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Detection of The Psla And Pela Genes in Multidrug-Resistant Pseudomonas Aeruginosa Isolated from Respiratory Infections

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Abstract

Background: *Pseudomonas aeruginosa* is a leading opportunistic pathogen that have the ability to produce biofilms using secreted extracellular polymeric substances are typically controlled by the pslA and pelA genes. The current study was undertaken to determine the prevalence of pslA and pelA genes in MDR *P. aeruginosa* isolated from respiratory infections, and to assess its correlation with antibiotic resistance profile.

Methods: Cross-sectional study carried in Hilla Teaching Hospital, Babylon Province-Iraq during the period from July 2024 to January 2025. Methods: 87 patients who presented with clinically suspected respiratory tract infections. Antimicrobial susceptibility tests were conducted using the Kirby–Bauer disk diffusion method according to CLSI guidelines as follows: ceftazidime, imipenem, meropenem, ciprofloxacin, levofloxacin, gentamicin and amikacin. Molecular Detection of pslA and pelA GenesMultidrug-resistant isolates were selected to be used for the molecular detectionof the pslA genes by conventional PCR.

Results: Ciprofloxacin (85.0%) and levofloxacin (82.5%) had the highest resistance rates, followed by ceftazidime (77.5%), meropenem (75.0%), and imipenem (70.0%); however, amikacin had the lowest resistance rate at 45%. In molecular analysis, the pslA gene was identified in 95.0% of MDR isolates and pelA gene was found in 87.5%. The presence of pslA and pelA genes is significantly associated with resistance to β-lactams, carbapenems and fluoroquinolones ($p < 0.05$; however, aminoglycosides showed no significant correlation.

Conclusion: The Pseudomonas aeruginosa Respiratory Isolates of Multidrug Resistant Bacteria (MDR) exhibited high frequency of pslA and pelA genes which imply that biofilm formation took a major part in bacterial persistence and MDR. The major correlation with biofilm-associated genes and antimicrobial

resistance indicates the emergence of pathogenic clones which are well adapted and higher virulent.

Keywords: pslA, pelA, Pseudomonas aeruginosa, Respiratory Infections

Introduction

Pseudomonas aeruginosa is a clinically relevant Gram-negative opportunistic human pathogen and one of the most common worldwide causative agents of hospital-acquired infections. It is associated with a range of diseases such as pneumonia, ventilator-associated pneumonia (VAP), chronic respiratory tract infection, bacteremia, urinary tract infections (UTI) and wound infections (Mohammed et al., 2024). *P. aeruginosa* is particularly known for causing different types of respiratory infections, which are associated with a high morbidity and mortality in immunocompromised patients, cystic fibrosis or chronic obstructive pulmonary disease (COPD) patients and mechanical ventilation. Its incredible ability of motility, resistance to multiple environmental stresses and adaptation to among general antimicrobial pressure are the important causes that contribute to this pathogen persistence in healthcare settings as well as its emergence into a major health threat worldwide (Abdulhaq et al., 2019).

Multidrug-resistant (MDR) *P. aeruginosa* is now considered a major therapeutic problem. Multidrug resistance Multidrug-resistant (MDR) organisms are generally recognized as resistant to at least one agent in three or more classes of antimicrobial drugs (Elfadadny et al., 2024). Many of the intrinsic and acquired resistance mechanisms, such as low outer membrane permeability, β-lactamases production, over-production of efflux pumps, target site mutations, and horizontal gene transfer. These processes typically work together with virulence factors to not only repel the phagocytosis but also make the pathogens more resistance to drugs. At the same time, carbapenem-resistant P. aeruginosa has been included among the 'priority pathogens' identified by the World Health Organization (WHO) needing novel antimicrobial strategies developed urgently (Masoumi &, Keshavarzi, 2024).

The ability of *P. aeruginosa* to form biofilms is one of the key pathogenic features that contributes to persistence in clinical infections. Biofilms are biofilm organized communities of microbes encased in a matrix of self-produced extracellular polymeric material from which they get structural and protective support against the host immune defense and antimicrobial agents (Thi et al., 2020). Biofilm-dwelling bacteria mostly show more than hundred- fold antibiotic tolerance comparing to planktonic cells which cause chronic and recurrent infection. Biofilm formation is especially important in respiratory infections, including antibiotic-resistant bacterial aggregates that stick to epithelial surfaces and medical devices, promoting disease progression and resulting in adverse clinical outcomes (Murakami et al., 2017).

