ISSN NO. 2320-5407

Journal homepage:http://www.journalijar.com
Journal DOI:10.21474/IJAR01

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH

RESEARCH ARTICLE

BDELLOVIBRIO BACTERIOVORUS.

Nurul husniyah binti che soh.
Caroline Jacob

Manuscript Info

Manuscript History:

Received:15 March 2016
Final Accepted: 12 May 2016
Published Online: May 2016

Key words:
Bdellovibrio bacteriovorus, biofilms, periodontopathogens, periodontitis

Corresponding Author

Nurul husniyah binti che soh.
Caroline Jacob

Abstract

Bdellovibrio bacteriovorus is a gram-negative bacteria that can specifically act upon other gram-negative bacteria. This bacteria is capable of attacking and removing bacteria that reside within biofilms, most commonly being dental plaque. Its unique morphology and life cycle allows it to penetrate into other gram-negative bacteria and multiply within the host bacteria's periplasm. A new life cycle begins once the bacteria bursts through cell envelope. The potential to rupture the cell walls of bacteria that reside within the dental biofilm can therefore open new horizons that can prevent dental plaque associated oral diseases

Introduction:-

Periodontal disease is amultifactorial infection whichis elicited by Gram-negative periodontopathogens. This is due to the destruction of periodontal structures which include tissues supporting the tooth, alveolar bone and periodontal ligament (1).The interactions between bacterial species with the host tissues cause damage to the periodontal structures, resulting in periodontal disease. Gram-negative bacteria isolated from various periodontal periodontal diseases includeAggregatibacteractinomycetemcomitans, Eikenellacorrodens, Fusobacteriumnucleatum, Prevotellaintermedia, Porphyromonasgingivalis and Tannerella forsythia (2).The difficulty in eliminating these bacteria within dental plaque has profound implications. Periodontal disease is now one of the most complex and well-known chronic infectious diseases that occur in humans (3).

Conventional therapies that include a combination of mechanical and chemical plaque control are constantly evolving to arrest periodontal disease(1).The use of antimicrobial drugs face the uncertainty of losing its effectiveness in view of pathogenic multidrug-resistant bacteria(4) as well as difficulty in penetrating thedental plaque biofilm. Bacteria within biofilms are 1000 times more resistant (5, 6) towards antimicrobial agents than their planktonic counterparts and therefore, an alternative approach is the need of the hour. Predatory bacteria that are symbiotic with the human body and can combat pathogenic periodontal bacteria may be one such solution.

Predatory prokaryotes which belong to the genus Bdellovibriobacteriovorus are Gram-negative bacteria that are well-known for their ability of feeding on other Gram-negative bacteria (7, 8).Bdellovibrio are used widely as they have the potential to prey on a wide range of human pathogens that grow both planktonically and in a biofilm (9-11). In a study by Dashiff et al in 2011, (12) the host specificity of B. bacteriovorus strain 109J was examined along with its ability to prey on oral pathogens associated with periodontitis such as A.ctinomycetemcomitans, E.corrodens, F.nucleatum, P.intermedia, P.gingivalis and T.forsythia. B. bacteriovorus 109J was able to remove metabolically inactive biofilms, biofilms of E.corrodens as well as biofilms composed of A.actinomycetemcomitans that were developed on hydroxyapatite surfaces and in the presence of saliva.(13)

The bdellovibrio bacteriovorus:-

Bdellovibrio-and-like organisms (BALOs) are small, highly motile Gram-negative obligate bacterial predators found in fresh and brackish water, sewage, water reservoirs, seawater (14-16), soil (7) and biofilms (15-17). BALOs form the two different and internally diverse families known as Bacteriovoracaceae and Bdellovibrionaceaewhich are classified under the order of Bdellovibrionales and cluster in the delta-proteobacteria class (18). The predatory bacteria form a deep branch in the α-proteobacteria. Many of the host-independent predatory bacteria or BALOs are pleomorphic, vibrio-to spiral-shaped cells that usuallymeasure across 0.3-0.4µm and 1-10µm width and length-wise respectively. Typically, host-independent (H-I) Bdellovibrios have been found to have a cytochrome a andc component that are sensitive towards both the oxytetracycline and vibriostatic pteridine. These components make most Bdellovibriosresistant or susceptible to a particular antibiotic (19). B. bacteriovorus are smaller than their prey, in contrast to protists (20). They are motile and uniflagellatedwith appendages situated on the non-flagellated pole that help the Bdellovibriobacteriovorus to bond to their prey tightly. These allow the enzymes that are secreted to burrow via their surface in between the outer membrane and the wall of peptidoglycan (8, 21-23).

