International Research Journal of Microbiology Vol. 12(3) pp. 1-3, May, 2023
Available online http://www.interesjournals.org/IRJM

Review Article

Biofilms from *Pseudomonas aeruginosa*

Aurora Elegane*

Department of Microbiology, Federal University of Rio De Janerio

*Corresponding Author's E-mail: aurora@fu.ac.rdj

Received: 01-May-2023, Manuscript No. IRJM-23-98401; **Editor assigned:** 03-May-2023, PreQC No. IRJM-23-98401 (PQ); **Reviewed:** 17-May-2023, QC No. IRJM-23-98401; **Revised:** 19-May-2023, Manuscript No. IRJM-23-98401 (R); **Published:** 26-May-2023, DOI: 10.14303/2141-5463.2023.43

Abstract

Pseudomonas aeruginosa is an opportunistic human pathogen that can cause severe acute and chronic infections in people whose immune systems are compromised. Its capacity to form biofilms that are resistant to antibiotics is to blame for its well-known persistence in clinical settings. Biofilm is a structure that is mostly made of autogenic extracellular polymeric substances. It serves as a scaffold to hold the bacteria together on surfaces, protects them from environmental stresses, prevents phagocytosis, and gives them the ability to colonize and stay in the same place for a long time. *P. aeruginosa* biofilms, their stages of development, and the molecular mechanisms by which biofilms invade and persist are reviewed in this article. Explosive cell lysis within bacterial biofilms to produce essential communal materials, as well as interspecies biofilms of *P. aeruginosa* and commensal Streptococcus that prevent *P. aeruginosa* from becoming virulent and may even improve disease conditions, will also be discussed. Late examination on diagnostics of *P. aeruginosa* diseases will be researched. In the end, therapeutic strategies for treating *P. aeruginosa* biofilms will be compiled, along with their benefits and drawbacks.

Keywords: Chronic infections, Biofilm, Autogenic polymeric substances, Environmental stresses, Communal materials

INTRODUCTION

Pseudomonas aeruginosa is a common Gram-negative bacterium that is responsible for both nosocomial infections and fatal infections in immune compromised individuals, such as cancer patients, post-surgery burn victims, and HIV-infected individuals. The World Health Organization designated *P. aeruginosa* as a priority pathogen for the research and development of new antibiotics in 2017 because it was recognized as one of the bacteria that posed the greatest threat to human life. Due to *P. aeruginosa's* adaptability and high intrinsic antibiotic resistance, common antimicrobial agents like antibiotics frequently lack efficacy, leading to an increase in mortality. The ability of *P. aeruginosa* to form biofilms, which shield them from environmental stresses, prevents phagocytosis, and confers capacity for colonization and long-term persistence, also hinders treatment of these infections. Such capacity is elevated by compelling cell-to-cell interchanges inside the microbial networks of *P. aeruginosa* known as majority detecting. Consequently, highly structured biofilms can form, which are frequently observed in patients with chronic infections like rhinosinusitis, wound infection, and lung infection. Over 90% of chronic wound infections are thought to be significantly influenced by biofilms, which can impede wound healing. Chronic wound infections affected approximately 6.5 million patients in the United States alone, posing a significant health care burden and having devastating financial repercussions worth more than $25 billion annually (Gale MJ, 2015).

P. aeruginosa biofilms, from their composition, structure, and development processes all the way up to the extraordinary abilities of P. aeruginosa to invade the immune system of its host and evade antibiotic treatments through biofilm-mediated resistance, which is primarily regulated by quorum sensing. With regards to difficulties confronting *P. aeruginosa* crushing diseases, ongoing diagnostics and restorative methodologies will be examined (Wu W, 2015).

Biofilm

The majority of bacteria can attach to a variety of surfaces in nature and form biofilms. The biofilm is a complex total of microorganisms encased in a self produced lattice of

Extracellular Polymeric Substances (EPS) and is one of the critical methodologies for the endurance of species during startling changes of living conditions like temperature vacillation and supplement accessibility. A biofilm's bacteria can evade host immune responses and resist antibiotics up to 1000 times better than planktonic bacteria. *P. aeruginosa* is a notable biofilm previous, which makes it an incredible model to study biofilm arrangement. *P. aeruginosa* needs a strong biofilm to compete, survive, and take over the cystic fibrosis lung polymicrobial environment. Additionally, *P. aeruginosa* successfully colonizes a wide range of surfaces, including medical devices like urinary catheters, implants, and contact lenses, as well as equipment for the food industry (Gomila A, 2018).

