Review Article

Microbiological Profile of Chronic and Aggressive Periodontitis- A Review

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ABSTRACT

Periodontitis is an microbial oral infection associated with the destruction of gingiva, cementum, periodontium and alveolar bone process. Periodontitis is broadly classified into chronic and aggressive forms. Both are distinguished in terms of microbiology, immunology, genetic influences and clinical presentation. The etiology of periodontitis would be because of accumulation of bacterial plaque, harbouring variety of pathogenic bacteria termed as periopathogens or periodontopathogens. The periopathogens ivolved in periodontitis are anaerobic bacteria such as Porphyromonas gingivalis, Prevotella intermedia,Tannerella forsythia, Treponema denticola, Fusobacterium nucleatum and Aggregatibacter actinomycetemcomitans. The microbiota of subgingival plaque contains more than 500 species of bacteria, nevertheless research has shown that Porphyromonas gingivalis, a Gram negative anaerobic bacterium, is the etiological agent which contributes to chronic periodontitis and is considered the keystone. Aggressive periodontitis is also influenced by microbiological, genetic, and host factors. The a comparative microbial profile between the two forms of periodontitis with the microbiological aspects of chronic and aggressive periodontitis can be explained in detail in this article.

Key words: Chronic periodontitis, Aggressive periodontitis, Microbiology, Periodontitis.

Introduction

Periodontal disease is a chronic bacterial infection characterized by persistent inflammation of connective tissue breakdown and alveolar bone destruction.[1-3]

Biofilms that colonize the oral cavity are the most complex in nature. Besides pathogenic microorganisms, genetic, environmental factors like smoking, systemic diseases, medications such as steroids, antiepileptic, and drugs for cancer therapy, poor placement of dental bridges, dental crowding, lack of teeth, pregnancy and use of contraceptive pills can contribute to periodontitis[4].

In recent research 800-1000 species were found to colonize the oral cavity among those about 50 species were strongly associated with periodontal disease[5]. The primary bacterial colonizers present in the gingival sulcus produce cytotoxic substances which alter the environment and enhance the colonization of secondary colonizers. These secondary colonizers being more pathogenic exceed its threshold levels in periodontal disease[6].The factors which initiates the periodontal disease might be the virulence factor, their activity, the composition of microbiota and the host immune factors. [7].

The causative organisms that are commonly found in periodontitis are *Porphyromonas gingivalis, Tannerella forsythia, Prevotella intermedia, Campylobacter rectus; Eikenella corrodens, Fusobacterium nucleatum, Actinobacillus actinomycetemcomitans, Peptostreptococcus micros, Treponema denticola, and Eubacterium spp* [8, 9] amongst which the most predominant are the Gram-negative anaerobes are capnophiles, spirochetes which enhances the initiation and progression of the inflammatory process. The bacteria's in the subgingival area increases in count invading the pocket epithelial cells and the underlying tissues. This review is focussed on the microbiological aspects and differences between chronic and aggressive periodontitis

The Implication of Microorganism in Periodontitis

One of the important paradigm shifts that have taken place in the last decade is the recognition and acceptance of the formation of dental plaque as a biofilm. [10] Van Leeuwenhoek in 1683 described micro-organisms in tartar and they called them as animalcules. The role of microorganisms in periodontal disease has been demonstrated through experimental gingivitis and models. Non-specific plaque hypothesis theory states that periodontal disease is a result of noxious product from the entire plaque. [11, 12] But later it was modified as a specific plaque hypothesis stating that destruction to the periodontal structures is caused with minimal deposits. [13, 14]

Earlier studies demonstrated that the number and proportion of different subgingival bacterial groups varied from periodontal health to disease [14]. Bacteria present in periodontally healthy sites were gram-positive facultative rods and cocci (75%) followed by a gradual decrease depending on the site say, gingivitis (44%) and periodontitis sites (10%) similarly there was a proportional increase in gram-negative rods from gingivitis (40%) and periodontitis (74%). [15]

Instead of being associated with one particular etiologic agent, many chronic diseases appear to follow the "microbial shift" hypothesis. Microbial shift (symbiosis) refers to the concept that some diseases are due to a decrease in the number of beneficial symbionts and/or an increase in the number of pathogens. This is termed as ecologic shift hypothesis.[16]