Polysaccharides also represent the polysaccharide if an external matrix of *P. aeruginosa* biofilms is composed mainly of three exopolysaccharides, alginate, Pel andPsl. Of these, Psl and Pel are essential during the initial attachment phase of biofilm development but also for maturation and maintenance (Chung et al., 2023). The polysaccharide Psl promotes adherence to surfaces and mediates cell-to-cell interactions, while Pel contributes to the architecture of biofilms as well as supports resistance to environmental stressors. Thus, Genes related to the production of these exopolysaccharides are considered major virulence factors (Al-Sheikhly et al., 2020).

PslA is a glycosyltransferase from psl operon that plays an important role in the biosynthesis of Psl polysaccharide, which is required for surface attachment and biofilm stability. Research has shown that disruption of psl genes strongly impairs biofilm formation and leads to increased susceptibility to antimicrobial agents, producing more susceptible bacteria (Omran & et al., 2026). Similarly, the pelA gene is responsible for providing one of the proteins necessary for the production of Pel polysaccharide and is part of the pel operon. In addition to allowing bacterial aggregation (Hachani et al., 2015), Pel mediated biofilms protect bacteria against host defences thus favouring infection chronicity as previously described using an intranasal pneumonic model in mice (Al-Sheikhly et al., 2019). Recent molecular studies have pointed to the high abundance of pslA and pelA genes in clinical P. aeruginosa isolates (Omran & et al., 2026).. Farhan et al. (2023) found that 94% and 86.4 % of biofilm-producing isolates were positive for pslA and pelA genes, respectively, alluding to their role in virulence as well as multidrug resistance (Kim et al., 2023). Likewise Masoumi and Keshavarzi (2024) showed a significant association between biofilm-associated genes and drug resistance in clinical isolates, where it was reported that strains with higher biofilm-forming capacity are associated with increased rates of multidrug-resistant. In addition, studies carried out in Iraq have also demonstrated the global dissemination of pslA and pelA genes among resistant P. aeruginosa isolates indicating that pslA and pelA are common genetic determinants associated with the persistence and pathogenicity of bacteria (Al-Sheikhly et al., 2020). While there is an expanding collection of evidence for the presence of biofilm-related genes, little information so far exists on the spread of pslA and pelA in multidrug-resistant P. aeruginosa strains isolated from respiratory tract infections. Because respiratory isolates are often linked to severe disease with high levels of antimicrobial resistance,

determining the mechanisms that cause biofilm formation is critical for the development of effective therapeutic and infection-control measures. Based on this information, the present study was aimed to investigate the detection of pslA and pelA genes in multidrug-resistant P. aeruginosa isolates obtained from respiratory infections and provide an understanding of their possible role in the pathogenicity and persistence of this clinically significant pathogen.

Materials and Methods

Patients and Methods

This study was a cross-sectional study conducted in Hilla Teaching Hospital at Babylon Province, Iraq during the period from July 2024 to January 2025. Eighty-seven patients having clinically suspected respiratory tract infections were included. Male and female patients 18 to75 years of age were included. Respiratory specimens were obtained from patients with clinical manifestations consistent with LRTI, including productive cough, dyspnea, fever and radiological evidence of pulmonary involvement. Patients with microbiologically proven Pseudomonas aeruginosa infection were exclusively included. Patients were excluded if they had received antimicrobial therapy within 72 hours before sample collection, had mixed bacterial infections or incomplete clinical records. The Scientific and Ethical Committee of the College of Medicine, University of Babylon approved the study protocol. Written informed consent was obtained from all participants prior to enrollment.

Sample Collection and Bacterial Isolation

All respiratory specimens were collected in sterile containers and immediately transported to the microbiology laboratory of Hilla Teaching Hospital for processing; these included sputum, tracheal aspirates, and bronchoalveolar lavage samples. The samples were inoculated in MacConkey agar, blood agar, and cetrimide agar (Oxoid Ltd., UK) incubated aerobically at 37°C for 24–48 h. Colony morphologies for *P. aeruginosa* (flat colonies and metallic sheen) along with β-hemolysis on blood agar, grape-like odor, and blue-green pigmentation were targeted during selection. Preliminary identification was conducted as per standard microbiological procedures using conventional microbiological and biochemical tests which included Gram staining, oxidase test, catalase test, citrate utilization test, motility test, Triple Sugar Iron (TSI) agar reaction oxidative-fermentative (OF) test and growth at 42°C according to Cheesbrough 2017. Isolates were confirmed as NTS using the API 20NE identification system (bioMérieux, France). Confirmed isolates were then held in tryptic soy broth with 20% glycerol at −80°C until further molecular characterization of the isolate. The quality control strain was Pseudomonas aeruginosa ATCC® 27853™.

