Review Article

Antibacterial effect of green tea against *Porphyromonas gingivalis*: a literature review

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Abstract: Green tea is regarded as a healthy beverage due to the biological action of polyphenols, specifically catechins. Researchers investigated the inhibitory effects of green tea (*Camellia sinensis*) plant on both Gram- positive and Gram-negative bacteria. Periodontitis is mainly initiated when dysbiosis of the subgingival microbiome takes place. *Porphyromonas gingivalis* is one of the key pathogens involved in the initiation and progression of periodontitis when its levels in subgingival biofilm overwhelm the host's immune system. It is the main pathogen that is significantly linked to severe periodontal disease. In this study, details about P. gingivalis including information about its structure, virulence factors were provided. Next, Important points related to green tea, including its active ingredients and antibacterial activity, have been clarified. Finally, the objective of this study was to demonstrate how green tea has antibacterial effects on P. gingivalis.

Keywords: Green tea, *P. gingivalis*, Periodontitis.

Introduction:

Porphyromonas gingivalis is a rod-shaped anaerobic pathogenic bacterium that belongs to the group Bacteroidetes [(1)]. The bacteria are members of more than 500 species living in the mouth, and can multiply in many cells, causing large-scale destruction of peripheral lesions [(2)]. HME aggregation in the cell surface of P. gingivalis results in black pigmentation on the blood agar surface [(3)]. It is able to survive in deep pockets of the period by fermenting amino acids, which are very rare places with sugar metabolism [(4)]. Some strains have capsules that inhibit host cell phagocytosis and help bacteria survive in hostile environments [(5)].

As a member of Gram-negative bacteria, It has an outer and cytoplasmic membrane, the outer membrane contains porins, Omp A-like protein, and other important proteins [(7)]. Several strains of *P. gingivalis* develop a capsule that precludes its phagocytosis by the host cells [(5)]. *P. gingivalis* adherence to the epithelial and bacterial cells surfaces is crucial for colonization and survival inside infection sites. Its adhesive capacity is mediated by fimbriae and haemagglutinins, as well as other adhesins found on its surface. However, subgingival colonization requires the production of particular molecules important for bacterial persistence, such as those involved in oxidative stress antagonism and nutrient intake [(8, 9)].

Periodontal disease

Periodontal disease are group of infectious diseases that cause gingival inflammation, periodontal ligament breakdown, alveolar bone resorption, and tooth exfoliation. The major cause of development and progression of these infections is the dental biofilm. Presence of a dysbiotic dental biofilm on the teeth's surfaces leads to series of inflammatory events in the periodontium, ranging from mild reversible conditions i.e., gingivitis, to periodontitis associated with tissue destruction and tooth exfoliation.

Periodontal disease are one of the most prevalent illnesses, according to the World Health Organization, with tooth loss affecting 5% to 15% of the worldwide population as a result of aggressive types of periodontitis [10].

Role of *P. gingivalis* in periodontal disease

Experimental animal models showed that periodontitis is a polymicrobial infection caused by a synergistic microbial community instead of individual pathogens. The role of *P. gingivalis* in such a microbial community is to shift the balance from homeostasis to dysbiosis [11]. Therefore, in periodontitis, there is an alteration in host-microbial equilibrium, where even commensal bacteria can trigger damaging inflammation. After six weeks of inoculating *P. gingivalis* in to a specific-pathogen-free periodontitis-mouse model, there was substantial alveolar bone loss [12].

In periodontitis, *P. gingivalis* adopts a variety of strategies to collaborate with other pathogens and escape immune clearance. It has the ability to bypass and manipulate host immunological mechanisms in order to aid colonization and survival of its own and other pathogenic microorganisms [13]. According to the keystone pathogen theory, *P. gingivalis*, even at low abundance, can induce periodontitis by causing dysbiosis in the commensal bacterial community, which leads to periodontitis [14]. Furthermore, studies have shown that *P. gingivalis* has negative effects on tooth supporting systems [15, 16].

