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REVIEW ARTICLE
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Porphyromonas gingivalis: Biology, virulence factors, and clinical importance in periodontal and coronary artery diseases

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Abstract

Porphyromonas gingivalis is a Gram-negative, obligate anaerobic bacterium long recognized as a principal periodontal pathogen and a keystone species in dysbiotic oral biofilms. Its capacity to colonize the subgingival niche, subvert host defenses, and persist in the periodontal pocket is mediated by a diverse arsenal of virulence determinants — notably gingipain proteases, fimbriae, lipopolysaccharide (LPS), outer-membrane vesicles (OMVs), capsule, and iron/heme-acquisition systems. These factors drive tissue destruction, chronic inflammation, and alveolar bone resorption characteristic of periodontitis, and increasingly implicate P. gingivalis in a range of systemic conditions. The microbial count of P. gingivalis is significantly higher in periodontitis patients compared to healthy individuals, with studies showing average counts of around 4.68±3.99×105 cells in periodontitis patients. This article reviews the organism's microbiology, key virulence mechanisms, pathogenic processes in periodontal disease, and the clinical implications for diagnosis, management, and systemic health.

KEYWORDS
Porphyromonas gingivalis, Virulence, Periodontal, Dysbiosis, pathogen

1 | INTRODUCTION AND MICROBIOLOGICAL OVERVIEW

Periodontitis is a common chronic inflammatory disease affecting the supporting structures of the teeth, including the gingiva, periodontal ligament, and alveolar bone. It primarily affects adults, with a global prevalence of about 50%, while severe forms occur in approximately 10% of the population, increasing notably after the third decade of life.[1] The disease results from the accumulation of dental plaque, leading to inflammation and destruction of periodontal tissues and the formation of periodontal pockets. Beyond tooth loss, periodontitis has been associated with several systemic conditions such as diabetes, atherosclerosis, rheumatoid arthritis, Alzheimer's disease, gastrointestinal disorders, and adverse pregnancy outcomes.[1]

Etiology and Pathogenesis The polymicrobial synergy and dysbiosis model explains the development of periodontal disease. In a healthy periodontium, bacterial communities are regulated by the host's immune response. However, when pathogenic species such as Porphyromonas gingivalis colonize, they disrupt this balance, leading to dysbiosis. This shift enhances the virulence of the bacterial community and triggers an ineffective yet destructive host immune response.[2]

Based on their pathogenicity and interrelationships, periodontal pathogens are grouped into microbial complexes. Early colonizers include bacteria from the yellow complex (Streptococcus species),

green complex (Aggregatibacter actinomycetemcomitans, Eikenella corrodens, Capnocytophaga, Campylobacter), and purple complex (Veillonella parvula, Actinomyces odontolyticus). These organisms prepare the environment for secondary colonizers of the orange complex, such as Fusobacterium nucleatum and Prevotella intermedia. The red complex, consisting of Treponema denticola, P. gingivalis and Tannerella forsythia, represents the most pathogenic group and is strongly connected with advanced periodontitis. Acute forms of the disease are also linked to increased colonization by A. actinomycetemcomitans and P. gingivalis.[3]

Transition from Gingivitis to Periodontitis The progression from gingivitis to periodontitis is associated with the emergence of specific oral pathogens, among which Porphyromonas gingivalis plays a key role. Though typically scarce in a healthy oral environment, P. gingivalis becomes active during gingival inflammation by exploiting iron released from the blood. Once active, it contributes to further tissue destruction and disease progression.

