

Research Article

Clinical Distribution and Resistance Profile of *Pseudomonas aeruginosa* Isolated from Patients in Al-Diwaniyah Province

Alaa Hassan Jabbar¹, Masar Riyadh Rashid Al-Mousawi¹, Ali Jalil Ali Alyassery¹

¹Department of Microbiology, College of Medicine, University of Kerbala, Kerbala, Iraq

Article information:

Received: 07-07-2025

Accepted: 10-08-2025

Correspondence: Alaa Hassan Jabbar

Email: alaa.hassan@uokerbala.edu.iq

ORCID: <https://orcid.org/0009-0008-8199-1323>

1323

<https://doi.org/10.70863/karbalajm.v18i2.4922>

Abstract

Background: *Pseudomonas aeruginosa* is a highly opportunistic pathogen known for its resistance to multiple antibiotics, posing significant challenges in clinical settings, particularly in nosocomial infections. Its resistance patterns will vary geographically, necessitating localized studies to guide effective treatment strategies. The study aimed to identify the distribution and resistance profile of *P. aeruginosa* in Al-Diwaniyah Province, Iraq.

Methods: A cross-sectional survey was conducted in two hospitals from November 2024 to May 2025. Clinical specimens (burn exudate, wound swabs, and urine) were collected and analyzed using culture, biochemical tests, and the VITEK-2 system. Antimicrobial susceptibility testing was performed via disk diffusion and MIC determination. Statistical analysis was performed using SPSS.

Results: From 240 patients included in this study, there were only 43 isolates of *P. aeruginosa*. The high infection rates occurred in males (62.8%) aged 17–32 years. Burn isolates exhibited the highest resistance, with 72.4% extensively drug-resistant (XDR) strains, while urinary tract infections (UTIs) were predominantly multidrug-resistant (MDR; 66.7%). Carbapenem resistance was alarmingly high (imipenem: 93.1% in burns and meropenem: 86.2%). UTIs were strongly associated with prior antibiotic use (100%), whereas burn and wound infections often occurred without antibiotic exposure (62% and 60%, respectively).

Conclusions: *P. aeruginosa* in Al-Diwaniyah Province shows significant resistance, particularly in burns and among young adults. The findings underscore the need for stringent infection control measures, antibiotic stewardship, and alternative therapies to combat rising resistance. Future research should explore novel treatments like phage therapy to improve patient outcomes.

Keywords: *Pseudomonas aeruginosa*, antibiotic resistance, multidrug-resistant, extensively drug-resistant, burn infections, urinary tract infections.

Introduction

Pseudomonas aeruginosa is a highly opportunistic and versatile pathogen that readily acquires resistance to many antibiotics, presenting as an acute clinical dilemma with a nosocomial context. Furthermore, critically ill patients are particularly at risk of this phenomenon, and resistance patterns of *P. aeruginosa* are geographically disparate and vary between institutions; hence, local analyses should be conducted for the purpose of applying effective strategies [1-2].

The bacterium is prevalent across various hospital departments, with Intensive Care Units (ICUs) being particularly high-risk due to immunocompromised patients and frequent use of invasive devices [3]. Studies report *P. aeruginosa* prevalence rates as high as 39.8% in ICUs. Respiratory wards also

show a high burden, with the pathogen isolated in approximately 59.6% of ventilator-associated pneumonia (VAP) cases [4]. Surgical wards and emergency departments frequently culture *P. aeruginosa* from wound swabs and surgical site infections (SSIs), while neurosurgery and critical care departments report prevalence rates ranging from 11.69% to 14.30% [1-2, 5].

P. aeruginosa causes diverse infections, including respiratory tract infections (RTIs), particularly in cystic fibrosis patients and those on mechanical ventilation. It is also a leading cause of bloodstream infections (BSIs), often with high mortality in immunocompromised individuals. The bacterium's biofilm-forming ability contributes to wound infections, SSIs, and UTIs, especially in catheterized patients. Additionally, it poses a severe threat in burn wound infections [6-8].

Antibiotic resistance in *P. aeruginosa* is a major concern, with high resistance rates to carbapenems (imipenem 34.6% and meropenem 35%) and fluoroquinolones (ciprofloxacin and levofloxacin exceeding 50% resistance in some regions) [9]. Aminoglycosides like amikacin and gentamicin show resistance rates between 37% and 48%, while colistin resistance exceeds 90% in certain studies. Multidrug-resistant (MDR) strains, resistant to three or more antibiotic classes, are rising, with some studies reporting rates up to 43.43% [1-2, 10-11].

