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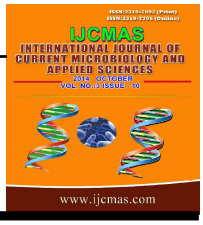

Review Article

Periodontopathic Bacteria: A Microbiological View

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A B S T R A C T

The periodontal disease is a chronic, degenerative disease which is localised on the gingiva, periodontal ligament, cementum and alveolar bone. The composition of the subgingival microbial flora and the level of pathogenic species differ from subject to subject as well as from site to site. The search for the pathogens of periodontal diseases has been underway for more than 100 years, and continues up today. The periopathogens of the oral microbial community may also disrupt the homeostatic relationship between dental plaque and the host. Bacteria are the prime etiological agents in periodontal diseases. *The currently recognized key Gram negative periodontopathogens include: Porphyromonas gingivalis, Prevotella intermedia, Bacteroides forsythus, Aggregatibacter actinomycetemcomitans, Fusobacterium nucleatum, Capnocytophaga species.*

Introduction

Periodontitis, a biofilm-associated inflammatory disease of the periodontium, is a major cause of tooth loss in the world. This disease appears to have multiple etiologies, the most studied of which are microbial pathogens and immunological.

The primary microbial factor contributing to disease is the oral microflora, while the primary immunological factor is the destructive host inflammatory response. Several techniques have been used clinically to treat periodontitis, but the most successful ones appear to address both the bacterial and inflammatory components of the condition. Therefore, this review will detail the microbial pathogens and molecular nature of periodontitis, and it will also compare the efficacy of traditional and emerging technologies for treating this costly disease.
Periodontitis infection of the tooth-supporting tissues, results from the accumulation of pathogenic bacterial plaque at and below the gingival margin (Pihlstrom, B.L et al., 2005).

Per·i·o·don·to·path·ic means that which cau ses pathologic states in the peridontium. It ty pically describes bacteria that cause periodo ntaldisease (Medical Dictionary for the

Dental Professions © Farlex 2012). The major periodontal pathogens are *Aggregatibacter* (formerly *Actinobacillus*) *actinomycetemcomitans, Porphyromonas gingivalis, Prevotella intermedia, Tannerella forsythia* (previously *forsythensis*), *Campylobacter rectus*, and *Treponema denticola* (American Academy of Periodontology. 1996, Haffajee, A. D., and S. S. Socransky. 1994, van Winkelhoff, A. J., 2002). In subgingival plaque, *P. gingivalis, T. forsythia*, and *T. denticola* have the strongest relation to periodontal tissue destruction (Socransky, S. S., et al 1998). Periodontal bacteria have different carriage profiles depending on the age, educational level, and periodontal status of the subjects (Kononen, E., et al 2007).

Source

For the present study searched the Pub Med database from 1992 through 2010 (both years included) for English language articles using the following search terms: 'Periodontal disease and Periodontitis', 'periodontal Infection', 'periodontal pathogens or periodontopathic, '*P. gingivalis* and Lipopolysaccharides. We selected and reviewed cross-sectional, longitudinal, cohort and *in vitro* studies that provided information related to periodontal infection and periodontopathic.

Periodontal Pathogens

Periodontal pathogens are bacteria that have been shown to significantly contribute to periodontitis. Although approximately 700 bacterial species have been identified in the oralcavity and nearly 300 species have been cultured and found to contribute to the biofilm of the periodontal pocket, there is a much smaller number of species that have been shown to be more closely related to the initial incidence and continued persistence of periodontitis, including (Picolos, DK., *et al*. 2005). The bacteria can irritate and inflame the gums below your teeth causing gingivitis. And when gingivitis become worse, bacteria penetrate further until it hits the deeper pockets to where the tissues layers and bones are at (periodontal membrane that holds the teeth in place)--causing periodontal disease that can lead to tooth loss.

The Microbiology of Oral Disease

The teeth are the only nonshedding surfaces in the body, and bacterial levels can reach more than 10^{11} microorganisms per mg of dental plaque. Human endodontal and periodontal infections are associated with complex microfloras in which approximately 200 species (in apical periodontitis) (Tronstad L. *1992*) *a*nd more than 500 species (in marginal periodontitis) (Moore W. E. C., Moore L. V. H 1994) have been encountered. These infections are predominantly anaerobic, with gram-negative rods being the most common isolates. The anatomic closeness of these microfloras to the bloodstream can facilitate bacteremia and systemic spread of bacterial products, components, and immunocomplexes.

Traditionally, the field of microbiology has focused on studying bacteria in planktonic culture-that is, in test tubes. However, the bacteria form microcolonies which then secrete a sticky extracellular polymeric substance which consists of polysaccharides, proteins, lipids, nucleic acids, and other polymers, and it helps the bacteria adhere to the surface, as well as to each other (Davey ME, O'Toole GA. 2000, Liu YQ, Liu Y, Tay JH. 2004). Upon secretion of the extracellular polymeric substance, the biofilm matures by becoming larger and taking on a distinctive architecture (Davey

ME, O'Toole GA. 2000). Usually, this structure includes separate regions of fast- and slow-growing cells (An D, Parsek MR. 2007), the presence of water channels which circulate metabolites (Davey ME, O'Toole GA. 2000), and the establishment of nutrient gradients (Hall-Stoodley L, Stoodley P. 2009). Such complex structural organization allows the biofilm to exhibit functional heterogeneity.