Antimicrobial Susceptibility Testing

Antimicrobial susceptibility testing was carried out by the KirbyBauer disk diffusion method. Antimicrobial agents from the following classes that are commonly used against P. aeruginosa were tested:

1. Ceftazidime (30 µg) - Oxoid Ltd., UK
2. Imipenem – (10µg) ∗ Bio-Rad, France.
3. Meropenem (10 µg) – Bio-Rad, France.
4. Ciprofloxacin (Mast Diagnostics, UK) (5 µg).
5. Levofloxacin (5 µg,) – HiMedia Laboratories, India.
6. Gentamicin (10 µg) - Bio-Rad, France.
7. Amikacin (30 µg) – Bioanalyse, Turkey.

Inhibition zone diameters were measured and interpreted in accordance with CLSI criteria as susceptible, intermediate or resistant. Multidrug-resistant (MDR) isolates were defined as those exhibiting resistance to at least one antimicrobial agent in three or more antimicrobial classes. Quality Control strain for susceptibility testing: *Pseudomonas aeruginosa* ATCC® 27853™.

Genomic DNA Extraction

All confirmed MDR P. aeruginosa isolates were subjected to genomic DNA extraction using the GeneJET Genomic DNA Purification Kit (Thermo Fisher Scientific, Lithuania) as detailed by the manufacturer. Spectrophotometric methods were used to assess DNA concentration and purity, and extracted DNA samples were stored at −20 °C until consideration.

Detection of pslA and pelA Genes on Molecular Basis

Biofilm-associated genes pslA and pelA were detected by conventional polymerase chain reaction (PCR) using previously published gene-specific primers. The PCR amplification was performed in a 25 µL final reaction volume consisting of 12.5 µL of 2 × PCR Master Mix (Promega, USA), 1 µL (10 pmol/µL) for forward primer, and reverse primers respectively, 3 µL of DNA template, and the remaining amount completed with nuclease-free water. Amplification reactions were performed in an Eppendorf Mastercycler (Germany) following the thermal conditions: 95°C for 5 min, followed by 35 cycles at 95°C for 30 s, 58°C for 40 s, and then at 72 °C for45s with a final extension step of72°C for7min. Electrophoresis of PCR products was performed on 1.5% agarose gel with ethidium bromide (0.5 µg/mL) and visualized under ultraviolet illumination with a gel documentation system (Bio-Rad, USA). Molecular size marker

— a 100-bp DNA ladder was utilized. The positive control was Pseudomonas aeruginosa ATCC® 27853™ and as negative control was used the nuclease-free distilled water.

Statistical Analysis

Data were analyzed using version 26.0 of SPSS (IBM Corp., Armonk, NY, USA). Categorical variables were reported as frequencies and percentages. The Chi-square test or Fisher's exact test when appropriate was used to evaluate associations between the presence of pslA and pelA genes and antimicrobial resistance patterns (Motevasel et al., 2024). Statistical significance was set at $p < 0.05$ (table 1).

Table 1. List of primers used for amplification of ureC and hpmA genes

Gene	Amplicon Size (bp)	Annealing Temperature (°C)	Forward Primer (5′–3′)
pslA	202	52	TGCCTGGAACATAATCACCGT
pelA	312	52	GACTGGGTGGTGCTCGAAG

Results

The MDR *P. aeruginosa* isolates exhibited high resistance rates against most of the antimicrobial agents tested. As for the highest frequency of resistance were involved in ciprofloxacin (85.0%) and levofloxacin (82.5%), followed by ceftazidime (77.5%) and meropenem (75.0%). A relatively high percentage of imipenem resistance was also noted (70.0%), suggesting a global presence of carbapenem resistance among the isolates. Aminoglycosides were relatively better, with gentamicin and amikacin having the lowest resistance rates of 60.0% and 45.0%, respectively. 55.0% of all isolates were still susceptible to amikacin, making it the most effective antimicrobial agent on activity (Table 2).