The term "periodontal diseases" includes any inherited or acquired disorders of the tissues that are supporting the teeth (gingiva, cementum, PDL, and alveolar bone) [17]. Chronic periodontitis is characterized by a low to moderate rate of progression that may include episodes of rapid destruction. [17, 18] It is subdivided according to the percentage of the involved sites into localized (<30%) and generalized (>30%). Furthermore, it can be subdivided according to the severity of the disease into mild (1-2 CAL), moderate (3-4 CAL). [19]

The most predominant periopathogen are anaerobic bacteria: *Porphyromonas gingivalis, Prevotella intermedia, Tannerella forsythia, Treponema denticola, Fusobacterium nucleatum*, and the relative anaerobe *Aggregatibacter actinomycetem comitans*. These organisms express a number of potential virulence factors and induce host inflammatory mediators, eventually leading to connective tissue breakdown and alveolar bone resorption [19.20].

Currently, based on the research of Socransky team stands out specific groups of bacteria (called complexes) with particular importance in the pathogenesis of periodontitis. *Porphyromonas gingivalis* creates with the species of *Tannerella forsythia and Treponema denticola* so called red complex, which appears to be associated with disease symptoms in chronic periodontitis [20].

The second important group of bacteria forms a orange complex, comprising 13 species, including *Fusobacterium nucleatum and Prevotella intermedia*. Orange complex bacteria are an essential link in allowing the colonization of periodontal tissue by a red complex [21, 22].

A separate group of bacteria forms a green complex, which includes species: *Capnocytophaga sputigena, C. gingivalis, and Eikenellacorrodens*. These species are also associated with disease symptoms in adult periodontitis, but with a milder clinical course in contrast to the red complex [21, 22].

Microbial Profile Associated with Chronic Periodontitis

Porphyromonas gingivalis

Porphyromonas gingivalis is an intensively studied periodontal pathogen. Isolates of this species are gram-negative, anaerobic, nonmotile, asaccharolytic rods that usually exhibit coccal to short rod morphologies of gingivalis is a member of the much investigated black pigmented Bacteroides group. Organisms of this group form brown to black colonies on blood agar plates and were initially grouped into a single species, Bacteroides melaninogenicus. [23] *P. gingivalis* is has the ability to secrete some virulence factors which penetrates the gingiva and destroys the tissue directly or indirectly, by inflammation. This microorganism has been shown to have extensive virulence factors, including a true collagenase, endotoxin, IgA, proteases, and low-molecular weight compounds including hydrogen sulfide and ammonia which induce bone resorption, destroy connective tissue, induce a variety of cytokines, and inhibit host protective mechanisms. [24]

P. gingivalis lypopolyssacharrides inhibits osteoblastic differentiation and mineralization in periodontal ligament stem cells which participate in periodontal tissue regeneration [24, 25].The "trypsin-like" enzymes cleave polypeptides at the C-terminal after arginine or lysine residue. These proteinases are commonly known as gingipains, namely gingipain R and K that cleave after arginine and lysine, respectively. [25] *P. gingivalis* involve directly in the colonization of the periodontal pocket, leading to the destruction of supporting periodontal tissue. In addition, the proteases also confer high resistance of the microorganisms to host defense mechanism. [26-28]

Gingipain, a virulence factor was found to degrade fibrinogen and host haem proteins which contribute to inhibition of blood coagulation and increase bleeding, thereby enhancing the availability of hemin for bacterial growth [29]. There is a high proliferation rate of *P. gingivalis* within periodontal pockets in which the count of erythrocytes are huge. Gingipains are also considered important in its capacity to degrade antibacterial peptides, such as neutrophil-derived α-defensins, complement

factors, such as C3 and C4, T cell receptors, such as CD4 and CD8 [30].

P. gingivalis is the main causative for chronic periodontitis. This secondary colonizer is found to express a plethora of virulence factors involved in colonizing the sub gingival plaque and modulating the immune responses of the host cells. In order to increase survival into the host, P. gingivalis is able to locally invade periodontal tissue, thereby avoiding the immune surveillance while maintaining its viability. [29]

Tannerella forsythia

T. forsythia has been noted in periodontal health and disease for its fastidious and anaerobic growth requirements for cultural detection. *T. forsythia* with periodontal and other oral infections has used non cultural approaches. [31]

T. forsythia possesses a surface-layer (S-layer) consisting of serrated structural subunits (about 10 nm wide and 10 nm high) in either oblique or tetragonal lattices and it lacks surface appendages such as fimbriae. [13] The S-layer has been shown to be composed of at least two high molecular weight glycoproteins of 220 and 210 kDa size encoded by the tfsA and tfsB genes, respectively [32].