This shift leads to qualitative and quantitative changes in the biofilm composition, disrupting the normal balance between the host and its microbiota. The resulting dysbiosis alters the host immune response, initiating inflammation and tissue breakdown. The inflammatory process is characterized by the release of enzymes and proinflammatory mediators such as matrix metalloproteinases (MMPs), IL-6, tumor necrosis factor (TNF)-α, interleukin (IL)-1β, and C-reactive protein (CRP). Increased levels of these mediators accelerate periodontal tissue destruction. Elevated cytokine levels in periodontal pockets have also been linked to systemic effects, including an increased risk of atherosclerotic cardiovascular disease.[4]

P. gingivalis is a non-motile, asaccharolytic, black-pigmented, Gram-negative anaerobe frequently isolated from the subgingival plaque of patients with chronic periodontitis. It can exist in multiple strain types with variable virulence repertoires, and adapts its gene expression in response to environmental cues within the periodontal niche. Because of its disproportionate ability to remodel the local microbiome and to manipulate host immune responses, it has been termed a “keystone pathogen” — a species whose presence alters community behavior and promotes dysbiosis even at low abundance.[5]

The aim of this article is to highlight the key virulence factors of Porphyromonas gingivalis and their role in the pathogenesis of periodontal and cardiovascular diseases.

2 | MAJOR VIRULENCE FACTORS AND THEIR ROLES

1. Gingipains (cysteine proteases) Gingipains — principally Rgp (arginine-specific) and Kgp (lysine-specific) proteases — are among the most important virulence determinants. They degrade host structural proteins, extracellular matrix components, and immunoregulatory molecules; process bacterial surface proteins (affecting adhesion and biofilm formation); and inactivate or dysregulate complement components and cytokines, helping the bacterium evade innate immunity and promote tissue destruction. Gingipains also liberate heme from host hemoproteins, contributing to heme acquisition.[6]

2. Fimbriae (FimA and Mfa1) Fimbriae are thin, hair-like structures found on most strains of P. gingivalis. They extend beyond the bacterial outer membrane and play key roles in biofilm formation, adhesion to host tissues, and invasion into host cells. P. gingivalis possesses two types of fimbriae—long and short. The long fimbriae are made up of FimA protein subunits, while the short ones are composed of Mfa1 subunits.[7]

These fimbriae attach to various host molecules such as proline-rich proteins, glycoproteins, fibrinogen, fibronectin, and lactoferrin, facilitating bacterial colonization and interaction with other oral bacteria to form biofilms. The long fimbriae bind to human GAPDH, promoting bacterial entry into cells and triggering immune responses. They can also adhere to hydroxyapatite and oral epithelial cells, activating Toll-like receptor 2 (TLR2) and inducing the production of inflammatory mediators such as IL-8, TNF-α, and NF-κB, which contribute to bone resorption.

In addition, long fimbriae help P. gingivalis evade host defenses by interfering with the complement system. The short fimbriae, on the other hand, interact with Streptococcus gordonii proteins (SspA and SspB) and promote differentiation of osteoclast precursors, enhancing bone resorption through the release of cytokines such as IL-1β, TNF-α, and IL-6.

Two major fimbrial systems (FimA and Mfa1) mediate adhesion to host cells, extracellular matrix molecules, and other bacterial species, facilitating colonization and biofilm maturation. Fimbriae also engage pattern-recognition receptors on host cells, modulating inflammatory signaling and cellular responses.[8]

3. Lipopolysaccharide (LPS) and lipid A heterogeneity The LPS of P. gingivalis is structurally atypical and highly heterogeneous; different lipid A structures can act as agonists or antagonists of toll-like receptor 4 (TLR4), allowing P. gingivalis to fine-tune host innate immune activation — either provoking inflammation or suppressing protective responses depending on the microenvironment.[9]

4. Outer membrane vesicles (OMVs) P. gingivalis secretes OMVs loaded with gingipains, adhesins, LPS, and other effectors. OMVs act as delivery vehicles, spreading virulence factors through the biofilm and into host tissues; they can disrupt epithelial barriers, modulate immune cells, and contribute to distant tissue exposure.[6]