Virulence factors

Lipopolysaccharide (LPS)

LPS is a pathogen-associated molecular pattern (PAMP) which has been recently defined as microbe-associated molecular pattern (MAMP) [17]. It is an integral part of the cell wall of Gram-negative bacteria i.e., outer cell membrane [18]. The basic units of LPS are lipid A and core, as well as long polysaccharide O-antigen repeats [19]. LPS was termed as endotoxin due to the toxic effect and the tendency to promote destructive inflammatory changes. The fatty acid acyl chain producing lipid A is responsible for the structural variations between LPS from different bacterial species. The varied manner in which host cells identify bacterial species might be explained by variations in LPS structure [13]. *P. gingivalis* endotoxins have a significant role in the initiation and progression of periodontitis. *P. gingivalis* LPS lipid A promote the immunological reactions in gingival tissues, creating a favorable condition for bacteria to thrive and eventually leading to periodontal disease [20]. Furthermore, when *P. gingivalis* LPS is co-cultured with *F. nucleatum*, its antigenicity and cytokine expression were increased, and the lipid A structure is altered due to upregulation of lpxA and lpxD genes involved in LPS synthesis [21].

Fimbriae

The outer surface membrane of *P. gingivalis* contain specific protrusions, called fimbriae. The role of *P. gingivalis* fimbriae in the pathogenesis of periodontitis was the subject of many studies [13]. Two varieties of *P. gingivalis* fimbriae were reported; long fimbriae (major fimbriae) with FimA subunit proteins and short fimbriae (minor fimbriae) containing Mfa1 subunit proteins. Despite their varying amino acid content and antigenic characteristics, FimA and Mfa1 are both important for tissues immune reactions and participated in the development of periodontal diseases [22]. Fibrinogen, fibronectin, proline-rich proteins and glycoproteins, statherins, and lactoferrin are among the host molecules and oral substrates used by *P. gingivalis* fimbriae to adhere to infected sites. This connection between extracellular and intracellular environments is crucial for cell anchorage, migration, survival and intracellular signal transduction cascades. Thus, the presence of *P. gingivalis* can delay the healing of the diseased periodontal tissue [23]. *P. gingivalis* coaggregation with *Actinomyces viscosus*, *Treponema denticola* and *Streptococcus gordonii* has been described to be mediated by fimbriae. Utilization of the complement system by the Long fimbriae for evasion of the immune system was also reported [13].

Gingipains

They are cysteine proteinase that known as "trypsin-like" enzymes [24]. They are categorized into two kinds: arginine specific proteinase (RgpA and RgpB) and lysine specific proteinase (Kgp) [25]. they are involved in periodontitis pathogenesis and are responsible for 85% of *P. gingivalis'* extracellular proteolytic actions [26]. Gingipain inhibitors have been investigated by several studies due to their potential therapeutic effectiveness in reducing the pathogenicity of *P. gingivalis* [27]. They increase nutrition acquisition through hemagglutination, hemolysis, and iron absorption from heme inside infected periodontium [28]. Other host systems that gingipain might target include the blood coagulation pathway and the complement system [29, 30]. The anaerobic microorganism's ability to colonize the oral mucosa and be a pathogenic bacterial species in the oral cavity is aided by these gingipain biological features. Furthermore, gingipains participate in the fimbria-mediated adhesion process by modifying and maturing pro-fimbrilin into fimbriae [31].

Capsule

Capsule is an exterior outer component that represents the bacteria's outline fence. water and polysaccharides are most often constituents of the capsules which prevent dehydration. Bacteria that have been encapsulated usually withstand the host tissues clearance processes, therefore the capsule acts as a protective bacterial component [13]. Chemical components of *P. gingivalis* capsules vary, allowing various strains to be classified into unique K-serotypes, whereas some strains are not encapsulated Which make them more susceptible to be eliminated via macrophages and dendritic cells. On the other hand, encapsulated strains are shielded from phagocytosis by the capsule and their survival rate are much higher [5, 32].

