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Pathogenicity and Virulence Factors of *Pseudomonas aeruginosa*: A Mini Review

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ABSTRACT

Objective: *Pseudomonas aeruginosa, also known as P. aeruginosa, is a Gram-negative opportunistic bacteria that can infect people with burn injuries, cancer, immunodeficiency, chronic obstructive pulmonary disease (COPD), cystic brosis, and severe infections as COVID-19 that need ventilation. Additionally, P. aeruginosa is frequently employed as a model bacterium in all fields of biology. Due to the extensive use of antibiotics and the sluggish development of effective antimicrobials, new theoretical and practical platforms are needed to screen and create mechanism-tested innovative medications to treat intractable infections, especially those caused by strains that are resistant to multiple treatments.* ***Method:*** *This article provides a comprehensive overview and discussion of the present status of P. aeruginosa biophysical features, behaviors, virulence factors, invasive regulators, and host defense mechanisms against its infection.* ***Results:*** *These findings point to new directions for future investigation and aid in the creation of innovative and/or substitute therapies to combat this clinically important infection.* ***Novelty:*** *Along with ongoing, vigourous attempts to comprehend P. aeruginosa bacterial pathogenesis, including virulence factors (LPS, quorum sensing, two-component systems, six-type secretion systems, outer membrane vesicles (OMVs), CRISPR-Cas and their control). the mechanisms of drug resistance caused by mammalian cell signaling pathways and known or unknown bacterial virulence factors.*

INTRODUCTION

Pseudomonas aeruginosa, an opportunistic Gram-negative pathogen, is a leading cause of hospital-acquired infections and poses a significant health threat, particularly to immunocompromised individuals [1]. This bacteria is classified as multidrug-resistant because it demonstrates both innate and acquired resistance mechanisms, resulting in high resistance rates against several antibiotic classes [2].

Pseudomonas aeruginosa infections are common, especially among hospitalised patients. *P. aeruginosa* is the third most common bacterium in urinary tract infections and surgical site infections, both occurring at 11%, and the second most common organism in nosocomial pneumonia, accounting for 17% of cases. According to the National Nosocomial Infections Surveillance (NNIS) System, it accounts for 9% of nosocomial infections, making it the fifth most prevalent bacterium overall [3].

Biofilm-forming bacteria are very different from free-floating bacteria. Growing in multicellular clusters encircled by an extracellular matrix that the bacteria themselves create is a prominent feature of biofilm-associated bacteria [4]. There are metabolically dormant cells in the bacterial community because this matrix shields the cells from outside influences and prevents tiny molecules from diffusing [5].

Communities of stable biofilms have the ability to develop motile cells, which are capable of quickly reproducing and spreading. Therefore, biofilms not only protect bacteria from the host's defense mechanisms, such as phagocytosis, but they also serve as a continuous reservoir of germs during the course of antibiotic treatment. In affluent nations, it is estimated that more than 60% of bacterial illnesses managed by physicians are linked to biofilm formation [6].

P. aeruginosa is high resistant to many types of antibiotics. There are mobile genetic elements, such as plasmids, transposons, and integrons, that make it easier for genes to be acquired through the process of horizontal gene transfer. Among these, integrons are known as natural gene capture systems in bacteria and play a significant role in the development of resistance to multiple kinds of drugs. These elements consist of two conserved regions (integrase and recombination site) separated by a variable region that harbors integrated gene cassettes, often carrying antibiotic resistance genes. Gene cassettes are mobile and typically contain a single gene along with a specific recombination site, known as the 59-base element. Therefore, they are essential for the lateral dissemination of antibiotic resistance genes among bacteria [7].

Various typing methods have been employed to investigate the genetic diversity and evolutionary patterns of *P. aeruginosa*, known for its high genetic variability. The advent of Whole Genome Sequencing has significantly advanced the study of the molecular epidemiology of Multi-Drug Resistant (MDR) P. aeruginosa [8].

More than 130 unique gene cassettes have been found in class 1 integrons, predominantly encoding proteins that confer resistance to all principal classes of antibiotics. This encompasses the family of quaternary ammonium compounds, erythromycin, aminoglycosides, sulfonamides, quinolones, chloramphenicol, fosfomycin, trimethoprim, β-lactams, and other clinically relevant antibiotics [9].

