Content available at: iponlinejournal.com

IP International Journal of Periodontology and Implantology

Journal homepage: www.innovativepublication.com

Review Article

The red bacteria: Porphyromonas gingivalis

Samriti Katoch[1,*]

[1] *Dept. of Periodontology, Seema Dental College and Hospital, Rishikesh, Uttarakhand, India*

ARTICLE INFO

Article history:
Received 28/11/2019
Accepted 17/12/2019
Available online 07-01-2020

Keywords:
Porphyromonas gingivalis
Chronic Periodontitis
Red Bacteria

ABSTRACT

Porphyromonas gingivalis is a gram negative, anerobic, non-spore & nonmotile rods or cocco-bacilli. It is a major periodontopathic bacteria involved in the pathogenesis of periodontal disease. It is considered as a major causative agent of chronic periodontitis. It contains a wide variety of virulence factors that helps it in colonization and evasion of host defences.

1. Introduction

Periodontal disease are a group of infectious, inflammatory disease with different clinical presentations and variety of different pathogenic mechanisms. According to the World Health Organization, periodontal disease affects 10–15% of adult populations worldwide.[1] (How KY, Song KP, Chan KG. Porphyromonas gingivalis : An overview of periodontopathic pathogen below the gum line. Front Microbiol 2017;7:53) In today's world the main dilemma of a periodontist is how to make a reasoned decision within the best interests of the patient despite so much uncertainty in the clinical information as well the clinical outcome. A successful treatment requires a correct diagnosis, that can only be achieved via thorough understanding of the complex pathobiology of the pathogenesis of periodontal disease. Although there is a vast variety of microorganisms involved in the etiology of periodontal disease. Porphyromonas gingivalis is a major periodontopathic bacteria involved in the pathogenesis of periodontal disease.[2]

Porphyromonas gingivalis is a gram negat ive, anerobic, black pigmented rod and is a member of the red complex along with Treponema denticola and Tannerella forsythia. It is considered as a major causative agent of chronic periodontitis. It is an opportunistic colonizing well adapted pathogen with the ability to invade gingival epithelial cells, periodontal ligament fibroblasts, osteoblasts and immune cells. It requires hemin and vitamin K in its nutrient medium. It appears as black-pigmented colonies in blood agar medium. It obtains energy through the fermentation of amino acids. I t is thought that infections with this organism induce the production of proinflammatory cytokines that damage the host tissue, promoting bacterial survival. It is normally found in 10%–20% of healthy subjects and 80%–90% of subjects with periodontitis. There is a positive correlation between the depth of the periodontal pocket and the presence of this bacteria.[3](Kívia Queiroz de Andrade, Cássio Luiz Coutinho Almeida-da-Silva, and Robson Coutinho-Silva, "Immunological Pathways Triggered by Porphyromonas gingivalis and Fusobacterium nucleatum: Therapeutic Possibilities?," Mediators of Inflammation, vol. 2019, Article ID 7241312, 20).

1.1. Species of the genus porphyromonas

The genus Porphyromonas includes both pigmented & non-pigmented asaccharolytic species. Members of this genus are 0.5-0.8 X 1.0-3.5 pm diameter, obligate anaerobes, non-spore & nonmotile rods or cocco -bacilli exhibiting smooth,

* *Corresponding author.*
E-mail address: smritikatoch293@gmail.com (S. Katoch).

https://doi.org/10.18231/j.ijpi.2019.026

raised colonies. The species of this genus are known for production of large amount of cell associated protoheme. On blood agar, initially the colonies are white to crème coloured. After 4 – 8 days the colonies darken to a deep red to black colour.[4]

2. Biochemical properties

P gingivalis is an asaccharolytic, anaerobic species and colonizes sites with low oxygen tension and abundance of nitrogenous substrates. The subgingival ecosystem provides an ideal environment for the growth of t his species due to low redox potential. Arginine is the primary substrate for this species. Endogenous nutrients are both rich in peptides and amino acids. Phenylacetic acid is the metabolic end product.[4]

2.1. Virulence factors of P gingivalis

The virulence attributes of microbial pathogens include the ability :

1. Enter a host
2. Find a unique ecological niche
3. Circumvent or subvert the host's normal defenses
4. Replicate in the new environment
5. Express specialized pathogenic traits.

