes. This was substantiated by a study in which dysbiosis and periodontitis could not be caused by *P. gingivalis* in C5aR sans host (mice) [23-25].

Conclusion

In the view of the foregoing information, it seems appropriate to conclude that the clinical picture of dental disease is a net result of an interaction between the pathogenic dental plaque and host tissue response. Dental plaque biofilm cannot be eliminated. However, the pathogenic nature of the dental plaque biofilm can be reduced by reducing the bio-burden (total microbial load and different pathogenic isolates within that dental plaque biofilm) and maintaining a normal flora with appropriate oral hygiene methods that include daily brushing, flossing and rinsing with antimicrobial mouth rinse.

Acknowledgement

I, Dr. Arjun MR am very grateful to Dr. Anil Melath, Professor and head of department periodontics, MAHE institute of dental sciences, and Mr. Ramesh Kumar chairman MAHE institute of dental sciences for the support and financial aid.

Conflict of Interest

I declare to have no conflict of interest.

References

1. Saini R, Giri PA, Saini S, Saini SR (2015) Dental plaque:A complex biofilm. Pravara Med Review 7(1): 9-14.
2. Marsh PD and Bradshaw DJ (1995) *ai* and *Å*