Table 2. Rates of antibiotic resistance recorded in the isolates of *Pseudomonas aeruginosa*

Groups	Resistant Isolates No. (%)	Susceptible Isolates No. (%)
Ceftazidime	**31 (77.5)**	**9 (22.5)**
Imipenem	**28 (70.0)**	**12 (30.0)**
Meropenem	**30 (75.0)**	**10 (25.0)**
Ciprofloxacin	**34 (85.0)**	**6 (15.0)**
Levofloxacin	**33 (82.5)**	**7 (17.5)**
Gentamicin	**24 (60.0)**	**16 (40.0)**
Amikacin	**18 (45.0)**	**22 (55.0)**

Among the MDR *Pseudomonas aeruginosa* isolates, the pslA gene was detected in 95% of strains, while pelA was identified in 87.5%. (Table 3).

Table 3. Presence of pslA and pelA genes in MDR *Pseudomonas aeruginosa* isolates

Groups	Positive No. (%)	Negative No. (%)
pslA	38 (95.0)	2 (5.0)
pelA	35 (87.5)	5 (12.5)

In this study, a significant association was observed between pslA and pelA biofilm-associated genes and antimicrobial resistance, both on univariate analysis and multivariate logistic regression analysis, in multidrug-resistant Pseudomonas aeruginosa isolates. There were significant correlations between both genes and resistance to ceftazidime (p = 0.0071), imipenem (p = 0.0359), meropenem (P 0.05), signifying a relatively greater retention of efficacy of aminoglycosides versus biofilm-forming isolates.

Table 4. Association between antibiotic resistance and presence of pslA and pelA genes in the isolates of MDR *Pseudomonas aeruginosa*

Antibiotics	pslA Chi Square (P value)	pelA Chi Square (P value)
Ceftazidime	**5.84 (0.016*)**	**4.62 (0.032*)**
Imipenem	**6.27 (0.012*)**	**5.11 (0.024*)**
Meropenem	**7.03 (0.008**)**	**5.76 (0.016*)**
Ciprofloxacin	**8.45 (0.004**)**	**6.98 (0.008**)**
Levofloxacin	**7.81 (0.005**)**	**6.15 (0.013*)**
Gentamicin	**2.34 (0.126)**	**1.88 (0.170)**
Amikacin	**1.95 (0.162)**	**1.42 (0.234)**

*** Significant at P<0.05; ** High significant at P<0.001**

Discussion

Multidrug-resistant (MDR) *Pseudomonas aeruginosa* is an emerging cause of disease worldwide, with serious implications for therapeutic delivery in the treatment of respiratory tract infections, given the biofilm formation capability exhibited by this species promoting bacterial persistence and antibiotic failure. MDR isolates were found to be resistant to most of the tested antimicrobial agents in the present study. The strongest antimicrobial activity was recorded against adriamycin and acyclovir, lowest resistance level was noted for ciprofloxacin and levofloxacin; more resistance for ceftazidime, meropenem and imipenem; while amikacin had relatively better antibacterial effect. Molecular analysis also showed that genes associated with biofilm formation, namely pslA and pelA, were highly prevalent, and β-lactams, carbapenems, and fluoroquinolones were all significantly associated with these genes.

Those high rates of resistance to fluoroquinolones and carbapenems may suggest the horizontal dissemination of resistant *P. aeruginosa* strains among patients with respiratory infections; These results align with findings from Pang et al. (2019) highlighted the fact that P. aeruginosa uses a number of important intrinsic and acquired resistance mechanisms, such as efflux pumps, low outer membrane permeability (OM), target-site mutations and β-lactamases production to withstand different classes of antimicrobial agents. Similarly, Botelho et al. (2019) reported that the pandemic of carbapenem resistance among

clinical isolates: a data-driven crime against humanity Wang et al. (2019) have also reported the latest overview of global and local trends; carbapenem resistance among clinical isolates is now more prevalent in some regions than antibiotic-resistant *Staphylococcus aureus*, as well as requiring increasingly complex and expensive management that at times offers relatively poor therapeutic outcomes.

In this study, resistance to ceftazidime was also very high and may be attributed to the production of extended-spectrum β-lactamases (ESBLs) and AmpC β-lactamases commonly found among nosocomial isolates. Horcajada et al. (2019) reported similar observations on finding had reported a marked increase in the prevalence of cephalosporin resistance among P. aeruginosa strains from hospitalized patients due to prolonged exposure and selective pressure exerted by antimicrobial. Likewise, Koulenti et al. Meyer et al. (2020) reported alarming resistance rates in non-bacterial respiratory isolates from ICU patients, detecting nine different serovars with overall lower susceptibilities to ceftazidime and carbapenems, restricting available treatment options.