5. Capsule and hemagglutinins Capsular polysaccharides confer resistance to phagocytosis and complement, while hemagglutinins and hemolysins facilitate erythrocyte binding and hemoglobin utilization — both critical for acquiring nutritional heme in the iron-limited gingival environment.[10] A study examined the role of the P. gingivalis capsule in modulating host immune responses and enhancing bacterial survival. Using the encapsulated strain W50 and its nonencapsulated mutant PgC, researchers found that the nonencapsulated strain induced

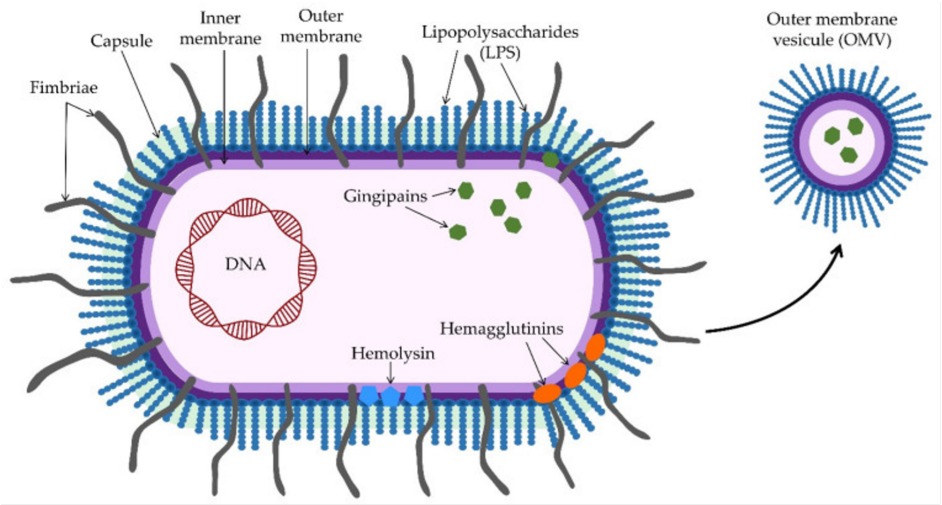

FIGURE 1 Figure 1: Cellular structure of Porphyromonas gingivalis and its most important virulence factors.

a stronger immune response, with significantly higher upregulation of cytokine and chemokine genes and increased expression of maturation markers in macrophages and dendritic cells. Phagocytosis rates for the nonencapsulated strain were 4.5- to 7-fold higher, but its survival within host cells was markedly lower than that of the encapsulated strain. In a mouse abscess model, the encapsulated strain showed greater virulence. These findings highlight that the P. gingivalis capsule helps the bacterium evade immune detection, survive within host cells, and enhance pathogenicity, making it a key virulence factor. [11]

6. Iron/heme acquisition and metabolic adaptations P. gingivalis relies on exogenous heme/iron and expresses multiple receptors and proteases to extract and import these resources. Efficient heme uptake is fundamental to growth, regulation of virulence gene expression, and oxidative stress resistance. [12]

Porphyromonas gingivalis can accumulate a protective, heme-containing pigment on its surface in the form of μ-oxo bisheme. In the body, its main heme sources are hemoproteins found in saliva, gingival crevicular fluid, and red blood cells. To obtain heme, P. gingivalis uses several mechanisms involving hemagglutinins, hemolysins, gingipains (Kgp, RgpA, RgpB), TonB-dependent receptors (HmuR, HusB, IhtA), and hemophore-like proteins (HmuY, HusA). Some proteins responsible for heme transport and storage within the cell, such as PgDps, are less well understood.