Metallo-β-lactamases (MBL) belong to Ambler class B and are resistant to clavulanic acid. These enzymes require divalent zinc cations as cofactors for their activity and can be inhibited by EDTA and other chelating agents that target divalent cations [10], [11].

Many integrons that carry gene cassettes for MBL also contain additional cassettes, such as those for aminoglycosides or chloramphenicol. As a result, the transfer of integrons can facilitate the simultaneous transfer of a complex multidrug-resistant phenotype. The spread of MBL genes among Gram-negative pathogens occurs through mobile DNA elements, which helps explain why the same gene may be associated with plasmids or integrated into chromosomes in different strains [12], [13], [14].

RESEARCH METHOD

Pathogenesis of *Pseudomonas aeruginosa*

Because of its metabolic flexibility, *Pseudomonas aeruginosa* is an opportunistic bacterium that can adapt to many habitats and persist in both wild and clinical settings [15]. Although infections in healthy people are rare, some groups are more susceptible than others, such as those with cystic fibrosis, those with severe burns, and people with weakened immune systems, such as HIV-positive people or cancer patients undergoing chemotherapy [16], [17], [18].

The respiratory system is the predominant location for infections induced by *P. aeruginosa*. The infection predominantly manifests as acute ventilator-associated pneumonia in hospital environments or as a significant pathogen contributing to chronic destructive lung disease in individuals with cystic fibrosis [19].