T cell immunity stimulation

T cells participate in a range of inflammatory disorders and play a key role in host immune responses. T cells have been reported to have a lesser proliferative capacity in periodontitis patients, indicating a diminished T cell activity[33]. Various periodontal infections can inhibit T cell activation [34] [35]. To evade the host's adaptive immunological response, *P. gingivalis* may cause reduction of IL-2 production and T cell proliferation due to the effect of gingipain Rpgs [36]. In a periodontitis animal model, the alveolar bone loss induced by *P. gingivalis* was shown to be less in IFN- and IL-6-deficient mice, indicating a deleterious effect of Th1 and Th2 cells on the periodontal tissues [37].

Hemagglutinins

The virulence of different pathogenic bacteria may be stimulated by the effect of hemagglutinins [38, 39]. These hemagglutinins on cell surfaces have been identified to function with fimbriae as fimbrial adhesins or non-filamentous surface molecules as non-fimbrial adhesins to allow bacteria to adhere to host cells [40]. Hemin absorption from erythrocytes, which is required for *P. gingivalis* growth, is also linked to hemagglutinin pathogenicity [41].

Outer membrane proteins

The cell wall of Gram-negative bacteria has a sophisticated multilayered structure. The term cell envelope is sometimes used to describe the onion-skin nature of this cell wall. The inner cytoplasmic membrane, which is made up of a thin peptidoglycan, is connected to the asymmetrical outer membrane. The structural integrity of the cell envelope is maintained by complex LPS, lipoproteins, and peripheral and transport proteins of the outer membrane via linking it to the peptidoglycans. About 20 main proteins make up the outer membrane of *P. gingivalis* most of them were heat modifiable [42].

Outer membrane vesicles

P. gingivalis interactions with its surroundings at a distance from the cell surface may be mediated by outer membrane vesicles. These vesicles include proteases that help in collagen breakdown, facilitate bacterial adhesion to cells, and enhance attachment amongst nonaggregating bacteria like *Capnocytophaga* ochracea and *Eubacterium saburreum*, implying that they may be involved in periodontal disease development [43].

Green tea

Green tea (GT) has been used as a medicinal and a healthy beverage since earlier. Chinese emperor Shen Nung discovered tea quite accidentally in 2737 B.C. [44]. He had a habit of boiling his drinking water before drinking it, and one day, some leaves from a nearby tree fell into the jar resulting in an exceptional tasting and perfumed drink. Recently, more than 3 billion cups of tea are estimated to be consumed every day being the second most common drink after water [45]. It is made by macerating and heat drying the leaves of the *Camellia sinensis* plant [46]. Because of the biological activities of its ingredients, the role of green tea on human health is receiving further focus. It has a high concentration of polyphenols, mostly catechins derivatives, which are believed to protect against cancer and cardiovascular disease [47]. Due to the presence of high content of catechins, specifically epigallocatechin-3-gallate (EGCG), GT has been shown to have a wide spectrum of antimicrobial action [48]. Moreover, Catechins have been found to interact with the cell wall and membrane of bacterial cells [49], causing irreversible damage to the cells by the formation of hydrogen peroxide [50].

Chemical structure of green tea

The chemical components of GT leaves have been extensively studied. Polyphenols are the primary components of tea leaves [51]. Diffusion and solubility of the constituents are major factors to enhance the extraction processes to obtain crude extracts of the plants' leaves [52]. Longer extraction periods enhance cell wall permeability in green tea leaves, whereas higher temperatures promote solubility constants. This allows more catechins from tea and other chemicals to be extracted. Flavonols and related flavonoids are integral components of tea plants (*Camellia sinensis L.O. Kuntze*) [53]. Multiple flavonol derivatives e.g. myricetin, quercetin, and kaempferol are glycosylated to create stable flavonol glycosides (FGs) via interactions with sugar molecules (e.g., glucose, galactose, and rutinose) [54]. Catechins (flavan-2-ols) like epicatechin (EC), epicatechin-3-gallate (ECG), epigallocatechin (EGC), and EGCG are the most frequent flavonoids identified in GT [55].