P. aeruginosa with MDR is defined as non-susceptibility to at least one agent in three or more antimicrobial categories relevant to this bacterium. This means the strain resists multiple classes of antibiotics but not all [12]. XDR *P. aeruginosa* refers to strains resistant to at least one agent in all but two or fewer antimicrobial categories. In practical terms, XDR strains are resistant to almost all available antibiotic categories but remain susceptible to one or two classes. This reflects a wider and more severe resistance profile than MDR strains [12-13]. Resistance mechanisms in *P. aeruginosa* include biofilm formation, efflux pumps, production of inactivating enzymes (e.g., beta-lactamases), and horizontal gene transfer, which facilitate the spread of resistance traits [6-7, 14]. Key risk factors for infection and resistance development include prior broad-spectrum antibiotic use (especially fluoroquinolones and carbapenems), immunocompromised status, invasive medical devices, and prolonged hospitalization in high-risk areas like ICUs and surgical wards [15-17]. Molecular studies confirmed the presence of multiple resistance genes, including ESBLs and carbapenemase genes (blaOXA variants), but no metallo-beta-lactamase (MBL) genes such as IMP or VIM were detected in some isolates, suggesting alternative resistance mechanisms [18].

Based on the influence of global antibiotic policies, China reports high carbapenem and fluoroquinolone resistance, while Italy shows reduced imipenem and gentamicin resistance but increased resistance to cefepime and ceftazidime. Saudi Arabia faces high piperacillin/tazobactam and colistin resistance, emphasizing the need for strict antibiotic stewardship. In Nigeria, ceftriaxone and ciprofloxacin resistance remain problematic, reflecting challenges in low-resource settings [10-19].

In Al-Diwaniyah Province in Iraq, data on clinical distribution and resistance patterns of *P. aeruginosa* remains limited. This study aims to assess its prevalence across hospital departments and

evaluate antibiotic resistance trends. By identifying high-risk areas and resistance profiles, the findings will inform optimized treatment protocols and enhance infection control measures, ultimately improving patient outcomes in local healthcare settings.

Materials and Methods

Study Design and Population

A cross-sectional survey was conducted in two large hospitals (Afak Hospital and the Specialized Center for Burns in Diwaniyah) in Al-Diwaniyah Province between November 2024 and May 2025. A total of 240 patients were involved in this study.

Inclusion criteria: All patients with urinary tract infection, burn, and wound infection were diagnosed on the basis of clinical symptoms and other investigations.

Exclusion criteria: The patients who have co-infection and those who are taking medications were excluded.

Sample Collection

Samples from involved patients were collected to include 100 urines, 100 burn swabs, and 40 wound swabs. A total of 43 isolates of *Pseudomonas aeruginosa* (27 males (62.8%) and 16 females (37.2%)) were collected from different specimens. The isolates included were 29 from burns, 5 from wounds, and 9 from urinary tract infections (UTIs). Identification of the isolates was carried out microscopically and by cultural characterization on MacConkey agar and cetrimide agar, and further identification was carried out by the VITEK-2 Compact system. The ages of the patients ranged from 1 to 70 years.

Bacterial identification

Samples were cultured on MacConkey agar, blood agar, and cetrimide agar and incubated aerobically at 37°C for 24 hours. A Gram stain test was performed on the isolates according to the protocol mentioned in the kit, and all isolates were negative for the Gram stain and pink in color. Cetrimide agar contains cetrimide (cetyltrimethylammonium bromide), a selective agent that inhibits most other bacteria by disrupting membranes, allowing resistant *P. aeruginosa* to grow selectively on it. It is supplemented with pancreatic digest of gelatin (nutrients), magnesium chloride, and potassium sulfate, which stimulate production of characteristic pigments, yellow-green fluorescent pyoverdine and blue pyocyanin. The combination of these pigments produces the distinctive green colonies that are also fluorescent under UV light, aiding easy

presumptive identification [12-13]. Bacterial isolates were identified using the VITEK-2 automated system (BioMérieux, France) with GN ID and AST N222 cards.

Antimicrobial Susceptibility Testing

The minimum inhibitory concentration (MIC) was further determined using the VITEK-2 automated system (BioMérieux, France) with AST N222 cards

Ethical approval

The study protocol was submitted to the relevant ethical committee by the health directorate in Al-Diwaniyah and approved by document No. 41 on 25 November 2024. Additionally, verbal approval was obtained from each participant before collecting samples, and health measures and safety protocols were followed during sample collection.