Composition of biofilm

One of the most important strategies for species survival against unanticipated changes in living conditions like temperature and nutrient availability is the biofilm, which is a complex aggregate of bacteria enclosed in a self-generated matrix of Extracellular Polymeric Substances (EPS). Polysaccharides, extracellular DNA (eDNA), proteins, and lipids make up the majority of the *P. aeruginosa* biofilm matrix, as evidenced by the research presented here. The network, which is liable for more than 90% of biofilm biomass goes about as a framework for grip to biotic and abiotic surfaces and safe house for encased microbes in cruel natural circumstances (anti-toxins and host safe reactions). Additionally, it supplies the biofilm community with a variety of public goods, including enzymes, cytosolic proteins, and vital nutrients. Communication between cells is also made easier by the matrix. Psl, Pel, and alginate are the three exopolysaccharides that play a significant role in biofilm architecture stability, surface attachment, and formation. Following is a discussion of each exopolysaccharide's function.

Psl is a neutral pentasaccharide that typically consists of l-rhamnose, d-glucose, and d-mannose moieties. During the initiation of biofilms in both nonmucoid and mucoid strains, this exopolysaccharide is required for sessile cells—cells that are attached to a surface—adhesion to surfaces and cell-to-cell interactions. The following are characteristics of Psl: i) Psl benefits biofilm communities but not unattached populations; ii) Mixed biofilms with Psl-producing cells showed better growth of non-Psl-producing cells (iii) Psl positive populations outnumber Psl negative populations during biofilm growth; (iv) Psl non-producers cannot profit from Psl producers . Psl contributes to the structural stability of a mature biofilm by occupying the periphery of the mushroom-like structure. Expanded Psl articulation is connected to enlistment of cell totals in a fluid Culture which is an aggregate seen in CF patients' sputum. Psl is a signaling molecule that encourages the production of c-di-GMP (bis-(3′-5′)-cyclic dimeric guanosine monophosphate), who's elevated level causes biofilms to be thicker and more durable. Psl is also effective in preventing persistent infection because it protects biofilm bacteria from antimicrobials and neutrophil phagocytosis (Pang Z, 2017).

Alginate is predominately delivered in the biofilm of mucoid Pseudomonas strains due to a transformation in mucA22 allele. The mucoid phenotypes, which indicate the transition from an acute to a chronic infection, are predominant in CF isolates. Mannuronic and guluronic acid residues make up alginate, a negatively charged acetylated polymer. Many significant elements of alginate including biofilm development, insurance from phagocytosis and opsonization, and diminished dispersion of anti-microbials through the biofilm has been proven and factual. The viscoelastic properties of biofilms are influenced by the ratios of mannuronic acid to guluronic acid, which impair cough clearance in the lungs of CF patients with *P. aeruginosa* infection. One of the most important components of biofilms is extracellular DNA (eDNA), which is released into the environment by cell lysis. Through the endolytic activity of endolysin Lys, which is encoded in the R- and F-pyocin gene cluster, environmental stress, such as antibiotic treatment, can lead to cell lysis. This can happen in the early stages of biofilm formation as well as during the planktonic phase, when rod-shaped bacteria rapidly transform into round cells as a result of structural damage to the cell wall and lysis. The delivered eDNA, cytosolic proteins and especially RNA are therefore epitomized into layer vesicles (MVs) which are framed through film parts starting from the lysed cells. The surface and stalk of the mushroom-like microcolonies can also contain eDNA . eDNA plays a role in a number of processes: i) as a source of nutrients for the biofilm's bacteria; ii) supporting cell association and arrangement by means of jerking motility; (iii) as a cation chelator that connects with divalent cations (Mg2+ and Ca2+) on the external film what's more, thusly enacts the sort VI discharge framework which spreads harmfulness factors inside the host; (iv) the affidavit of eDNA caused biofilm climate and contamination destinations to become acidic, restricting the entrance of antimicrobial specialists; (v) the neutrophil-activated inflammatory process can be influenced by the presence of eDNA in P. aeruginosa biofilms (Moradali MF, 2017) .

Significance of Quorum in biofilm development

The advancement of *P. aeruginosa* biofilms requires populace wide coordination of person cells inside bacterial networks. *P. aeruginosa* can communicate between individual cells and ultimately orchestrate collective behavior, which is necessary for the adaptation and survival of whole communities, thanks to the use of multiple interconnected signal transduction pathways known as Quorum Sensing (QS). In response to changes in cell density and environmental cues or stresses, *P. aeruginosa* enters the QS mode. QS Includes the creation, discharge and collection of flagging atoms called autoinducers (man-made intelligence) whose particularity and focus are detected by transcriptional controllers, bringing about the outflows of explicit arrangements of qualities on a populace wide

scale. Notwithstanding biofilm advancement, QS has been connected to the guideline of other physiological cycles, including destructiveness factor creation, stress resistance, metabolic change and host-organism associations. As a result, new targets for alternative or complementary treatments to conventional antibiotics and antimicrobials may be discovered by comprehending and controlling these chemical communication systems. The Las, Rhl and PQS frameworks in the QS organization of *P. aeruginosa* assume significant parts in the creation of the useful components that affect biofilm improvement. These incorporate rhamnolipid , pyoverdine , pyocyanin , Pel polysaccharides , furthermore, lectins . Rhamnolipid is a glycolipidic compound (also known as a biosurfactant) with rhamnose in it. It keeps the pores and channels between microcolonies open so that liquid and nutrients can pass through mature biofilms. Pyoverdine is able to transport iron from the environment to the cell, which is necessary for the formation of biofilms. Twitching motility is preferred over sessile growth in an iron-limited environment, preventing biofilm formation. A cytotoxic secondary metabolite, pyocyanin causes cell lysis and releases the DNA of cells into the extracellular space (eDNA–one of the biofilm components) (Romling U, 2012) (Sen CK, 2009).