Interestingly, P. gingivalis can also exploit heme acquisition systems of other bacteria to meet its needs. It follows a unique mechanism for obtaining heme from hemoglobin, requiring the oxidation of oxyhemoglobin (Fe^{2+}) to methemoglobin (Fe^{3+}) before heme release. This process involves its own gingipains and may also rely on proteases from other bacteria, such as interpain A from Prevotella intermedia, or on pyocyanin from Pseudomonas aeruginosa. Once oxidized, P. gingivalis can break down methemoglobin or extract heme directly through the HmuY hemophore. [13]

3 | PATHOGENESIS IN PERIODONTAL DISEASE

Biofilm formation and community modulation Through adhesins, fimbriae, and OMVs, P. gingivalis establishes and integrates into multi-species biofilms. Its activities remodel the local microbiome toward a dysbiotic state — favoring inflammation-promoting species and suppressing microbes associated with health. Such ecological shifts amplify pathogenic potential beyond the contribution of P. gingivalis alone. [14]

Immune subversion and chronic inflammation By degrading complement factors, chemokines, and immunoglobulins and by manipulating TLR signalling (via atypical LPS and other effectors), P. gingivalis evades clearance and promotes a maladaptive, chronic inflammatory response. Persistent inflammation leads to connective tissue breakdown and recruitment/activation of osteoclasts. Gingipains additionally activate host matrix metalloproteinases and directly degrade extracellular matrix, accelerating attachment loss. [10]

Bone resorption and tissue destruction Inflammation driven by P. gingivalis and its ability to skew osteoclast/osteoblast activity results in alveolar bone loss — the hallmark of periodontitis. P. gingivalis promotes alveolar bone resorption by inducing inflammation and directly stimulating osteoclasts. It does this by invading host cells and triggering signalling pathways, such as those involving Toll-like receptors (TLR2 and TLR4), which lead to the production of inflammatory molecules like RANKL. This disrupts the balance between bone-building osteoblasts and bone-resorbing osteoclasts, resulting in the breakdown of the alveolar bone that supports the teeth. [15]

Proinflammatory cytokines (e.g., IL-1β, TNF-α, RANKL) induced or modulated by bacterial factors are central mediators of osteoclastic bone resorption.

4 | CLINICAL IMPORTANCE

Diagnostic relevance Detection of P. gingivalis in subgingival plaque or by molecular assays can indicate increased risk of progressive periodontal destruction, though presence alone is insufficient for diagnosis — strain differences and host susceptibility modulate disease expression. Quantitative measures and assessment of virulence gene expression can improve risk stratification.[16] Its presence and load in subgingival plaque or saliva indicate active infection and predict disease severity. Molecular tests like PCR are highly reliable for this purpose.

Elevated levels of P. gingivalis or its antibodies act as biomarkers for systemic inflammatory processes associated with neurodegeneration, CVD, and certain cancers.

Implications for periodontal therapy Traditional mechanical debridement (scaling and root planing) reduces biofilm burden, but persistent or recolonizing P. gingivalis populations — particularly strains rich in virulence determinants — may necessitate adjunctive measures: local/systemic antimicrobials, host-modulation therapies, or approaches targeting specific virulence factors (e.g., gingipain inhibitors, vaccines in experimental stages). Understanding virulence profiles may guide personalized treatment strategies.[9]

Associations with systemic disease A growing body of evidence links P. gingivalis to systemic conditions, including atherosclerotic cardiovascular disease, adverse pregnancy outcomes, rheumatoid arthritis (via citrullination and anti-citrullinated protein antibody pathways), and neurodegenerative disorders such as Alzheimer's disease. Mechanisms proposed include systemic dissemination of bacteria or OMVs, chronic systemic inflammation, molecular mimicry, and direct proteolytic or immunomodulatory effects at distant sites. While causality is not fully established for many associations, the connections underscore the broader health significance of controlling periodontal infection.[17]

Several studies have shown that individuals with cardiovascular diseases generally have poorer oral health compared to the healthy population. Research indicates that chronic periodontal disease increases the risk of developing coronary artery disease by about 25%.[18] Inflammation plays a key role in the development of atherosclerosis, and chronic periodontal inflammation is now recognized as a contributing factor in its progression.

Chronic periodontitis alternates between active and stable phases and triggers persistent, low-grade systemic inflammation that contributes to atherosclerosis. Periodontal pathogens have been detected in atherosclerotic plaques, with Porphyromonas gingivalis being the most frequently identified bacterium in such lesions.