Amikacin being the least resistant among aminoglycosides and thus showing the highest susceptibility. This observation is consistent with the results of Karampatakis et al. (2023), who stated that amikacin continuously stayed at the forefront as one of the most potent agents towards MDR P. aeruginosa isolates, since it is less prone to enzyme modification than other aminoglycosides. This lower resistance to gentamicin and amikacin (up to 68.8%) can therefore provide clinicians with valuable therapeutic alternatives for severe respiratory infections.

Molecular analysis showed that the pslA gene was found in 95.0% of isolates and pelA gene was identified in 87.5%. Conclusion Our results demonstrate that biofilm-associated genes are common in respiratory MDR P. aeruginosa action to be taken. The dominance of pslA relative to pelA indicates that Psl polysaccharide synthesis may be particularly important in the earliest stages of biofilm development and bacterial colonization. Psl-mediated adherence is important for surface attachment and cell-cell aggregation, which are necessary steps in mature biofilm formation (Maunders & Welch, 2017).

The high prevalence of pslA gene was consistent with the finding by Farhan et al. (2023) that observed (94%) and (86.4%) pslA point mutation affected clinical isolates of biofilm producers and pelA present in all isolates respectively. Similarly, Al-Sheikhly et al. (2020) have shown that both genes are widely distributed among Iraqi isolates of P. aeruginosa which indicate that these genetic determinants may be highly conserved and contribute significantly to bacterial pathogenicity. Murakami et al. (2017) also showed evidence that disruption of psl genes significantly inhibits biofilm formation and reduces antibiotic tolerance, suggesting a crucial role for Psl polysaccharides in chronic infections.

In the current study, one other noteworthy result was a rather rates of characterizing both presence of pslA and pelA genes that were significantly associated with resistance to ceftazidime, carbapenems, and fluoroquinolones. A significant association was found in the correlation of pslA with ciprofloxacin resistance compared to that of other factors. These findings indicate that biofilm development evidently plays a major role in the multidrug resistance of P. aeruginosa. Biofilms act as fortresses that block the penetration of antibiotics, decrease the metabolic activity of bacteria and allow the appearance, within them, of persistent cells that can resist antimicrobial attacks (Hall & Mah, 2017).

The more substantial association for pslA compared to pelA suggests that Psl exopolysaccharides potentially have a greater impact on antimicrobial tolerance. Maunders and Welch (2017) proposed that Psl is principally relevant during initial biofilm establishment and maintenance and can act as a protective scaffold to help shield polysaccharide-producing bacterial communities from environmental stressors and host defenses. As a result, they are commonly found among the isolates that withstand antibiotic treatment for extended periods and acquire multidrug resistance.

The lack of a statistically significant association between aminoglycoside resistance and either the pslA or pelA gene indicates that resistance to gentamicin and amikacin may rely on mechanisms independent from biofilm formation (aminoglycoside-modifying enzymes, ribosomal target modification, efflux). Pang et al. (2019) reported similar observations that many resistance determinants act in concert to effect phenotypes in both species, where their relative contributions depend on the class of antimicrobial agent evaluated. A recent study has shown that chloroxylenol inhibited biofilm formation of *Pseudomonas aeruginosa* via a 2.25% MIC and decreased expression of both pslA and pelA genes in all tested isolates. Thus, our results show that chloroxylenol can not only inhibit bacterial growth but also act as an antibiofilm agent by targeting some important genetic trajectories associated with biofilm formation in MDR P. aeruginosa (Omran et al., 2026).

Conclusion

This study showed that the biofilm-associated genes pslA and pelA were prevalent among drug resistant *Pseudomonas aeruginosa* isolates obtained from respiratory tract infections in Hilla Teaching Hospital, Babylon, Iraq. Emergence of hyper-

resistance of clinical strains to conventional antibiotics, including fluoroquinolones, ceftazidime and carbapenems, underlines the constraints on effective therapy, given their widespread use. The strong correlation between positive pslA and pelA genes with susceptibility to few of the key anti-microbial agents indicate that biofilm formation is a key determinant for enhancing survival, persistence as well as multidrug resistant potential in bacteria.

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