Statistical Analysis

Data were analyzed using SPSS version 26 (IBM, USA). Descriptive statistics were used to summarize demographic and clinical data. Chi-square tests were employed to compare categorical variables, with a p -value < 0.05 considered statistically significant.

Results

According to the data presented in Table 1, *P. aeruginosa* infection rates varied significantly by age and sex. The most affected age group was 17-32 years, with a significantly higher infection rate of 62.8% ($p = 0.0001$). Similarly, males exhibited a significantly higher infection rate of 62.8% ($p = 0.0120$) compared to females.

Table 2 presents a detailed analysis of MDR and XDR *P. aeruginosa* isolates from UTIs, burns, and wounds and age groups, which revealed statistically significant variations ($p = 0.0001$). In UTIs, MDR isolates were predominantly found in the 17-32 year age group (66.6%), with lower prevalence in the 1-16 and 33-48 year categories (16.6% each), while no cases were detected in patients aged 49 years or older. Conversely, XDR isolates in UTIs were most common in younger patients (66.7% in 1-16-year-olds and 33.3% in 17-32-year-olds), with no occurrences beyond age 32. For burn infections, MDR resistance was highest in the 17-32 group (62.5%), minimal in the 1-16 group (12.5%), and absent in the 33-48 group. In contrast, XDR isolates in burns showed the highest prevalence in the 17-32 group (47.6%), followed by equal proportions in the 33-48 and 49-64 year groups (23.8% each), with only 4.8% in the youngest group. Wound infections exhibited the most distinct pattern, with MDR isolates exclusively present in the

17-32 year age group (100%), while XDR isolates were evenly distributed across the 1-16, 17-32, and 33-48 year groups (33.3% each) and completely absent in older patients. These findings demonstrate clear age- and infection-specific trends in antimicrobial resistance profiles.

Table 3 presents the antibiotic susceptibility patterns of *P. aeruginosa* isolates from UTIs, burns, and wounds, with all comparisons showing statistically significant differences ($p < 0.05$). The data reveal distinct resistance profiles across infection sources. For imipenem, burn isolates exhibited the highest resistance rate (93.1%), while UTI isolates showed the greatest susceptibility (44.9%). A similar trend was observed for meropenem, where burn isolates had the highest resistance (86.2%), whereas UTI isolates displayed both the highest intermediate resistance (unique to this antibiotic) and the highest susceptibility. Fluoroquinolones (levofloxacin, ciprofloxacin, ofloxacin, norfloxacin, and nalidixic acid) followed a comparable pattern, with burn isolates demonstrating the highest resistance (62.1%) and UTI isolates the greatest susceptibility (77.8%). Notably, all *P. aeruginosa* isolates were resistant to nalidixic acid, with the highest proportion observed in burn infections (67.4%).

Table 4 evaluates the rate of *P. aeruginosa* infections in relation to prior antibiotic use for UTIs, burns, and wounds. In UTIs infections, all *P. aeruginosa* (100%) are linked to antibiotic uptake, whereas no infections are found in non-antibiotic uptake cases. In burns, the rate of infection with *P. aeruginosa* was 62% of patients who are not on antibiotics, whereas 38% are associated with those who take antibiotics, with a significant difference ($P = 0.0164$). Similarly, in wounds, the rate of infection with *P. aeruginosa* was 60% of patients who have not taken antibiotics, whereas 40% are associated with those who have taken antibiotics, with a significant difference ($P = 0.0455$).

Table 5 examines the relationship between infection recurrence and prior antibiotic use across different sample types, revealing significant associations in all cases. For UTIs, recurrence was strongly linked to prior antibiotic use (88.9%), while non-recurrent infections occurred only without prior antibiotic use (11.1%), with a highly significant statistical association ($p = 0.001$). In contrast, burn infections exhibited only non-recurrent cases, with a higher proportion occurring without antibiotic uptake (58.6%) compared to those with prior antibiotic exposure (37.9%).

The difference that was statistically significant ($p = 0.0312$).

Similarly, wound infections also showed exclusively non-recurrent cases, where infections without prior antibiotic use were more frequent (60%) than those with prior antibiotic use (40%), which demonstrates a significant association ($p = 0.0455$).