By using energy metabolism intermediates, 11 amino acids that are required for synthesis of protein can be produced by the B. bacteriovorus HD100, whilethe degradation pathway for 10 amino acids is absent. Yet, all the enzymes for the production of the full range of activated tRNAs are present. These are associated with B.bacteriovorus'ability to biosynthesize protein only while it has access to the prey's amino acids (24).

The Bdellovibrio spp.are different from all other bacterial parasites as they have a biphasic growth cycle which includes a free-swimming attack phase and an intraperiplasmic growth phase (25).B. bacteriovorus has proved to be very effective in combating biofilms (26-28) due to its ability to penetrate deep within the biofilms of the prey and terminate them effectively. These characteristics make them different from other biological tools such asbacteriophages and protists (28). Bacteriophages are a group of viruses that are bactericidal and are capable of infecting archae or single-celled prokaryotic organisms while protists, though associated with motility multiply bybinary fission (29).

Mechanism of predation:-

The predatory life cycle of Bdellovibriocomprises of eight stages. In stage I or the attack phase, a single sheathed polar flagellum allows the predatory bacteria to swim at high speed (30). At four independent loci, there are six clusters of motility and flagella synthesis genes which are in combination with six copies of flagellin genes. The B. bacteriovorusremains reversibly attached to the prey cell for a short time once it collides with a prey cell (31). In stage II, it becomes irreversibly anchored through the pole opposite the flagellum. Once it enters the prey periplasm,B. bacteriovorus, sheds its flagellum. This occurs in stage III and proceeds with cellular events such as DNA replication and cellular biopolymers synthesis in stage IV. On entering the periplasm, there is a change in the morphology of the prey which forms a bdelloplast that makes up the fifth stage. In stage VI,the filamentous cell form septa and flagella to produce few offspring attack-phase cells. The mechanism is different though the gene products for chromosome partitioning and septation are homologous to those encoded by known genes (mreB, mbl, ftsZ, and smc). This is because a single long filamentous cell divides into many identically sized progeny cells (32). These progeny develop into flagellated cells that are available for further attack in the exhausted prey protoplast which occurs in stage VII. In addition to the development of flagella, B. bacteriovorusforms hydrolytic enzymes (33), which dissolve the remaining peptidoglycan layer ofthe prey's cell outer membrane. This constitutes the eighth stage, where the release of enzyme is responsible for the release of progeny.

S. aureusis capable of contributing to the production of the biofilm, which in turn contains extracellular polymeric substances or EPS that houses bacterial cells(34).The EPS matrix is produced by extracellular DNA, polysaccharides and proteins which adhere strongly to surfaces, causing difficulties in removing biofilms. In order to overcome the biofilm, hydrolytic enzymes such as proteases and DNases are used to eliminate the EPS (35-37).The S. aureusbiofilm formation has been shown to be inhibited by Bdellovibriobacteriovorussupernatant as the latterproduces several hydrolyticenzymes, particularly proteinase K (38). These enzymes are able to hydrolyze the macromolecules of the prey, thereby allowingpredatory bacteria to work effectively against the biofilm (30). In addition, various Yersinia strains are also affected by the predation. This is proven when the optical densities of the strains were significantly mitigated by predation of Bdellovibrio bacteriovorus (39).