The process of biofilm formation. Initially, individual bacterial cells attach to a surface. These cells then produce a sticky extracellular polymeric substance, which aids in attachment and allows the biofilm to grow larger. From the oral cavity to the intestinal tract, the human body constantly interacts with the biofilms that constitute our normal microbial flora. Research has shown that the composition of this microflora is closely tied to the health status of the host.

In the case of the oral cavity, attempts to characterize the normal microbial flora have met with challenges. First, over 700 species have been detected in the oral cavity, over half of which have never been cultivated (Aas J A et al., 2005). Second, there is substantial diversity in the content of the microflora between individuals (Nasidze I et al., 2009) and between different oral sites within the same individual (Aas J A et al., 2005, Avila M et al., 2009).

Third, view has indicated that dietary changes combined with poor hygiene can cause a shift in the composition of the oral microflora (Al-Ahmad A et al 2009, Avila M et al., 2009). Finally, some evidence suggests that the oral microbiome changes as humans age (Kang JG et al., 2006). Such variation makes it difficult to identify a "typical" oral microbiome for a healthy individual.

The Role of LPS in the Outer Membrane of Gram-negative Bacteria

The function of the outer membrane of Gram-negative bacteria is to act as a protective permeability barrier. The outer membrane is impermeable to large molecules and hydrophobic compounds from the environment. LPS is essential to the function of the outer membrane. First, it establishes a permeability barrier that is permeable only to low molecular weight, hydrophilic molecules. In the *E. coli* the ompF and ompC porins exclude passage of all hydrophobic molecules and any hydrophilic molecules greater than a molecular weight of about 700 daltons. This prevents penetration of the bacteria by bile salts and other toxic molecules from the GI tract. It also a barrier to lysozyme and many antimicrobial agents. Second, in an animal host, it may impede destruction of the bacterial cells by serum components and phagocytic cells. Third, LPS may play a role as an adhesin used in colonization of the host. Lastly, variations in LPS structure provide for the existence of different antigenic strains of a pathogen that may be able to bypass a previous immunological response to a related strain (Christian R. H. Raetz and Chris Whitfield. 2002). For example, the lipopolysaccharide (LPS) of *P. gingivalis* revealed an unusually low inflammatory potency, and, in fact, a *P. gingivalis* lipid A structure acting as TLR4 antagonist that inhibits inflammation has been discovered and characterized (Darveau *et al*., 1995).

Lipid A is the lipid component of LPS. It contains the hydrophobic, membrane-anchoring region of LPS. Lipid A consists of a phosphorylated N-acetylglucosamine (NAG) dimer with 6 or 7 fatty acids (FA) attached. Usually 6 FA are found. All FA in Lipid A are saturated. Some FA are attached

directly to the NAG dimer and others are esterified to the 3-hydroxy fatty acids that are characteristically present. The structure of Lipid A is highly conserved among gram-negative bacteria. Among *Enterobacteriaceae* Lipid A is virtually constant.

The primary structure of Lipid A has been elucidated and Lipid A has been chemically synthesized. Its biological activity appears to depend on a peculiar conformation that is determined by the glucosamine disaccharide, the PO_4 groups, the acyl chains, and also the KDO-containing inner core.

The literature as it currently stands appears to indicate that oral diseases, or pathogenic bacteria, is at least partially responsible for the development of periodontitis. However, despite great advances in our knowledge of the underlying microbial basis of this disease, the fact still remains that periodontitis has multiple etiologies which have yet to be fully understood. Thus, while a microbial pathogens is known to play a significant role in the development of periodontitis, it must also be investigated in order for clinicians and researchers to fully understand disease progression. Because of the various risk factors that contribute to periodontitis, it is possible that there will be no "magic bullet" treatment. It is also likely true that the underlying cause of periodontitis is different in different patients. For instance, one patient's periodontitis may be due to the oral microflora due to poor hygiene, while another patient's periodontitis may be due to an underlying genetic abnormality that leads to a destructive immune response. In light of this, periodontitis is perhaps better described not as a disease but as a symptom of an underlying condition. For successful treatment, it is imperative that this underlying cause be identified and addressed. Indeed, the complexity of periodontitis emphasizes the necessity of "individualized medicine" and implementing a treatment that is highly tailored to the specific needs of the patient.

Overall, the goal is to find the best treatment. From a biological perspective, the most successful treatments will likely need to attack the integrity of the periodontal biofilm and suppress the destructive host inflammatory response. From a clinical perspective, the best treatments are those that are simple, affordable, and able to confer a clinically relevant benefit to the patient.

Conversely, although our views are consistent with alterations in the oral commensal bacterial community being directly responsible for disease, we cannot exclude the possibility that a low-abundance pathogen is also increased in the presence of *P. gingivalis* and may have direct effects on host periodontal function.

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