Discussion

P. aeruginosa is a dangerous opportunistic pathogen, particularly in immunocompromised individuals, due to its ability to colonize epithelial sur-

faces, impair host defenses, and cause severe systemic infections, leading to high morbidity and mortality rates [19].

MDR *P. aeruginosa* is especially problematic in patients with recent antibiotic use, prolonged hospitalization, or those who are immunocompromised [19].

The findings of this study demonstrate a significant association between *P. aeruginosa* infection and demographic factors, particularly age and sex. Our results showed the highest infection rate in the 17-32-year-old age group, matching with results reported in other studies [22].

Table 1: *Pseudomonas aeruginosa* infection according to sexes and ages of patients

<i>P. aeruginosa</i> appearance No. (%)					
Age group (year)				Total No. (%)	p-value
1-16	17-32	33-48	49->64		
6 (13.9%)	27 (62.8%)	6 (13.9%)	4 (9.3%)	43 (100%)	0.0001*
Sex				Total No.	
Male		Female			
27(62.8%)		16 (37.2%)		43(100%)	0.0120*

*Significant difference at the 0.05 level by chi-square test

Table 2: Antibiotic resistance for *P. aeruginosa* in patients according to age groups

Infection types	Antibiotic resistance	Age category (year) No. (%)				Total No.	p-value
		1-16	17-32	33-48	49->64		
UTI (n=9)	MDR	1 (16.6%)	4 (66.6%)	1 (16.6%)	0 (0%)	6	0.0001*
	XDR	2 (66.7%)	1 (33.3%)	0 (0%)	0 (0%)	3	0.0001*
Burn (n=29)	MDR	1 (12.5%)	5 (62.5%)	0 (0%)	2 (25%)	8	0.0001*
	XDR	1 (4.8%)	10 (47.6%)	5 (23.8%)	5 (23.8%)	21	0.0001*
Wound (n=5)	MDR	0 (0%)	2 (100%)	0 (0%)	0 (0%)	2	0.0001*
	XDR	1 (33.3%)	1 (33.3%)	1 (33.3%)	0 (0%)	3	0.0001*

*Significant difference at the 0.05 level by chi-square test

MDR: multidrug resistant, XDR: extensively drug-resistant

Table 3: Antibiotic susceptibility for *P. aeruginosa* in patient according to type of infection

Antibiotic	R/I/S	Site infection No. (%)			Total No.	p-value ($p \leq 0.05$)
		UTIs	Burn	Wound		
Imipenem	R	5 (55.5%)	27(93.1%)*	4 (80%)	36 (83.7%)	0.0074*
	S	4 (44.9%)*	2 (6.9%)	1(20%)	7 (16.3%)	0.0001*
Total No.		9	29	5	43	
Meropenem	R	3 (33.3%)	25 (86.2%)*	4 (80%)	32 (74.4%)	0.0001*
	I	2 (22.2%)*	2 (6.8%)	0 (0%)	4 (9.3%)	0.0001*
	S	4 (44.4%)*	2 (6.8%)	1 (20%)	7 (16.3%)	0.0001*
Total No.		9	29	5	43	
Levofloxacin	R	2 (22.2%)	18 (62.1%)*	3 (60%)	23 (53.4%)	0.0001*
	S	7 (77.8%)*	11 (37.9%)	2 (40%)	20 (46.5%)	0.0001*
Total No.		9	29	5	43	
Ciprofloxacin	R	2 (22.2%)	18 (62.1%)*	3 (60%)	23 (53.4%)	0.0001*
	S	7 (77.8%)	11 (37.9%)	2 (40%)	20 (46.5%)	0.0001*
Total No.		9	29	5	43	
Ofloxacin	R	2 (22.2%)	18 (62.1%)*	3 (60%)	23 (53.4%)	0.0001*
	S	7 (77.8%)*	11 (37.9%)	2 (40%)	20 (46.5%)	0.0001*
Total No.		9	29	5	43	
Nalidixic acid	R	9 (20.9%)	29 (67.4%)*	5 (11.6%)	43 (100%)	0.0001*
Norfloxacin	R	2 (22.2%)	18 (62.1%)	3 (60%)	23 (53.4%)	0.0001*
	S	7 (77.8%)	11 (37.9%)	2 (40%)	20 (46.5%)	0.0001*
Total No.		9	29	5	43	

*Significant difference at the 0.05 level by chi-square test

S: sensitive, R: resistance, I: intermediate

Table 4: Percentage of *P. aeruginosa* isolation according to prior antibiotic use.