Pyocyanin has the ability to bind to the eDNA and increase the viscosity of the solution. This increases the physicochemical interactions between biofilm matrices and the environment and encourages cellular aggregation. In the biofilm matrix, cationic-anionic interactions between pel polysaccharides and eDNA can also strengthen the biofilm structure. Lectins are soluble proteins that are found in the outer membrane. There are two types of lectins: LecA, which binds to galactose and its derivatives, and LecB, which binds to fucose, mannose, and oligosaccharides that contain mannose. As a result of their adhesive properties, lectins are able to adhere to biological surfaces like epithelium and mucosa while also retaining cells and exopolysaccharides in a growing biofilm (Donlan RM, 2002) (Rollet C, 2019).

CONCLUSION

P. aeruginosa's capacity to form dense and persistent biofilms makes it difficult to treat infections caused by *P. aeruginosa* with antibiotics. Polysaccharides (Pel, Psl, and alginate) and extracellular DNA make up the biofilms of *P. aeruginosa*. These biofilms protect the bacterial communities from antimicrobial agents' exogenous stresses. Quorum sensing, a sophisticated cell communication system that works in a hierarchical manner and is actively inducible when cell density increases or when nutrients (like iron and phosphate) are restricted, is responsible for *P. aeruginosa's* extraordinary ability to form biofilms. When multiple species are also involved in the formation of polymicrobial communities within the biofilms, the complexity of antimicrobial treatments for *P. aeruginosa* biofilm is increased. *P. aeruginosa* can now be identified through the development of molecular diagnosis techniques. *P. aeruginosa's* genetic, microbial physiology, and biochemical markers, which can be characterized using, for instance, polymerase chain reaction, cell cultures for antibiotic-resistance profiling, and nanoparticle biosensor, respectively, are crucial to these techniques. The enrichment of bacterial cells and the use of biochemical markers for detection, the absence of a biomarker database, as well as the high cost and/or time commitment, remain major obstacles to diagnosis. The biofilm lifestyle, composition, and phenotypes vary depending on a number of factors, including nutrition conditions and the presence of other bacterial species, making it difficult to translate these promising findings into clinical trials. In order to provide high-throughput and specific diagnosis at an early stage of *P. aeruginosa* growth prior to the development of biofilms, further research is required on the development of more advanced techniques. On the other hand, a deeper comprehension of the genetic pathways that underlie all biofilm life cycles in *P. aeruginosa* merits further investigation. This knowledge could assist in making well-informed decisions regarding the development of therapeutic strategies that could hinder the abilities of bacterial attachment and biofilm maturation.

REFERENCES

1. Gale MJ, Maritato MS, Chen YL, Abdulateef SS, Ruiz JE (2015). Pseudomonas aeruginosa causing inflammatory mass of the nasopharynx in an immunocompromised HIV infected patient: A mimic of malignancy. 1: 40-43.
2. Wu W, Jin Y, Bai F, Jin S (2015). Pseudomonas Aeruginosa. In Molecular Medical Microbiology, 2nd ed. Academic Press Cambridge USA. 753-767.
3. Gomila A, Carratalà J, Badia JM, Camprubí D, Piriz M, et al (2018). Preoperative oral antibiotic prophylaxis reduces Pseudomonas aeruginosa surgical site infections after elective colorectal surgery: A multicenter prospective cohort study. BMC Infect Dis. 18: 507.
4. https://www.who.int/publications-detail-redirect/WHO-EMP-IAU-2017.12
5. Pang Z, Raudonis R, Glick BR, Lin TJ, Cheng Z (2019). Antibiotic resistance in Pseudomonas aeruginosa: Mechanisms and alternative therapeutic strategies. Biotechnol Adv. 37: 177-192.
6. Moradali MF, Ghods S, Rehm BH (2017). Pseudomonas aeruginosa Lifestyle: A Paradigm for Adaptation, Survival, and Persistence. Front Cell Infect Microbiol. 7: 39.
7. Romling U, Balsalobre C (2012). Biofilm infections, their resilience to therapy and innovative treatment strategies. J Intern Med. 272: 541-561.
8. Sen CK, Gordillo GM, Roy S, Kirsner R, Lambert L, et al (2009). Human skin wounds: A major and snowballing threat to public health and the economy. Wound Repair Regen. 17: 763-771.
9. Donlan RM (2002). Biofilms: Microbial life on surfaces. Emerg Infect Dis. 8: 881-890.
10. Rollet C, Gal L, Guzzo J (2019). Biofilm-detached cells, a transition from a sessile to a planktonic phenotype.