Role in treating periodontal disease:-

BALOs are able to predate upon the Gram-negative human pathogens by using the secreted hydrolytic enzymes like proteases/peptidases (26) and other hydrolases. Host-independent mutants are capable of growing within the periplasmic space which is smaller and more turbid in comparison with those that are formed by wild-type B. bacteriovorus (40). Host independent (HI) mutants of B. bacteriovoruscultures have shown extensive action of extracellular protease (41). Various Gram-negative pathogenic bacterial strainssuch as Yersinia, Serratia, Salmonellaand Acetobacterare capable of beingpredated and infected by the wild-type B. bacteriovorusHD100 (39). Evidence has shown that A. actinomycetemcomitansis susceptible to B. bacteriovorusHD100 predation in an oral cavity-like environment (13). The incubation of both microorganisms for 8-12 h showed an approximate 2.43log10 decrease in pathogen viability by using a 1.14:1 predator: prey ratio. In addition, the efficiency of the B. bacteriovorus HD100 was not affected by different A. actinomycetemcomitans strains. Experiments aimed at enhancing the biofilm removal aptitude of B.bacteriovorus with the aid of extracellular-polymeric-substance-degrading enzymes, demonstrated that proteinase-K inhibits predation. Furthermore, A. actinomycetemcomitans biofilms treated with DspB, a poly-N-acetylglucosamine (PGA) -hydrolyzing enzyme, increased biofilm removal. The predation of B. bacteriovorus109J towards A. Actinomycetemcomitanswas very similar to that of B. bacteriovorus HD100 towards the same bacteria (13). Predation kinetics was conducted, combiningA. actinomycetemcomitans with various Bdellovibrio concentrations. The results demonstrated a higherconcentration of predatory cells with a great decrease in pathogen viability (42).

Advantages:-

The Bdellovibrio sp. hasbecome an attractive potential bio-control agent due to their intrinsic ability to parasitize and lyse prey cells. Another advantage of using B. bacteriovorus as a predatory bacteria, is that they effectively hydrolyze its prey's macromolecules through a cache of 150 proteases/peptidases (26) together with other hydrolases. Furthermore, they may be used as therapeutic agents as they are able to maintain their ability to attack multidrug-resistant bacteria regardless of their resistance towards antimicrobial drugs. BALOs are generally regarded as safe, unable to infect eukaryotic cells (43) and do not induce a strong immunological response (44).

Disadvantages:-

Even though Bdellovibrios have shown many benefits against microorganisms, they are not capable of predating Gram-positive strains (7, 9, 26) that may be pathogenic (10, 12). Theyare also unable to prey on Staphylococcus aureus, whichis one of the most frequent nosocomial infection-associated multidrug resistant pathogens. In addition, B. bacteriovorusisunable to prey on P. gingivalis, P. intermedia, T. forsythia and F. nucleatum ATCC 10953when used as host cultured both planktonically or as a biofilm (45). In the presence of high concentrations of glucose or glycerol and in low pH, the activity of BALOs is said to reduce significantly (19). Furthermore, the presence of other bacteria and the physiological status of potential prey is said to affect the activity of the predatory bacteria. (19).

Conclusion:-

The evidence points to predatory Bdellovibriobacteriovorusas being effectively capable of attacking and reducing the formation of biofilm which harbor drug-resistant bacteria. Their unique complement of proteases and other hydrolyses provides a valuable reservoir of enzyme-based antimicrobial substances.They appear to be potentially safe and may providea large spectrum of advantages to manage chronic infectious diseases. Bdellovibriomay soon be considered as living antibiotics in future pharmacological applications due to absence of evidence concerning invasion of mammalian cells byBdellovibrio.

References:-

1. Essche, M. V., Quirynen, M., Sliepen, I., Loozen, G., Boon, N., Eldere, J. V., &Teughels, W. (2010). Killing of anaerobic pathogens by predatory bacteria. Molecular Oral Microbiology,26(1), 52-61.
2. Dzink, J.L., Tanner, A.C., Haffajee, A.D. and Socransky, S.S. (1985) Gram negative species associated with active destructive periodontal lesions. J ClinPeriodontol 12: 648–659.
3. Albandar, J.M. and Kingman, A. (1999) Gingival recession, gingival bleeding, and dental calculus in adults 30 years of age and older in the United States, 1988– 1994. J Periodontol 70: 30–43.
4. Van Winkelhoff, A.J., Herrera, D., Oteo, A. and Sanz, M. (2005) Antimicrobial profiles of periodontal pathogens isolated from periodontitis patients in The Netherlands and Spain. J ClinPeriodontol 32: 893–898.