Type of infection	<i>P. aeruginosa</i> infection rate		p-value
	Not receiving antibiotics	Receiving antibiotics	
UTI (n=9)	0 (0%)	9 (100%)	0.0001*
Burn (n=29)	18 (62 %)	11 (38%)	0.0164*
Wound (n=5)	3 (60%)	2 (40%)	0.0455*

*Significant difference at the 0.05 level by chi-square test

Table 5: Linkage of the recurrence of infection to prior antibiotic use in each sample type

Site infection	Recurrent infection state	Antibiotic uptake state		p-value
		Non-antibiotic uptake cases	Antibiotic uptake cases	
UTI (n=9)	Non-recurrent	0 (0%)	1 (11.1%)	0.0001*
	Recurrent	8 (88.9%)	0 (0%)	0.0001*
	p-value	0.0001*	0.0001*	
Burn (n=29)	Non-recurrent	17 (58.6%)	12 (41.4%)	0.0312*
	Recurrent	0 (0%)	0 (0%)	-----
	p-value	0.0001*	0.0001*	
Wound (n=5)	Non-recurrent	3 (60%)	2 (40%)	0.0455*
	Recurrent	0 (0%)	0 (0%)	-----
	p-value	0.0001*	0.0001*	

*Significant difference at the 0.05 level by chi-square test

In Baghdad city in Iraq, different age distributions were found, with 14.7% in children (0-14 years), 29.9% in young adults (15-24 years), and 55.4% in adults (≥ 25 years), while Hilal (2023) reported higher incidence rates among older age groups, particularly 41-50 years [23]. Also, hematological studies identified a median age of 58.5 years for *P. aeruginosa* bloodstream infections, with neutropenia as a key risk factor [22]. These discrepancies may reflect population-specific differences in exposure risks, immunity, or healthcare access. The predominance of infections among young adults in our study could be attributed to occupational exposures, hospital contact, or lifestyle factors [25].

Regarding sex distribution, our finding of higher infection rates in males (62.8%) aligns with several studies [20, 26]. Umar et al. (2020) reported bacterial infections in 52.7% males and 47.3% females [26]. An Ethiopian study also found higher prevalence in males (21.4%) than females (15.8%) [27]. However, contrasting results exist an equal prevalence between sexes. The observed male predominance in most studies may be linked to greater physical injury rates or occupational exposures [29].

The present findings demonstrate significant variation in resistance profiles depending on the clinical source of infection, with burn wounds showing the highest proportion of XDR strains (72.4%), followed by wound infections (60% XDR). In contrast, UTIs were predominantly associated with MDR strains (66.7%), though a substantial proportion (33.3%) exhibited XDR resistance. This has corroborated the findings of recent research conducted in Nigeria, which found that wound swabs

had the highest prevalence (7.8 %), followed by ear swabs (3.4 %) and urine samples (1.4%) [20]. This suggests that wound infections play a primary role in the transmission and maintenance of *P. aeruginosa* infection in the hospital setting. Also, Oliveira et al. (2017) demonstrated that chronic wounds and burns provide an ideal environment for biofilm formation, facilitating the development of resistance due to prolonged antibiotic exposure and impaired immune responses [30]. The predominance of MDR strains in UTIs may reflect different selection pressures in the urinary tract, where antibiotic use patterns and anatomical factors could contribute to resistance evolution [31].

The current study found significant age-related trends in *P. aeruginosa* antibiotic resistance, with MDR strains being most common in younger patients (17–32 years) across UTIs, burns, and wounds. In contrast, XDR strains showed wider age distribution, particularly in burn infections. The higher MDR prevalence in younger populations may reflect distinct risk factors like immune responses or antibiotic exposure, while XDR persistence in older burn patients suggests chronic wound environments contribute to resistance development [32].

P. aeruginosa isolates exhibited varying antibiotic susceptibility patterns based on infection type. Burn wound isolates showed alarmingly high resistance, particularly to carbapenems (93.1% to imipenem and 86.2% to meropenem) with a 60% meropenem resistance. This finding was in agreement with other studies [33-35], in which resistance rates of meropenem were found to be 60%,

60%, and 87%, respectively. This suggests declining carbapenem efficacy, likely due to frequent clinical use.