Virulence factors of P gingivalis can be characterised as following:

1. Involved in attachment & colonization
2. Involved in evading host responses
3. Involved in damaging host tissues

2.2. Capsule

The presence of a capsule in P gingivalis has been considered an important anti phagocytic virulence factor. Some strains appear to be devoid of such a thick capsule. The electron dense-ruthenium red staining layer is the polysaccharide capsule. KI – K6 are the capsular serotypes. The chemical composition comprises galactose, glucose, glucosamine, rhamnose, mannose, methylpentose and galactosamine. The highly encapsulated P gingivalis strains exhibit decreased autoagglutination, lower buoyant densities increased resistance to phagocytosis, serum resistance. They were more hydrophilic than the less encapsulated strains.[5]

2.3. Fimbriae

All of the P gingivalis strains contain fimbriae arranged in a peritrichous fashion over the surface of the cell. Length varies from 0.5 – 1.6 μm in length & 5 nm in width approximately. Fimbriae are composed of fimbrillin monomers 45 kDa approx, which are assembled into nine units per turn of the mature hairlike helix tertiary structure. They facilitate adherence to salivary proteins, extracellular matrix, eukaryotic cells and bacteria of either the same or other species.[4] They also seem to have chemotactic ability. Both major and minor fimbriae are involved in the induction of proinflammatory cytokines and production of matrix metalloproteinases (MMPs), such as IL-1, IL-6, IL-8, TNF -α and MMP-9, by various host cells. Porphyromonas gingivalis fimbriae can signal through either TLR2 or TLR4. Two different signalling pathways can be induced by fimbriae, one mediates production of proinflammatory cytokines, such as IL-6 and TNF-α, and another that mediates the expression of cell adhesion molecules, such as ICAM-1.[7]

This putative periodontal pathogen expresses three distinct fimbriae - long fimbriae encoded by fim A gene, short fimbriae encoded by the mfa1 gene and accessory fimbriae encoded by fim C,D,E gene.[8]

2.4. Lipopolysaccharide: Accessory Fimbriae (Fim C,D,E)

It is the major macromolecule, amphipathic in character found on the outer surface of gram-negative bacteria. It is typically composed of three domains: Lipid A (hydrophobic end), a short core oligosaccharide, and an O-antigen (hydrophilic end). LPS helps the bacteria to maintain its structural integrity and establishes a selective permeability barrier that limits the entry of hydrophobic molecules. Endotoxic activity is confined to the lipid-A, while significant immunobiological activity is contained within the O -Antigen.[9]

2.5. Proteinases

There are two distinct families of proteases produced by P gingivalis namely as cysteine (trypsin-like) proteases & serine proteases. P gingivalis proteases are involved in activation of the host matrix metalloproteinases, degradation of extracellular matrix proteins such as collagen, inactivation of plasma proteinase inhibitors & cleavage of cell surface receptors.

2.6. Gingipains

They constitute a group of cysteine endopeptidases produced by P gingivalis and are product of three genes referred to as gingipain R(rgp), Arg/ Lys specific gingipains (rgpA), (rgpB), gingipain K (kgp). Gingipains degrade fibrinogen and host heme proteins which contribute to inhibition of blood coagulation and increase bleeding, thereby enhancing the availability of hemin for bacterial growth. Gingipains are also involved in degradation of antibacterial peptides, complement factors, such as C3 and C4, T cell receptors, such as CD4 and CD8.[10]

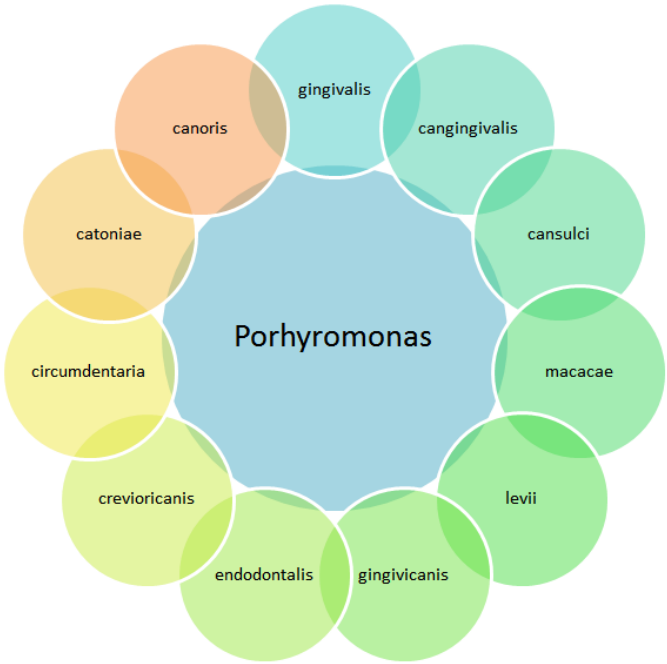

Fig. 1: Porphyromonas gingivalis species.[4]

Table 1: Virulence facto rs of Porphyromonas gingivalis[5]

Tissue destruction	Host evasion
Collagenase Trypsin-like protease Gelatinase Aminopeptidase Phospholipase A Alkaline phosphatase Acid-phosphatase-Chondroitin sulfatase Hyaluronidase Keratinase Heparinase	Degradation of plasma protease inhibitors Degradation of iron transport proteins Inhibition of polymorphonuclear leukocytes Chemotaxis inhibitors Decrease phagocytosis Lysis and intracellular killing Fibrinolysin Free-radical formation