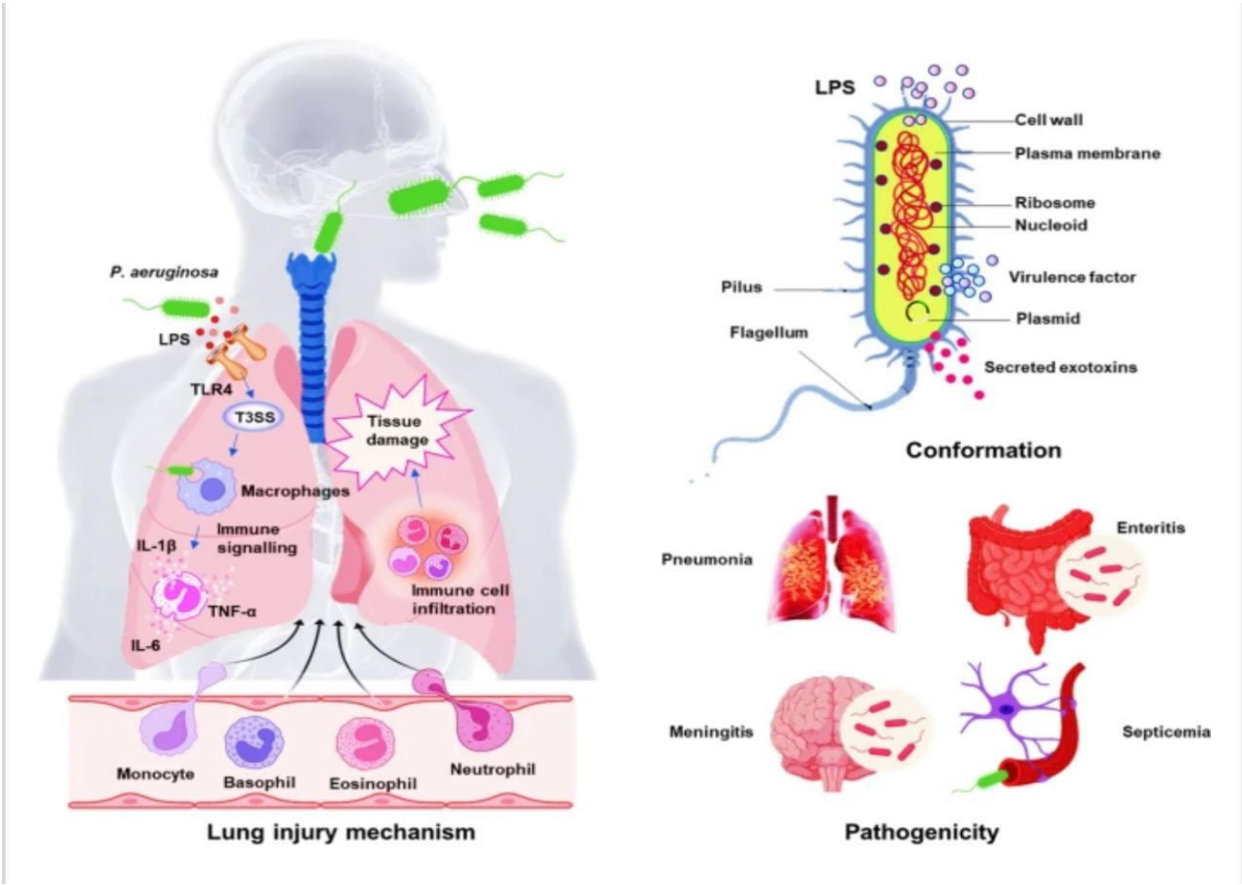

Figure 1. Pathogenesis diagram for P. aeruginosa. P. aeruginosa may infect practically every organ and can be found everywhere, including medical settings.

RESULTS AND DISCUSSION

Virulence Factors of *P. aeruginosa*

The capacity of *P. aeruginosa* to form ordered communities, or biofilms, when adhered to biotic or abiotic surfaces is one of the main elements influencing its pathogenicity. According to the research, *P. aeruginosa* shows a number of virulence factors that promote its pathogenicity, and Quorum Sensing, a cell-to-cell communication technique, is responsible for controlling the production of many virulence factors and the growth of biofilm. Some of these factors facilitate adhesion, while others disrupt intracellular signalling in host cells, affecting the integrity of the extracellular matrix. By infecting the host and eluding its immune system, this bacteria may cause a variety of ailments, making infections very challenging to treat [19].

Adhesion proteins, toxins including Exotoxin A and Exoenzyme S, and the Nan1 and Las genes are some of the virulence factors of P. aeruginosa. The outer membrane proteins, such as OprI and OprL, play a crucial role in bacterial interaction with the environment and contribute to antibiotic resistance. This is due to their involvement in efflux transport systems that influence cell membrane permeability, thereby enhancing bacterial resistance to available treatments [20].

P. aeruginosa secretes various virulence factors, including exotoxin A, exoenzyme S, and several proteases such as protease I (neutral protease) and protease III (alkaline phosphatase). Additionally, it produces elastase, hemolysins (both thermolabile phosphatase and thermostable acid glycolipid), as well as enterotoxin, collagenase, lecithinase, and lipase [21].