5. Costerton, J. and Keller, D. (2007) Oral periopathogens and systemic effects. Gen Dent 55: 210–215.
6. Costerton, J.W., Stewart, P.S. and Greenberg, E.P. (1999) Bacterial biofilms: a common cause of persistent infections. Science 284: 1318–1322
7. Stolp, H. and Starr, M.P. (1963) Bdellovibriobacteriovorus gen. et sp. n., a predatory, ectoparasitic, and bacteriolytic microorganism. Antonie Van Leeuwenhoek 29: 217–248.
8. Sockett, R.E. (2009) Predatory lifestyle of Bdellovibriobacteriovorus. Annu Rev Microbiol 63: 523–539
9. Kadouri, D. & O'Toole, G. A. Susceptibility of biofilms to Bdellovibrio bacteriovorus attack. Appl Environ Microbiol 71, 4044–4051 (2005).
10. Dashiff, A., Junka, R. A., Libera, M. &Kadouri, D. E. Predation of human pathogens by the predatory bacteria Micavibrioaeruginosavorus and Bdellovibrio bacteriovorus. J ApplMicrobiol 110, 431–444 (2011).
11. Kadouri D, Venzon NC, O'Toole GA (2007) Vulnerability of pathogenic biofilms to Micavibrioaeruginosavorus. Appl Environ Microbiol 73: 605–614.
12. Dashiff, A., and D. E. Kadouri. "Predation of oral pathogens by Bdellovibrio bacteriovorus 109J." Molecular oral microbiology 26.1 (2011): 19-34.
13. Van Essche, M., Quirynen, M., Sliepen, I., Van Eldere, J. and Teughels, W. (2009a) Bdellovibrio bacteriovorus Attacks Aggregatibacter actinomycetemcomitans. J Dent Res 88: 182–186.
14. Dashiff, A., &Kadouri, D. (2010). Predation of oral pathogens by Bdellovibrio bacteriovorus 109J. Molecular Oral Microbiology,26(1), 19-34.
15. Kelley, J. I., B. F. Turng, H. N. Williams, and M. L. Baer. 1997. Effects of temperature, salinity, and substrate on the colonization of surfaces in situ by aquatic bdellovibrios. Appl. Environ. Microbiol. 63:84–90.
16. Schoeffield, A. J., and H. N. Williams. 1990. Efficiencies of recovery of bdellovibrios from brackish-water environments by using various bacterial species as prey. Appl. Environ. Microbiol. 56:230–236.
17. Williams, H. N., H. N. Schoeffied, D. Guether, J. Kelley, D. Shah, and W. A. Falkler. 1995. Recovery of bdellovibrios from submerged surfaces and other aquatic habitats. Microb. Ecol. 29:39–48.
18. Davidov, Y., and Jurkevitch, E. (2004) Diversity and evolution of Bdellovibrio-and-like organisms (BALOs), reclassification of Bacteriovoraxstarrii as Peredibacterstarrii general nov., comb. nov.& description of the Bacteriovorax–Peredibacter clade as Bacteriovoracaceae fam. nov.Int J SystEvolMicrobiol 54: 1439–1452.
19. Dwidar M, Monnappa AK, Mitchell RJ. The dual probiotic and antibiotic nature of Bdellovibrio bacteriovorus. BMB Rep. 2012;45:71–8. [PubMed]
20. Dwidar M, Monnappa AK, Mitchell RJ (2012) The dual probiotic and antibiotic nature of Bdellovibrio bacteriovorus. BMB Rep 45(2): 71–78.
21. Ruby, E. G. 1989. Cell-envelope modifications accompanying intracellular growth of Bdellovibriobacteriovorus, p. 17–34. In J. W. Moulder (ed.), Intracellular parasitism. CRC Press Inc., Boca Raton, FL.
22. Starr, M. P., and R. J. Seidler. 1971. The bdellovibrios. Annu. Rev. Microbiol. 25:649–678.
23. Thomashow, M. F., and S. C. Rittenberg. 1979. The intraperiplasmic growth cycle—the life style of the bdellovibrios. In J. H. Parish (ed.), Developmental biology of prokaryotes. University of California Press, Berkeley, CA.
24. Seidler, Ramon J., and Mortimer P. Starr. "Isolation and characterization of host-independent bdellovibrios." Journal of bacteriology 100.2 (1969): 769-785.
25. Varon, M., and M. Shilo. 1980. Ecology of aquatic bdellovibrios, p. 1–48. In Advances in aquatic microbiology, vol. 2. Academic Press, New York, N.Y.
26. Rendulic, S. et al. A predator unmasked: Life cycle of Bdellovibriobacteriovorus from a genomic perspective. Science 303, 689–692 (2004).
27. Dwidar, M., Hong, S., Cha, M., Jang, J. & Mitchell, R. J. Combined application of bacterial predation and carbon dioxide aerosols to effectively remove biofilms. Biofouling 28, 671–680 (2012).
28. Dwidar, M., Leung, B. M., Yaguchi, T., Takayama, S. & Mitchell, R. J. Patterning bacterial communities on epithelial cells. PloS One 8, e67165 (2013).
29. Corliss, J. O. (n.d.). Protist - Fossil protists and eukaryotic evolution. Retrieved March 14, 2016.
30. L. S. Thomashow, S. C. Rittenberg, J. Bacteriol. 163, 1038 (1985).
31. J. C. Burnham, T. Hashimoto, S. F. Conti, J. Bacteriol. 96, 1366 (1968).
32. M. Shilo, Curr. Top. Microbiol. Immunol. 50, 174 (1969).
33. H. B. Fackrell, J. Robinson, Can. J. Microbiol. 19, 659 (1973).
34. Flemming, H. C., Neu, T. R. & Wozniak, D. J. The EPS matrix: The ''House of biofilm cells'' J Bacteriol 189, 7945–7947 (2007).

