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Porphyromonas gingivalis and ***Porphyromonas endodontalis*** Taylor Veltmeyer Bench C 23/09/2016 [1]

Contents

[hide]

[edit] Classification

[edit] Higher order taxa

Kingdom: Bacteria Phylum: Bacteroidetes Class: Bacteroidetes Order: Bacteroidales Family: Porphyromonadaceae Genus: Porphyromonas

[edit] Species

Porphyromonas gingivalis Type Strain: strain 2561 = ATCC 33277= CCUG 25893 = CCUG 25928 = CIP 103683 = DSM 20709 = JCM 12257 = NCTC 11834. Sequence accession no. (16S rRNA gene) for the type strain: AB035459. Basonym: Bacteroides gingivalis Coykendall et al. 1980.

Porphyromonas endodontalis Type strain: strain HG370 = ATCC 35406= JCM 8526 = NCTC 13058. Sequence accession no. (16S rRNA gene) for the type strain: AY253728. Basonym: Bacteroides endodontalis van Steenbergen et al. 1984.

[edit] Description and significance

Porphyromonas gingivalis and *Porphyromonas endodontalis* are two species that belong to the phylum Bacteriodetes. For the purpose of this study, the main focus will be *Porphyromonas gingivalis* as both species have very similar attributes, however *P.*

gingivalis is the most prevalent and primary cause of disease, *P. endodontalis* is an associated organism involved in the disease and differences amongst the two will be mentioned in detail. ***Porphyromonas gingivalis*** is a Gram-negative anaerobic bacterium, that is non-motile and rod-shaped and is also the major etiological causative agent of chronic periodontitis. *P. gingivalis* is commonly found in the oral cavity and implicated in certain forms of periodontal disease. Also found in upper gastrointestinal tract, respiratory tract and colon. It has also been isolated from women with bacterial vaginosis. *P. endodontalis* is commonly found in infected dental root canals and submucous abscesses of endodontal origin. It is also occasionally found on oral mucous membranes and periodontal pockets. Genome was first sequenced and cultured by the American Type Culture Collection (ATCC), and it is important as *P. gingivalis* is not only isolated to the oral cavity/mouth. Both strains of Porphyromonas have been cultured in lab and both produce black-pigmented bacteria, however culturing is not the best way of identifying the species, Polymerase Chain Reaction produces a higher frequency of species production. The importance of studying disesease associated with *P. gingivalis* is that according to the World Health Organisation (WHO), periodontal disease affects 10-15% of the adult population worldwide [1].

[edit] Genome structure

The genome of *P. gingivalis* has been described in 2003 and revealed 1,990 open reading frames (i.e. protein-coding sequences), encoded by 2,343,479 bp, with an average G+C content of 48.3%. An estimated 463 genes are essential and the origin of replication is located at oriC which is juxtaposed by the genes dnaA and PG1949 [2].

[edit] Cell structure and metabolism

The cell wall struture for *P. gingivalis* is similar to that of other gram-negative bacteria. It has a thin peptidoglycan layer in the periplasmic layer between the inner and outer lipid membranes. The cell wall comprises of two cell membranes, the inner membrane and the outer membrane and both have different composition. The inner membrane is a phospholipid bilayer that contains numerous integral inner membrane proteins [3], whilst the outer membrane is an asymmetrical bilayer, meaning it consists of phospholipids and lipopolysaccharides in the inner and outer leaflets, respectively. The cell membrane is also a selective barrier used to protect and facilitate ,movement of specific substances through outer membrane porins [4]. The formation of biofilm is of high importance to *P. gingivalis* as it enhances the transmission, but more importantly, the pathogenicity of the infective pathogen strain. It also aids in protecting the species from human immune responses and antibiotics. Dental plaque is a common form of biofilm of the teeth. The accumulation of microorganisms subjects the teeth and gingival tissues to high concentrations of bacterial metabolites which results in dental disease [5]. Formation and maintenance of the periodontal biofilms is associated with periodontal microflora interaction, which is mediated by outer membrane proteins [3]. This means that patients with periodontitis will produce higher levels of cytokines, including Interleukin (IL-), 1β and 6 as a response to the biofilm periodontitis infection through the T helper cells [6]. *Porphyromonas* species are non-motile bacterium, however not all bacteriodetes share this quality. A study conducted by Nakayama K. in 2014 showed that

Porphyromonas species, including *P. gingivalis* and other bacteriodetes, contain the type IV secretion system (T9SS). T9SS in gram-negative bacterial organisms has been linked to gliding motility, as the bacterium is motile but there is no motility components (eg. flagella or type IV pili), that are influencing the motility [7]. This leads to the postulation that since the phylum bacteriodetes all share origin and all contain T9SS, that genetic adaptation and mutation may occur making *Porphyromonas gingivalis* and other bacterium motile. *P. gingivalis* utilises iron in the form of heme transported from hemin through ABC Transporters. Outer membrane receptors, proteases (eg. gingipains) and lipoproteins are used to collect iron/heme as *P. gingivalis* doesn't produce siderophores. Proteolytic activities of gingipan R and K assist in maturation of various cell surface proteins (eg. fimA fimbrilin major fimbriae, 75-kDa protein subunit minor fimbriae, hemagglutinins and haemoglobin receptor proteins), [8]. *Porphyromonas gingivalis* also lives in metabolic symbioses with *Treponema denticola*, both exhibit symbiosis in growth and synergistic virulence upon co-infection [9].