They provide a protective shield containing ion-traps and molecular sieves for metabolites in the environment. T.forsythia S-layer has been shown to promote epithelial cell adherence and invasion [33]. This might promote multiple species biofilm formation observed between the two species and could lead to disease severity [34].

Treponema denticola

Treponemes are members of the Spirochaetes phylum, it is from both Gram-positive and Gram-negative bacteria.*T. denticola* is one member of the oral treponemes. [35] A large body of experimental evidence supports the importance of the oral treponemes, including T. denticola, in the progression of periodontal diseases.[36]

T. denticola bind to a variety of oral surfaces, including the tooth surface, to extracellular matrix proteins, including laminin, fibronectin, and heparin and host cells, such as human gingival fibroblasts. This adherence likely plays an important role in the localization of many bacteria along the matrix border, in close proximity to membrane proteins and other molecules. [37, 38]The collagen binding proteins of T. denticola bind type I, IV, and V collagens, play a role in adherence and colonization by this microorganism. [39] leucine-rich repeat protein (LrrA) play a role in binding to T. forsythia, but not to P. gingivalis or F. nucleatum, and to mediate binding to epithelial cells of p.gingivalis.[39,40]They are also important for epithelial cell invasion and virulence in a mouse alveolar bone loss model by T. forsythia.[40,41] In both of these species, the leucine-rich repeat proteins are members of the CTD family of proteins that are secreted and attached to the surface by novel mechanisms. [40]

HbpA and HbpB are two low iron protein secreted by outer membrane which bind to hemin. These proteins are necessary for efficient iron utilization, although this microorganism has the ability to replace the function of these proteins by accessing a variety of sources of host iron for nutrition [41].

Fusobacterium nucleatum

F. nucleatum belongs to the family bacteroidaceae. Gram-negative, an anaerobic, spindle-shaped rod that has been recognized as a part of the subgingival microbiota for about 100 years [42, 43].

F.nucleatum reacts on an inflammatory response during periodontal disease. Within the gingival epithelium,bacterial biofilm forms at the surface of the tooth containing antimicrobial peptides have a crucial role in the maintenance of periodontal health. Among periodontopathogenic bacteria, all F. nucleatum strains tested and showed the highest sensitivity to hβD-3 and LL37 when compared with those of other bacteria. [44]

Campylobacter rectus

Campylobacter rectus is a putative gram-negative anaerobic, motile, short and rod bacterium which is associated with several forms of human periodontal disease.[45] The surface layer(S-Layer) and cytotoxic activity have been characterized and thought to be a major virulence factors.This s –layer protein is assumed to be involved in résistance of C.rectus to phagocytic uptake and to bactericidal activity of the serum [46,47].

These S- layer proteins consists of a single layer, forms regularly arranged structures on the outer surface of various bacteria. They play a role in virulence of several pathogens by rendering the bacteria resistant in killing and providing structure for adherence to host cells. It was reported that the S-layer negative bacteria were more adherent to human gingival fibroblasts than were other starins of *C.rectus* with intact S-layer [48, 49].

Bacterial adherence is modified by bacterial cell surface structures called adhisions which recognize specific receptors on the particular host cell surface present on the outer membrane [50].

Microbial Profile Associated with Aggressive Periodontitis

Aggressive periodontitis is a disease characterized by a rapid loss of alveolar bone around more than one tooth of the permanent dentition.[14] The amount of destruction is associated with the number of local irritants [15].

Aggressive periodontitis which was called as "juvenile periodontitis" is considered to be prevalent in children and adolescents during the circumpubertal period. It is characterized by rapid loss of connective tissue attachment and alveolar bone with familial aggregation. It is caused by both pathogenic microflora and abnormality in host defense mechanisms. Aggressive periodontitis can be subdivided into localized (LAP) and generalized.

Actinobacillus actinomycetemcomitans was first isolated from a cervicofacial actinomycotic lesion in and initially designated Bacterium actinomycetemcomitam. [51]

Studies have shown that (Faveri M in 2009) the proportions of Aggregatibacter actinomycetemcomitans were elevated in shallow and intermediate pockets of localized aggressive subjects.