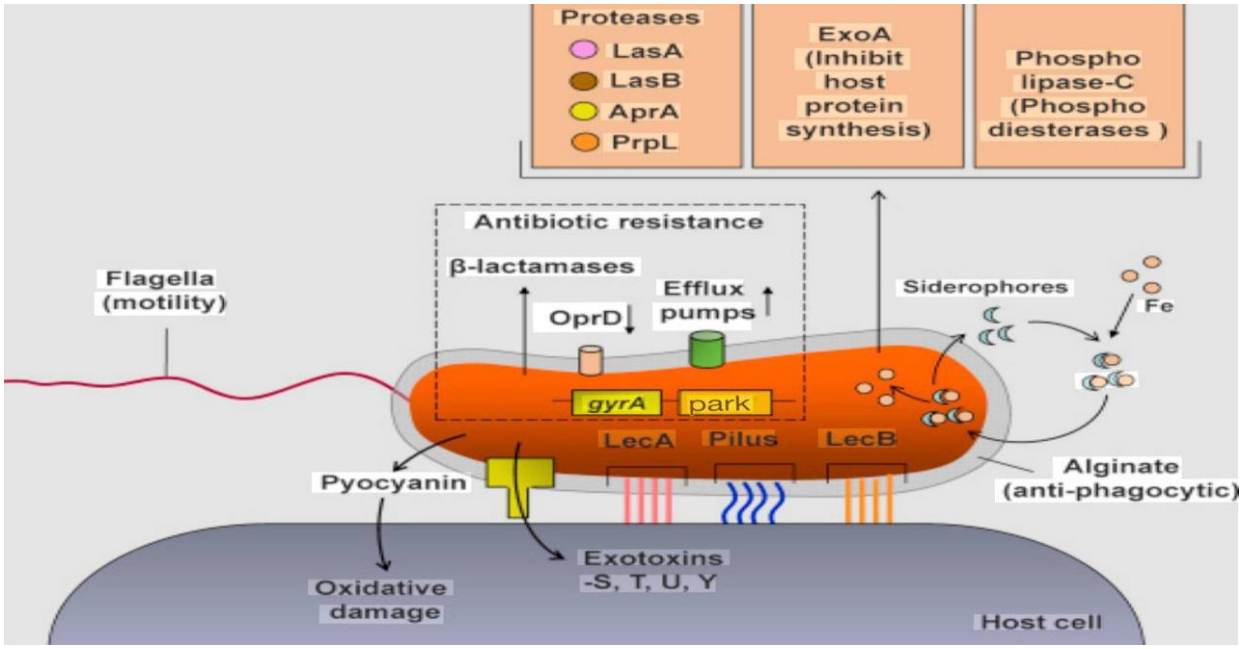

Figure 2. Schema of Virulence Factors of P. aeruginosa.

Pili (Fimbriae)

The electrostatic surface analysis of modeled pilus fibers derived from *P. aeruginosa* pilin monomers suggests that an exposed region of positive charge may be a universal characteristic of all type IV pili [22]. Among the many functions that are associated with DNA, type IV pili are responsible for natural transformation and the production of biofilm. *P. aeruginosa* has the ability to bind DNA through its pili, a function that relies on the intact quaternary structure of the pilus. Studies have shown that the pilus tip plays a key role in this process, as tip-specific antibodies inhibited DNA binding, whereas base-specific antibodies did not have the same effect. During the process of biofilm development, both in vivo during infection and ex vivo on abiotic surfaces, it is possible that DNA binding through the Pilus pathway is essential [23].

For many Gram-negative human infections, type IV pili are crucial colonisation factors. Despite being essential for infection, these filamentous structures are adaptable and perform a variety of bacterial tasks. In addition to their role in colonization, type IV pili help different bacterial species with twitching motility, biofilm formation, natural transformation, and bacteriophage infection [24].

Capsule (Alginate)

Alginate, a capsule-like polysaccharide produced by the opportunistic bacteria *Pseudomonas aeruginosa*, is essential for eluding host defences. The majority of the proteins involved in the synthesis of alginate are encoded by the algD operon, a 12-gene cluster. The gene that encodes the enzyme AlgL, a lyase that degrades alginate, is included in this operon [25].

Molecularly speaking, alginate is a polysaccharide that is simple, unbranched, and has a high molecular weight. β-D-mannuronic acid and its C5 epimer, α-L-guluronic acid, are the two uronic acids that make up this chemical. In addition to Pseudomonas species, several bacterial species in the genus Azotobacter can also produce alginate during the cyst formation process. This material is integrated into the cyst's outer layer of defense. Alginate, which is a part of the gelatinous cell wall and is present in brown algae, is another important source of alginate [26].

Enzymes and toxins

Pseudomonas aeruginosa is capable of producing a wide range of extracellular toxins. Phytotoxic substances, colours, hydrocyanic acid, proteolytic enzymes, phospholipase, enterotoxin, exotoxin, and biofilm-forming slime are some of these toxins [27].

A family of protein exotoxins produced by *P. aeruginosa* is the main factor that determines its pathogenicity. Leukopenia, acidosis, circulatory collapse, hepatic necrosis, pulmonary edoema, haemorrhage, and renal tubular necrosis are all possible side effects of these exotoxins [28].

There is a possibility that phospholipase plays a role in the breakdown of pulmonary surfactant, which ultimately leads to atelectasis. When combined with the necrosis of pulmonary tissue, this event has the potential to make a significant contribution to the pathogenesis of lung injury in persons who have pneumonia caused by *Pseudomonas aeruginosa.* Moreover, strains of *P. aeruginosa* can produce an enterotoxin that is probably responsible for diarrheal illnesses [29].