35. Izano, E. A., Amarante, M. A., Kher, W. B. & Kaplan, J. B. Differential roles of poly-N-acetylglucosamine surface polysaccharide and extracellular DNA in Staphylococcus aureus and Staphylococcus epidermidis biofilms. Appl Environ Microbiol 74, 470–476 (2008).
36. Iwase, T. et al. Staphylococcus epidermidisEsp inhibits Staphylococcus aureus biofilm formation and nasal colonization. Nature 465, 346–U100 (2010).
37. Lauderdale, K. J., Malone, C. L., Boles, B. R., Morcuende, J. &Horswill, A. R. Biofilm dispersal of community-associated methicillin-resistant Staphylococcus aureus on orthopedic implant material. J Orthop Res 28, 55–61 (2010).
38. Park, J. H. et al. Extracellular protease in Actinomycetes culture supernatants inhibits and detaches Staphylococcus aureus biofilm formation. Biotechnol Lett34, 655–661 (2012).
39. Monnappa, A. K.,Dwidar, M.&Mitchell, R.J. Application of bacterial predation to mitigate recombinant bacterial populations and their DNA. Soil BiolBiochem 57, 427–435 (2013).
40. Cotter TW, Thomashow MF. A conjugation procedure for Bdellovibrio bacteriovorus and its use to identify DNA sequences that enhance the plaque-forming ability of a spontaneous host-independent mutant. J Bacteriol. 1992 Oct;174(19):6011–6017. [PMC free article][PubMed]
41. Dori-Bachash, M., Dassa, B., Pietrokovski, S. &Jurkevitch, E. Proteome-based comparative analyses of growth stages reveal new cell cycle-dependent functions in the predatory bacterium Bdellovibrio bacteriovorus. Appl Environ Microbiol 74, 7152–7162 (2008).
42. Van Essche, Mark, et al. "Killing of anaerobic pathogens by predatory bacteria." Molecular oral microbiology 26.1 (2011): 52-61.
43. Schwudke, D., Linscheid, M., Strauch, E. et al. (2003) The obligate predatory Bdellovibrio bacteriovorus possesses a neutral lipid A containing alpha-d-mannoses that replace phosphate residues: similarities and differences between the lipid As and the lipopolysaccharides of the wild type strain B. bacteriovorus HD100 and its host-independent derivative HI100. J BiolChem 278: 27502–27512.
44. Nitsche-Schmitz, D. P., Rohde, M. &Chhatwal, G. S. Invasion mechanisms of Gram-positive pathogenic cocci. ThrombHaemostasis 98, 488–496 (2007).
45. Essche, M. V., Quirynen, M., Sliepen, I., Loozen, G., Boon, N., Eldere, J. V., &Teughels, W. (2010). Killing of anaerobic pathogens by predatory bacteria. Molecular Oral Microbiology,26(1), 52-61.