Actinobacillus actinomycetemcomitans

This is the primary pathogen for aggressive periodontitis, especially in its localized form. Six serotypes of A.a (a, b, c, d, e, and f) are described based on the composition of O polysaccharide of their lipopolysaccharide and there are phenotypically nonserotypeable strains of A.a which lack expression of serotype-specific polysaccharideantigen.[52] A highly leukotoxic clonal type of A. A serotype b was first isolated, in the early 1980s, from an 8-year-old male child with localized aggressive periodontitis. A significant feature of *A.Actinomycetemcomitans* is its surface ultrastructure which includes fimbriae, vesicles, and extracellular amorphous material. (53, 54)

A prominent feature of the surface of A. actinomycetemcomitans is vesicles. These structures, which are lipopolysaccharide in nature, originate from and are continuous with the outer membrane. Vesicles are also released into the external environment in large numbers. [56] Virulence factors are attributes of a microorganism that enable it to colonize a particular niche in its host, overcome the host defenses and initiate a disease process. These factors frequently involve the ability to be transmitted to susceptible hosts. The virulence of *A.a* has strong assocation with periodontal diseases and related extra oral infections. Many of these virulence factors may be involved in the pathogenesis of periodontitis [55].

In most cases, adhesions are proteinaceous structures found on the surface of the bacterial cell [56]. The adhesion of A. actinomycetemcomitans to the gingival crevice epithelium helps in the colonization of this organism and destruction associated with periodontal disease. *A. actinomycetemcomitans* strains that have been tested adhere strongly to epithelial cells [57].

LPS causes enormous destruction to host cells and tissues leading to periodontal disease. it was found that the prevalence of A.a was less in patients with LAP whereas elevated levels of P.g, Tannerella forsythia, *T.denticola*, *P.intermedia*, and *Campylobacterrectus for detection of microorganisms in subgingival* using polymerixzation chain reaction(pcr)(Takeuchi) .

Summary and Conclusion

However, it is clear that chronic and aggressive forms of periodontitis are not monoinfections. Periodontal disease occurs when there is a disruption in the host microbe homeostasis associated with health. Comparisons of the microbiology of chronic and generalized aggressive forms of periodontitis are in the early phases. It is clear that chronic and generalized aggressive periodontitis are not only caused by gram-negative anaerobic infections, but are also caused by gram-positive bacteria and even non-bacterial microbes from the Archaea domain. Studies have suggested that individuals with generalized aggressive periodontitis have higher subgingival levels of *Selenomonas sp.* and *T. lecithinolyticum* compared to patients with chronic periodontitis.

However, more microbiological data could be generated by combined culture and culture-independent methods from patients with unambiguously defined cases of generalized aggressive periodontitis or chronic periodontitis.