Antimicrobial effects of green tea

GT has been shown to exhibit antibacterial activities against Gram-positive and Gram-negative bacteria, fungi, and viruses [56, 57]. GT's antibacterial efficacy is attributed to polyphenolic catechins. The antibacterial activity of EGCG, the main constituent of GT catechins, alone and in conjunction with various antibiotics have been thoroughly investigated against a variety of bacteria, including multidrug-resistant strains such as methicillin-resistant *Staphylococcus aureus* and *Stenotrophomonas maltophili* [57]. When GT was used systemically as a traditional antibacterial agent, it was reported that it had a poor antibacterial activity. When administered as a topical treatment for superficial bacterial infections, GT catechins can be effective [46, 58]. Incorporation of theses extracts with vacuum cleaner filters and face masks was also applied in order to reduce airborne pollution. It is useful as an antibiotic combination ointment for the prevention of impetigo [46, 59]. The MIC values of GT aqueous extracts demonstrated significant antibacterial action against the primary dental biofilm colonizers [60].

Antibacterial effects of green tea against *Porphyromonas gingivalis*

Various studies attempted to establish the minimum inhibitory concentration (MIC) of GT extracts against *P. gingivalis*. Fournier-Larente et al., reported MIC value of 6.25 mg/ml [61]. While Araghizadeh et al. [62], reported that the MIC value of GT aqueous extracts against clinically isolated *P. gingivalis* was 12.5 mg/ml. In another study, it was reported that 0.5% of GT aqueous extracts prepared by maceration showed inhibitory effects against *P. gingivalis* [63]. The MIC values of GT aqueous extract across the latter experiments, however, are inconsistent. This may be explained by the various phytochemical compositions of the studied extracts, since different extraction techniques for herbs may have produced varying amounts of active ingredients.

The presence of caffeine in GT aqueous extract, which has been shown to have antibacterial properties against Gram-negative bacteria, may be responsible for the antibacterial activity of the compound [64]. In earlier investigations, GT catechins have been the most frequently identified phytochemical component with antibacterial action against *P. gingivalis*. [65-67].

Conclusion

Based on previously reviewed literature, GT contains beneficial antimicrobial properties. It is also clear from several *in vitro* studies that GT extracts have an inhibitory action against *P. gingivalis*. Isolation of the bioactive ingredients of GT aqueous extracts for further verification of their antibacterial properties and studying the antibacterial effects of extracts on the other bacteria of the red complex, i.e., *T. denticola* and *Tannerella forsythia* may enrich future research in this area.

Conflict of interest

The authors have no conflicts of interest to declare.

Author contributions

All authors participated in study conception, literature search, writing the draft, and approval of the final version of manuscript.

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Informed consent

Informed consent was obtained from all individuals or their guardians included in this study.

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التأثير المضاد للبكتريا للشاي الأخضر ضد البورفيروموناس جينجيفاليس: مراجعة الأدبيات

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المستخلص

يعتبر الشاي الأخضر مشروبا صحيا بسبب العمل البيولوجي لبوليفينول الشاي الأخضر ، وتحديدا الكاتيكين. حقق الباحثون في الآثار المثبطة لنبات الشاي الأخضر (كاميليا سينينسيس) على كل من البكتيريا إيجابية الجرام وسالبة الجرام. عندما تتجاوز مستويات *البورفيروموناس جينجيفاليس* في الأغشية الحيوية تحت اللثة قدرة المضيف على الدفاع ضدها ، فإن العامل الممرض الرئيسي يرتبط ارتباطا كبيرا بأمراض اللثة الشديدة. كان الهدف من هذه الدراسة الأدبية هو إظهار كيف أن الشاي الأخضر له تأثير مضاد للبكتيريا على *البورفيروموناس جينجيفاليس*. في هذه الدراسة ، قدمنا أولا تفاصيل حول البورفيروموناس جينجيفاليس ، بما في ذلك معلومات حول هيكلها. عوامل الضراوة التي تعزز التهاب اللثة وتقدمه. بعد ذلك تناولنا بعض النقاط المهمة المتعلقة بالشاي ، بما في ذلك مكوناته النشطة وأنشطته المضادة للبكتيريا.