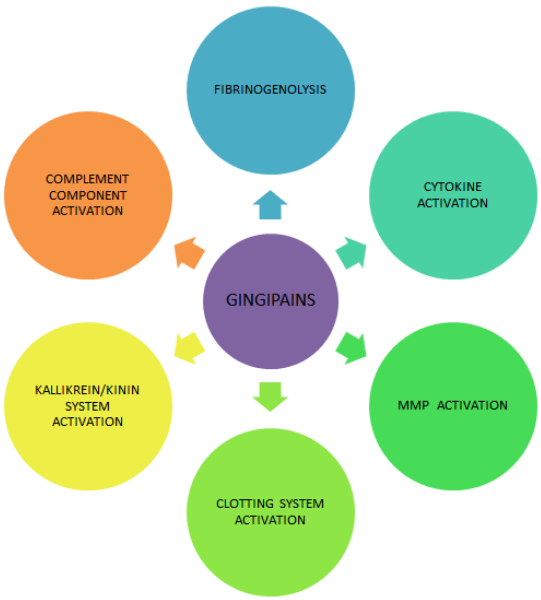

Fig. 2: Pathogenic activities of Gingipains[6] (Modified from Potempa J, Banbula A, Travis J. Role of bacterial proteinases in matrix destruction and modulation of host responses. Periodontol 2000 2000;24:153-192.

2.7. Relationship with periodontal disease

Subgingival microbiota of periodontal healthy children and adolescents harbour very few P gingivalis. However it is found as the most predominant organism in aggressive periodontitis cases. Adults with healthy periodontium reveal subgingival P *gingivalis* in less than 10% of sites. Chronic periodontitis patients may reveal 40-100% sites positive for this organism. Deep periodontal pockets harbour a considerably higher proportion of P *gingivalis* as compared to shallow periodontal pockets. The high pathogenic potential and abundance of this organism in chronic periodontitis lesions are the main reasons for designating the species as a putative periodontal pathogen.[11]

3. Conclusion

So we can say that Porphyromonas gingivalis possesses all of the chemical and biological characteristics that make it an important member of the periodontopathic microbiota. Its infection is related to a typical periodontal eco-pathology so this pathogen may be controlled by periodontal treatment and emphasizing the significance of high standard oral hygiene. The most promising approach to the control of periodontal infections would appear to be by controlling their ecology. Substantial data accumulated over the years has implicated the involvement of only a small proportion of bacteria, which reside in the subgingival niche, in the initiation and progression of periodontal disease. There is strong evidence that points to Porphyromonas gingivalis as the keystone species in the development of chronic periodontitis.

4. Source of funding

None

5. Conflict of interest

None

References

1. How KY, Song KP, Chan KG. Porphyromonas gingivalis: An overview of periodontopathic pathogen below the gum line. *Front Microbiol*. 2017;7:53.
2. Slots J. Actinobacillus actinomycetemcomitans and Porphyromonas gingivalis in periodontal disease: introduction. *Periodontol*. 1999;20:7–13.
3. De KQA, Almeida-Da-Silva C, Coutinho-Silva R. Immunological Pathways Triggered by Porphyromonas gingivalis and Fusobacterium nucleatum: Therapeutic Possibilities? ; 2019,.
4. Olsen I, Shah HN, Gharbia SE. Taxonomy and biochemical characteristics of Actinobacillus actinomycetemcomitans and Porphyromonas gingivalis. *Periodontol*. 1999;20:14–52.
5. Holt SL, Kesavalu L, Walker S, Genco CA. Virulence factors of Porphyromonas gingivalis. *Periodontol*. 1999;20:168–238.
6. Potempa J, Banbula A, Travis J. Role of bacterial proteinases in matrix destruction and modulation of host responses. *Periodontol*. 2000;24:153–192.
7. Bostanci N, Belibasakis GN. Porphyromonas gingivalis: an invasive and evasive opportunistic oral pathogen. *FEMS Microbiol Lett*. 2012;333:1–9.
8. Enersen M, Nakano K, Amano A. Porphyromonas gingivalis fimbriae. *J Oral Microbial*. 2013;(5).
9. Lu Q, Darveau RP, Samaranayake LP, Wang C, JL. Differential modulation of human(beta) defensins expression in human gingival epithelia by Porphyromonas gingivalis lipopolysaccharide with tetra- and penta-acylated lipid A structures. *Innate Immun*. 2009;15:325–335.
10. Sroka A, Sztukowska M, Potempa J, Travis J, Genco C. Degradation of host heme proteins by lysine- and arginine-specific cysteine proteinases (gingipains) of Porphyromonas gingivalis. *J Bacteriol*. 2001;183:5609–5616.
11. Slots J, Ting M. Actinobacillus actinomycetemcomitans and Porphromonas gingivalis in human periodontal disease: occurrence and treatment. *Periodontology*. 1999;20:82–121.

Author biography

Samriti Katoch Post Graduate

Cite this article: Katoch S. The red bacteria: Porphyromonas gingivalis. *Int J Periodontol Implantol* 2019;4(4):120-123.