References

1. Brown LJ, Brunelle JA, Kingman A. Periodontal status in the United States, prevalence, extent, and demographic variation. J Dental Res. 1988-1991; 75:672-683.
2. Yamamoto M, Kobayashi R, Kono T, Bolerjack B, Gilbert R.Induction of IL-10- producing CD4 T-cells in Chronic Periodontitis. J Dent. Res. 2011; 90(5):653-658.
3. Suvan J, Aiuto F, Moles DR, Petrie A, Donos N. Association between overweight/obesity and periodontitis in adults. A systematic review. Obes. Rev. 2011; 12(5):381-404.
4. Escudero N, Perea MA, Bascones A. Revision de la periodontitis cronica: Evolucion y su aplicacion. Avances en Periodoncia e Implantología Oral 2008; 20(1):27-37.
5. Colombo AP, Boches SK, Cotton SL, Goodson JM, Kent R, Haffajee AD, et al., Comparisons of subgingival microbial profiles of refractory periodontitis, severe periodontitis, and periodontal health using the human oral microbe identification microarray. J Periodontol. 2009; 80:1421-1432.
6. Socransky S.S, Haffajee A.D. clinical Periodontology and implant Dentistry, 4th ed. J. lindhe, t. Karring, n. Lang, eds.2003; 106-149
7. Lamont RJ, Jenkinson HF. Oral Microbiology at a Glance. Hoboken, NJ, USA: Wiley-Blackwell 2010.
8. N. Lang, P. M. Bartold, M. Cullinan et al., Consensus report: aggressive periodontitis. Annals of Peridontology. 1999; vol. 4, p.53.
9. Socransky S.S., Haffajee A.D. Microbiology of p.gingivalis. Periodontol. 2000, 2002; 28:12-55.
10. Davey ME. Techniques for the growth of Porphyromonas gingivalis biofilms. Periodontol 2000. 2006: 42: 27–35.
11. Syed SA, Loesche WJ: Bacteriology of human experimental gingivitis: effect of plaque age. Infect immum 1978; 21:821.
12. Theilade E, Wright WH, Jensen SB et al., experimental gingivitis in man. II. A longitudinal clinical and bacteriological investigations, J periodontal res 1996; 1:1,
13. Slots J: subgingival microfluroa and periodontal disease, J clin periodontal, 1979; 6.351.
14. Loe H, Theliade E, Jensen SB: Experimental gingivitis in man, J periodontal 1965; 36:177.
15. Socransky SS, Haffajee AD. Periodontal microbial ecology. Periodontol 2000. 2005; 38:135–187.
16. Frank DN, St Amand AL, Feldman RA, Boedeker EC, Harpaz N, Pace NR. Molecular phylogenetic characterization of microbial community imbalances in human inflammatory bowel diseases. Proc Natl Acad Sci U S A. 2007; 104:13780–13785.
17. The American Academy of Periodontology.Periodontal diseases of children and adolescences. J Periodontol. 1996; vol. 67: 57–62.
18. Clerehugh and A. Tugnait, "Diagnosis and management of periodontal diseases in children and adolescents," Periodontology 2000, 2001; vol. 26, no. 1, pp. 146–168.
19. Haffajee AD, Socransky SS, Patel MR, Song X. Microbial complexes in supragingival plaque. Oral Microbiol Immunol. 2008; 23: 1 96-205.
20. Socransky SS, Haffajee AD. Periodontal microbial ecology. Periodontol 2000. 2005; 38: 1 35-1 87.
21. Socransky SS, Haffajee AD, Cugini MA, Smith C,Kent RL Jr. Microbial complexes in subgingival plaque. J Clin Periodontol. 1 998; 25: 1 34-1 44.
22. Socransky SS, Haffajee AD. Periodontal microbial ecology. Periodontol 2000. 2005; 38: 1 35-1 87.
23. Hajishengallis, G., Darveau, R. P., and Curtis, M. A. The keystone-pathogen hypothesis. Nat. Rev. Microbiol. 2012; 10, 717–725.
24. Van Steenbergen, T. J., Delemarre, F. G., Namavar, F., and De Graaff, J. Differences in virulence within the species Bacteroides gingivalis. Antonie Van Leeuwenhoek 1987; 53:233–244.
25. Travis, J., Pike, R., Imamura, T., and Potempa, J. Porphyromonas gingivalis proteinases as virulence factors in the development of periodontitis. J. Periodontal. Res. 1997; 32, 120–125.
26. Andrian E, Grenier D, and Rouabhia M. In vitro models of tissue penetration and destruction by Porphyromonas gingivalis. Infect. Immun. 2004; 72: 4689–4698.
27. Dubin G, Koziel J, Pyrc K, Wladyka B, and Potempa J. Bacterial proteases in disease–role in intracellular survival, evasion of coagulation/fibrinolysis innate defenses, toxicoses and viral infections. Curr. Pharm. Des. 2013; 19, 1090–1113.
28. Potempa J, Banbula A, and Travis J. Role of bacterial proteinases in matrix destruction and modulation of host responses. Periodontol 2000.2012; 153–19.
29. Hajishengallis G, Abe T, Maekawa T, Hajishengallis E, and Lambris J. D. Role of complement in host–microbe homeostasis of the periodontium. Semin. Immunol. 2013; 25, 65–72.
30. Nikaido H. Molecular basis of bacterial outer membrane permeability revisited. Microbiol. Mol. Biol. Rev. 2003; 67: 593–656.
31. Dzink JL, Tanner AC, Haffajee AD, Socransky SS. Gram negative species associated with active destructive periodontal lesions. J Clin Periodontol 1985; 12: 648–659.
32. Lee SW, Sabet M, Um HS, Yang J, Kim HC, Zhu W. Identification and characterization of the genes encoding a unique surface layer of Tannerella forsythia. Gene. 2006; 371:102–111
33. Sharma A, Inagaki S, Sigurdson W, Kuramitsu HK. Synergy between Tannerella forsythia and Fusobacterium nucleatum in biofilm formation. Oral Microbiol Immunol. 2005; 20:39–42
34. Sharma A, Sojar HT, Glurich I, Honma K, Kuramitsu HK, Genco RJ. Cloning, expression, and sequencing of a cell surface antigen containing a leucine-rich repeat motif from Bacteroides forsythus ATCC 43037. Infect Immun. 1998; 66:5703–5710.
35. Grenier D. Characterization of the trypsin-like activity of Bacteroides forsythus. Microbiology 1995: 141: 921–926.
36. Cimasoni G, McBride BC. Adherence of Treponema denticola to modified hydroxyapatite. J Dent Res. 1987; 66:1727–1729.
37. Haapasalo M, Muller KH, Uitto VJ, Leung WK, McBride BC. Characterization, cloning, and binding properties of the major 53-kilodalton Treponema denticola surface antigen. Infect Immun. 1992; 60: 2058–2065.