Pseudomonas exotoxin A (ToxA or PE) is an ADP-ribosylating toxin that has been extensively studied as the effector component of molecularly targeted poisons. The toxA gene encodes exotoxin A, a type II secreted extracellular enzyme. In the human host, this enzyme induces necrosis, severe tissue injury, and cell death, either independently or in conjunction with other hydrolases. Exotoxin A is an ADP-ribosyl transferase that inhibits protein synthesis in mammalian cells by transferring an ADP-ribosyl group to elongation factor 2 [30].

A significant number of *P. aeruginosa* strains are responsible for the production of exotoxin A, which is the primary virulence component. It is integral to the pathogenesis of *P. aeruginosa*. This toxin is acknowledged as extremely harmful to mammalian cells [31].

Flagella

Pseudomonas aeruginosa generally has a solitary unsheathed polar flagellum, which serves many functions in virulence alongside its principal role in swimming motility. Unlike the peritrichous flagella seen throughout the cell surface of multiflagellated enteric bacteria, this flagellum is polar in position, indicating a distinct variation in the quantity and placement of these bacteria [32].

Enteric bacteria's flagellum and *P. aeruginosa* are quite similar. However, aside from those necessary to understand its function and the notable differences in its assembly process compared to enteric bacteria, its structural characteristics are usually not described in detail [33].

The presence of glycosyl groups on *P. aeruginosa's* flagellin and the variety of genes generating its main structural protein, flagellin, are two examples of the differences. Additionally, a unique set of specialized regulators that are present at both the transcriptional and translational phases of the process are necessary for flagellar formation [34].

There is not enough information available on the flagellar mechanism of other *pseudomonads*. Organisms such as *P. putida*, *P. fluorescens*, and *P. syringae* have been shown to share structural traits and regulatory processes through the examination of genomic data from particular species [35].

Biofilm formation

Microorganisms and their extracellular materials combine to produce a biofilm, which is an ordered structure stuck to a surface. Its development has a substantial impact on foreign items or implanted medical devices inside the human body [36].

When bacteria grow and multiply, they exhibit a unique lifestyle known as the biofilm phenotype, in which their cells are grouped in immobile clusters. Once bacteria form a biofilm within the human body, the ensuing infection frequently becomes resistant to treatment and may develop into a chronic illness [37]. Biofilm-associated chronic infections are marked by a high degree of resistance to antibiotics and other traditional antimicrobial therapies, as well as a great capacity to evade host immune responses [38].

Bacterial populations that stick to surfaces and are enmeshed in a self-made matrix made of extracellular DNA, proteins, and polysaccharides are known as biofilms. These structures are thought to be important for the pathogenicity and persistence of around 80% of human microbial infections, including many diseases related to healthcare, particularly when internal medical devices are involved [39]. Antibiotic resistance in biofilm-forming bacteria can be 10–1,000 times higher than that of their free-floating (planktonic) counterparts, making treatment of these illnesses more difficult. Biofilms also help a variety of human infections survive in the environment, spread, and become more infectious [40].

Many different kinds of foreign materials have been created as part of medical developments in recent decades. Concerns regarding biofilm development and device-related infections have grown in importance as the usage of biomaterial-based devices in urology continues to expand. Biofilms are microbial communities with unique phenotypic characteristics that are composed of cells adhering to a surface and encased in a matrix rich in exopolysaccharides. According to research, biofilm development can begin quickly after catheter implantation because microbes stick to a layer of host proteins that covers the catheter's surface [41].