[edit] Ecology

Porphyromonas gingivalis is classified as an obligate anaerobic microorganism based on its oxygen requirements during infection. It commonly inhabits the oral cavity and is implicated in a number of disease, including periodontal disease and gingivitis. Have also been found outside the microbiota in the upper gastrointestinal tract, the respiratory tract, the colon and has even been isolated from women with bacterial vaginosis [10]. Establishing a connection between *Porphyromonas gingivalis* and the interactions with the host is paramount as it aids in elucidating therapeutical approaches for assistance in periodontal infection. *Porphyromonas gingivalis* interacts with the host species through the cellular structures. The most critical factor for disease manifestation is the fimbriae, as it promotes the invasion of the targeted sites for infection as well as bacterial adhesion to the infected area. It is also essential for interruption of the cellular signalling pathway via extracellular matrix proteins/integrins and for the bacterial organism invasive event to host [11]. The complement system is affected in the signalling pathway, *Porphyromonas gingivalis* fimbriae bind CD14 and activate Toll-like receptor 2 (TLR2) and phosphatidylinositol 3-kinase-mediated signalling. This leads to an induction of the high-affinity conformation of CR3 in leukocytes. This is usually standard in enhancing cell interaction between leukocytes and endothelial and aids in migration. *Porphyromonas gingivalis* have co-opted this pathway for fimbriae adhesion with CR3; this induces downregulation of IL-12 p70 which is a cytokine that would generally target this pathogen for destruction by macrophages [12]. Another feature of *P. gingivalis* is that it exploits complement-TLR crosstalk to deviate defences and escape termination. It does this by expressing C5 convertase-like enzymatic activity and this leads to remodelling of periodontal microbiota to a dysbiotic state which is the predominant cause of inflammatory periodontitis [13].

[edit] Pathology

As stated earlier, *Porphyromonas gingivalis* infections are predominant in the oral cavity of the mouth and cause of periodontal disease and gingivitis. Virulence factors that lead to disease include capsules, fimbriae, lipopolysaccharides, proteases and the

outer membrane proteins. The microbiota of the mouth contains over 700 bacterial species and despite this diversity, the oral cavity is a stable community of microbiome life. Imbalance in microbiota leads to disease and change to the species in the gingival sulcus is, more often, from gram-positive, facultative, fermentative microorganisms to predominantly gram-negative, anaerobic, chemoorganotrophic, proteolytic organisms and this is highly associated with the destruction of periodontal tissue [14]. Periodontal disease refers to the inflammatory pathologic state of gingiva and supporting structures, (eg. gingival, alveolar bone, periodontal ligament, and cementum), and are broken down into two major categories; Gingival disease and Periodontitis [15]. Gingival disease classified by inflammation of gingival tissue by accumulation of dental plaque. Attachment of the teeth is unaffected and can remain indefinitely without causing periodontitis [16]. On the contrary, periodontitis is classified by irreversible plaque-induced inflammation of periodontal tissue causing destruction of periodontal ligaments and alveolar bones. This causes formation of periodontal pockets and subgingival plaque, [16]. *Porphyromonas gingivalis* is not only isolated to the mouth, also cause bacterial vaginosis in women.

[edit] Application to biotechnology

A number of methods are used to colonize *Porphyromonas gingivalis*, including culturing in vivo and in vitro, as well as Polymerase Chain Reaction. PCR has a much higher frequency of determining pathogens present as oppose to either of the culturing methods [17]. There are a number of potential targets for developing therapeutic drugs to combat *P. gingivalis*. The first involves biofilm formation and bacterial dipeptidyl peptidase IV (DPPIV) activity as these both contribute to the pathogenic potential of *P. gingivalis* [18]. The second target for therapeutic drug targeting is the cysteine proteases as the enzymes involved are both involved in the destruction of periodontal tissues and interrupting host-defence mechanisms through the degradation of both complement factors and immunoglobulins which leads to disease [19]. The third target would be through the Toll-like Receptors and activated CR3 that P. gingivalis uses to co-opt and optimize progression of disease [20].

[edit] Current research

Current research includes the aforementioned study of gram-negative bacteriodetes being classed as gliding motility [7]. Various studies conducted show a causal link between *Porphyromonas gingivalis* infections and systemic disease patients, including diabetes mellitus, AIDS, leukemia, and Down's syndrome [21;22]. The most alarming research to date is the connection *P. gingivalis* infections have with cardiovascular disease, (heart attacks, coronary artery diseases and strokes), and diabetes [23;24]. Research from the AAP said that it people with any form of gum disease are twice as likely to suffer from coronary heart disease as oppose to people who have healthy gums.

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1. ↥ MICR3004

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