38. Li M, Liu Z, Umemoto T. Collagen-binding proteins of human oral spirochetes. Zhonghua Kuo Qiang Yi Xue Za Zhi 1999: 34: 165–167.

39. Ikegami A, Honma K, Sharma A, Kuramitsu HK. Multiple functions of the leucine-rich repeat protein LrrA of Treponema denticola. Infect Immun. 2004; 72:4619-4627.

40. Rosen G, Genzler T, Sela MN.Coaggregation of Treponema denticola with Porphyromonas gingivalis and Fusobacterium nucleatum is mediated by the major outer sheath protein of Treponema denticola. FEMS Microbiol Lett. 2008; 289:59-66.

41. Hofstad T. The genus Fusobacterium, In M. P. Starr, H. Stolp, H. G. Tru¨per, A. Balows, and H. G. Schlegel., the procaryotes. A handbook on habitats, isolation, and identification of bacteria. 1981. Springer Verlag p. 1464–1467.

42. Moore, W. E. C., L. V. Holdeman, and R. W. Kelley. Genus II. Fusobacterium Knorr 1922, In N. R. Krieg and J. G. Holt (ed.), Bergey's manual of systematic bacteriology. 1984 vol. 14 A, p. 631–637.

43. Dzink JL, Tanner ACR, Haffajee AD, Socransky SS. Gram negative species associated with active destructive periodontal lesions. J Clin Periodontol. 1985; 12: 648-659

44. Kolenbrander PE. Oral microbial communities: biofilms, interactions, and genetic systems. Annu Rev Microbiol. 2000; 54: 413-37.

45. Vandamme P, falson E, Rossau R et al revision of camphylobacter, helicobacter, and wolinella taxonomy: emendation of generic descriptions and proposal of arcobacter gen. nov. int J syst bacteriod. 1991;41(1): 88-103.

46. Dzink JL. Socranskey SS. Haffajee AD,the predominant cultivable microbiota of active and inactive lesions of destructive periodontal disesases. J clin periodontal. 1988:15(5); 316-323.

47. Rams TE, Feik D, Slots J. campylobacter rectus in human periodontitis. Oral microbiol immunol. 1993; 8(4): 230-235.

48. Beveridge TJ, Pouwels PH, Sara M.et al. Functions of S-layer. FEMS Microbiol Rev. 1997; 20: (1-2)99-149.

49. Nitta H, Holt SC, Ebersole JL,. Purification and characterization of campylobacter rectus surface layer protein. Infect Immun. 1997;65(2): 478-483.

50. Pei ZH, Ellison RT, 3rd, Blaster MJ. Identification, purification, and characterization of major antigenic proteins of campylobacter jejuni. J boil chem. 1991; 266(25): 16363-16369.

51. Zambon JJ. Actinobacillus actinomycetemcomitans in human periodontal disease. J Clin Periodontol. l985; 12: 1-20.

52. Meyer DH, Fives-Taylor PM. Characteristics of adherence of Actinobacillus actinornyceterncomitans to epithelial cells. Infect Immun. 1994; 62: 928-935.

53. Holt SC, Tanner AC, Socransky SS. Morphology and ultrastructure of oral strains of Actinobacillus actinomycetemcomitans and Haemophilus aphrophilus. Infect Immun. 1980; 30: 588-800.

54. Holt SC, Tanner AC, Socransky SS. Morphology and ultrastructure of oral strains of Actinobacillus actinomycetem comitans and Haemophilus aphrophilus. Infect Immun.1980; 30: 588-800.

55. Fives-Taylor P, Meyer D, Mintz K. Characteristics of actinobacillus actinomycetemcomitans invasion of and adhesion to cultured epithelial cells. Adv Dent Res. 1995; 9: 55-62.

56. Rosan B, Slots J, Lamont RJ, Listgarten MA, Nelson GM. Actinobacillus actinornyceterncornitans fimbriae. Oral Microbiol Immunol. 1988; 3: 58-63.

57. Meyer DH, Fives-Taylor PM. Characteristics of adherence of Actinobacillus actinornyceterncomitans to epithelial cells. Infect Immun. 1994; 62: 928-935.

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