Biofilm-dwelling bacteria experience genetic activation that results in altered molecular targets, altered cell envelopes, and decreased susceptibility to antimicrobial therapies—a phenomenon known as intrinsic resistance. Antimicrobial doses 1,000–1,500 times greater than those needed to eradicate the same bacterial speBiofilm-dwelling bacteria experience genetic activation that results in altered cell envelopes, alte red molecular targets, and decreased susceptibility to antimicrobial therapies
a phenomenon knAntimicrobial doses 1,000–1,500 times greater than those needed to eradicate the same bacterial species in their planktonic state can be tolerated by these biofilm-associated bacteria. Early-stage biofilm formation has been reported to be effectively inhibited by beta-lactam antibiotics and aminoglycosides. On the other hand, because of their potent ability to penetrate biofilm structures, fluoroquinolones show effectiveness against both early and mature biofilms. Surprisingly, biofilms containing these antibiotics can still be seen one to two weeks after therapy ends [42]. Many scientists concur that antibiotics primarily work by slowing the growth of biofilms by focusing on free-floating (planktonic) bacteria that are not yet incorporated in the biofilm. Nonetheless, during the acute febrile stages of a biofilm-related infection, antimicrobial treatment remains both necessary and effective, as the febrile response is typically triggered by planktonic bacteria rather than those residing within the biofilm [45]

Quorum sensing

P. aeruginosa cells utilize the quorum sensing (QS) mechanism for communication by synthesizing tiny signalling molecules. These molecules regulate the expression of virulence factors, biofilm formation, secondary metabolite production, and host interactions in response to population density [46].

P. aeruginosa employs three primary interrelated quorum sensing (QS) mechanisms that can operate alone or in concert. A variety of QS signalling molecules are responsible

for regulating the pathways, which include las, rhl, pqs, and the recently discovered iqs system. The N-acyl homoserine lactones, also known as AHLs, are the ones that have been investigated the most. They are composed of a homoserine lactone ring that is connected to a fatty acyl side chain that has between four and twenty carbon atoms [47].

Disrupting quorum sensing (QS) by obstructing its signaling or intercepting signaling molecules is regarded as a vital strategy in formulating antibacterial and anti-disease measures, especially against pathogens such as *P. aeruginosa* in the medical field. The interruption of quorum sensing through the use of quorum sensing inhibitors (QSI) or the interception of signaling molecules by quorum-quenching enzymes (QQE) does, in fact, result in a reduction in the pathogenicity that is regulated by quorum sensing [48].

QSI is believed to be a naturally developed mechanism, either by QS-emitting organisms to recycle or eliminate their own QS signals or by QSI-producing organisms as part of a competitive interaction with QS signal-producing organisms. Consequently, bacteria that coexist with *P. aeruginosa* in the same ecological niche during infections could be potential sources of novel QSI molecules [49]. QS regulates various processes, including bioluminescence, sporulation, competence, antibiotic production, biofilm formation, and the secretion of virulence factors [50]

Hemolysin

Hemolysin is a cytolytic toxin present in microorganisms, characterized by its ability to lyse erythrocytes, a feature that is associated with the pathogenesis of these microorganisms [51]. The hemolysin of *Pseudomonas aeruginosa* has been discovered to function as a detergent, facilitating the solubilization of a variety of phosphatides. It substantially increased the enzyme activity rates when it was added to reaction mixtures that contained phospholipase C and phosphatides. The enhanced dispersion of the substrates was likely the cause of this stimulation of enzyme activity [52].

The hemolysin produced by *Pseudomonas aeruginosa* demonstrates a cytopathic effect on blood and tissue culture cells. Both the membrane and the cytoplasm are affected by its effects, which include the induction of lysis and the compromising of the cellular architecture. As a result of normal serum and albumin, the hemolytic activity of hemolysin is brought under control [53].

The hemolytic and cytolytic effects are attributed to the alteration of the molecular structure of the membranes. It is possible that the variability is due to the differential availability of reactive sites on the cells, or it may be an indication that the two activities are linked to two different enzymes [54].

Hemolysin may enhance virulence by increasing the availability of iron in the absence of aerobactin, thereby exerting a toxic effect on leukocytes and other nucleated cells [55].

Siderophores

Siderophores are diminutive organic compounds synthesized by microorganisms in conditions of iron scarcity, aiding in the acquisition of iron by these organisms. At a physiological pH range of 7.35 to 7.40, the ferric form of iron is considered insoluble and so unavailable in the environment [56].