Microorganisms from dental plaque and their by-products can enter the bloodstream during dental procedures such as scaling or surgery, allowing them to reach distant organs. This dissemination is linked to conditions like subacute bacterial endocarditis, coronary heart disease, atherosclerosis, and ischemic stroke.[19] Bacteraemia originating from the oral cavity may damage heart valves, leading to bacterial endocarditis. Furthermore, P. gingivalis has been identified as a risk factor for prothrombotic conditions, which may worsen outcomes in patients with atrial fibrillation.[20] Chronic periodontitis, which often includes periods of acute inflammation, can trigger a persistent low-grade systemic inflammatory response that contributes to the development of atherosclerosis and increases the risk of cardiovascular disease.

Porphyromonas gingivalis plays a significant role in this process by damaging endothelial cells, disrupting vascular integrity, and promoting the formation of atherosclerotic plaques. Endothelial dysfunction, characterized by vascular leakage, can be worsened by hypertension, dyslipidemia, and inflammatory mediators, leading to vessel blockage and higher circulating levels of low-density lipoprotein (LDL)[21] P. gingivalis induces oxidative stress in endothelial cells, a key factor in atherosclerosis development.[22] Its proteolytic enzymes, gingipains—lysine-specific (Kgp) and arginine-specific (Rgp)—promote oxidative stress by consuming antioxidants and causing lipid peroxidation.[23] The bacterium also increases the production of reactive oxygen species (ROS) via the TLR–NF–κB signaling pathway.[24]

The resulting infection activates immune and inflammatory responses, including the release of interleukins (IL-1β, IL-6, IL-8) and tumor necrosis factor-alpha (TNF-α)[????]. P. gingivalis also upregulates CD36, a scavenger receptor that facilitates the oxidation and accumulation of LDL within macrophages, forming oxidized LDL (oxLDL). Similarly, the bacterium can oxidize high-density lipoprotein (HDL), impairing cholesterol removal from macrophages and further promoting atherosclerosis. Additionally, P. gingivalis disrupts the balance between proatherogenic (Th17) and atheroprotective (Treg) cytokines and can transform macrophages into lipid-laden foam cells, a hallmark of early atherosclerotic lesions. These processes collectively drive inflammatory changes in blood vessels.[25]

Animal studies have shown that infection with P. gingivalis accelerates coronary and aortic atherosclerosis and increases lipid deposition. In mice, exposure to P. gingivalis heat-shock protein GroEL increased the expression of adhesion molecules (ICAM-1, VCAM-1), toll-like receptor-4, and oxidized LDL receptors.[26] In vitro studies have also demonstrated that P. gingivalis upregulates IL-1β and TNF-α in brain endothelial cells, leading to cell death through the ROS/NF-κB pathway.[27]

5 | PREVENTION AND FUTURE DIRECTIONS

Effective periodontal disease control remains grounded in mechanical plaque control and risk factor management (smoking cessation, glycemic control). Future adjuncts may include targeted inhibitors of gingipains, vaccines or immunotherapies tailored to major adhesins or proteases, therapeutics that restore microbial homeostasis, and diagnostics that identify high-virulence strains. Continued research into strain-specific pathogenicity, OMV biology, and host–microbe interactions will be central to translating mechanistic insights into clinical interventions.[24]

6 | CONCLUSION

Porphyromonas gingivalis is a multifaceted periodontal pathogen whose virulence derives from proteolytic enzymes, adhesins, immunomodulatory LPS, OMVs, and sophisticated nutrient-acquisition strategies. These factors enable colonization, immune evasion, persistent inflammation, and alveolar bone loss. Beyond local periodontal destruction, mounting evidence implicates P. gingivalis in systemic diseases, elevating the importance of effective detection and control in clinical practice. Addressing the bacterium's virulence mechanisms offers promising avenues for adjunctive therapies and improved patient outcomes.

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