Under these circumstances, bacteria synthesize siderophores that exhibit a high affinity for ferric iron. The ferric iron-siderophore complexes are translocated into the cytosol, where ferric iron is converted to ferrous iron, rendering it available to the bacterium. Siderophores have recently garnered significant interest owing to their prospective applicability across diverse domains. Microbial ecology makes use of them to encourage the proliferation of a wide variety of bacteria that cannot be cultivated, and they have the ability to alter the populations of microorganisms [57].

Iron is an essential metal that is required by all living things. It plays a role in a number of cellular processes, including the electron transport chain, and it is a cofactor for a great number of enzymes [58].

Microorganisms growing under aerobic conditions require iron for various functions, including the reduction of oxygen for ATP synthesis, the formation of heme, and other essential processes [59].

Siderophores, which are iron-chelating agents with a low molecular weight (<10 kDa), are produced in large quantities by a wide range of bacteria, including *Pseudomonas*, *Azotobacter*, *Bacillus*, *Enterobacter*, *Serratia*, *Azospirillum*, and *Rhizobium*, in response to environments that are lacking in iron. Siderophores form complexes with free iron and facilitate its transport into the cell via membrane receptor molecules, which are expressed by five genes within an operon that becomes inactivated once adequate iron has been absorbed by the cells [60].

Urease

Urease is acknowledged as a virulence factor of *P. aeruginosa* and is deemed a significant pathogenic element in bacteria. Through the action of this enzyme, urea is hydrolyzed into ammonia and carbonate, which then undergoes spontaneous decomposition, resulting in the production of an additional molecule of ammonia and carbonic acid. There is a high degree of prevalence of the urease phenotype across the whole bacterial kingdom, and gene clusters that encode this enzyme have been identified from a wide variety of bacterial species [61].

The role of urease is classified based on the type of infection, as revealed by the complete nucleotide sequence. In urinary tract infections, urease contributes to the formation of renal stones due to the breakdown of urea into ammonia and carbon dioxide, which increases the pH to around nine, causing salts to precipitate in mucus and form stones. Urease aids *P. aeruginosa* in surviving the stomach's acidic environment during gastrointestinal tract infections [62].

Lipase

Due to its importance in both medicine and industry, the lipase enzyme from *Pseudomonas aeruginosa* has been the subject of much research over the past few decades. It contributes to a number of waste treatment procedures, including as the bioremediation of oil waste. This enzyme's capacity to hydrolyze long-chain triglycerides is very well-known [63].

Lipases have been used to break down castor oil, either to produce technical lipases or to create new products. Some of these enzymes are thermostable, making them more suitable for demanding industrial conditions, and they account for over 65% of the global market [64].

These enzymes are utilized in various fields, particularly in the paper industry, detergents, pharmaceuticals, waste degradation, textiles, food processing, pharmaceuticals, leather tanning, silk degumming, liquid glue production, cosmetics, meat tenderization, cheese manufacturing, growth promotion, and many other applications [65].

P. aeruginosa produces several important enzymes, including protease, lipase, urease, and asparaginase, along with other compounds like alginate, which is widely utilized in various biotechnological applications. There has been growing interest in the lipase and protease enzymes of *P. aeruginosa* [66].

CONCLUSION

Fundamental Finding : Because *P. aeruginosa* is an opportunistic infection that causes significant morbidity, crippling illnesses, shorter life spans, and high mortality in humans, it has a sophisticated regulatory system that is interconnected and mutually controlled to deal with the harsh external and internal environment, and *Pseudomonas aeruginosa* has a high incidence of antibiotic resistance and the capacity to secrete a number of virulence factors that may be used in their pathogenicity, resulting in cell invasion and damage. **Implication :** *P. aeruginosa* is a serious infection that has been linked to increased patient morbidity and death and affects all demographics. **Limitation :** The long-running arms race between humans and *P. aeruginosa*, along with both a high rate of cell damage and antibiotic resistance, indicates persistent challenges in effective disease control. **Future Research :** Developing novel strategies to counteract the bacterium's growing dangers requires a deeper comprehension of these virulence traits, and further research and analysis are necessary because of the long-running arms race between humans and *P. aeruginosa*.

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