In contrast, UTI isolates demonstrated better susceptibility, with higher sensitivity to fluoroquinolones (77.8% levofloxacin) and retained imipenem sensitivity (44.9%). For wound infections, resistance to ciprofloxacin and norfloxacin was notably high, while ofloxacin maintained a better sensitivity. This aligns with previous studies, which stated that ciprofloxacin had lower effectiveness, with resistance rates reaching up to 80% [36-37]. However, emerging meropenem resistance (22.2%) in UTIs warrants surveillance. Conflicting fluoroquinolone resistance rates in UTIs (63% and 25%) were reported by many studies [38-39], reflecting temporal and regional variability in resistance trends, possibly driven by evolving antibiotic pressures [3-8].

The study revealed distinct patterns in *P. aeruginosa* infections based on antibiotic receiving and infection type. For UTIs, all cases were associated with prior antibiotic use, demonstrating that antimicrobial exposure is a critical risk factor likely due to disruption of normal flora and selection of resistant strains. In contrast, burn and wound infections showed an inverse relationship, with higher infection rates among patients without prior antibiotic receiving (62% in burns, 60% in wounds). This suggests that in these cases, non-antibiotic factors, including compromised skin barriers, environmental contamination, and *P. aeruginosa*'s inherent biofilm-forming ability, play a more dominant role in infection establishment. The pathogen's capacity to colonize damaged tissue and medical devices appears to drive infections independently of antibiotic pressure in wound and burn settings [38]. Studies indicate that *P. aeruginosa* exhibits high levels of multidrug resistance, with resistance rates reaching 100% for certain antibiotics like ampicillin and varying resistance to others such as ciprofloxacin and imipenem [37-41]. Furthermore, in UTIs, *P. aeruginosa* demonstrates a remarkable ability to develop tolerance under specific conditions, which can enhance its resistance up to 6000-fold, complicating treatment efforts [42]. In burn wards, a positive correlation exists between antibiotic usage and resistance rates, suggesting that increased antibiotic application can exacerbate resistance [43].

This study reveals crucial differences in *P. aeruginosa* infection patterns based on infection type and antibiotic exposure. For UTIs, we observed a strong dependence on prior antibiotic use, with all cases and nearly all recurrences linked to antibiotic

exposure. In contrast, burn and wound infections showed inverse relationships with antibiotic use. Most cases occurred without prior antibiotic exposure (62% burns and 60% wounds), and all were non-recurrent. In accordance with these results, many results demonstrated that prior antibiotic exposure, particularly to fluoroquinolones and carbapenems, significantly increases the risk of *P. aeruginosa* UTIs and recurrence due to resistance selection [7, 44-45]. Also, Holt *et al.* (1994) showed that *P. aeruginosa* colonization in burns is primarily driven by environmental exposure and compromised skin barriers rather than antibiotic pressure [46]. They linked biofilm formation in chronic wounds to persistent *P. aeruginosa* infections, emphasizing the need for debridement over antibiotics.

Conclusions

P. aeruginosa is a major opportunistic pathogen in Al-Diwaniyah Province, with high infection rates among males and individuals aged 17–32. Burn wounds showed the highest XDR strains, while UTIs were mainly MDR. Carbapenem resistance was widespread, especially in burn cases, highlighting the need for alternative treatments. UTIs were linked to prior antibiotic use, whereas burn and wound infections often arose independently, emphasizing environmental and host factors. To combat resistance, stricter hygiene, better surveillance, and antibiotic stewardship are essential. Future research should explore novel therapies like phage therapy to improve outcomes and reduce *P. aeruginosa* infections in the region, emphasizing the urgent need for local antimicrobial stewardship programs and infection prevention interventions.

Funding: There is no funding for this research.

Conflict of interest: The authors state that there is no conflict of interest

Contribution authors

Conceptualization, M.R.R.; Methodology, A.J.A.; Formal analysis and investigation, A.H.J.; Writing, A.H.J.; Resource, M.R.R.; Supervision, A.J.A. and M.R.R.

References

1. Lyu J, Chen H, Bao J, Liu S, Chen Y, Cui X, *et al.* Clinical distribution and drug resistance of *Pseudomonas aeruginosa* in Guangzhou, China from 2017 to 2021. *Journal of Clinical Medicine*. 2023 ;12(3):1189. DOI:10.3390/jcm12031189.
2. Saeed M, Rasheed F, Afzal RK, Hussain S, Riaz S, Ahmad A. *Pseudomonas aeruginosa*: Evaluation of pathogen burden and drug-resistance trends in a tertiary care hospital. *J Coll Physicians Surg Pak*. 2018;28(4):279-83. <https://pmc.ncbi.nlm.nih.gov/articles/PMC6861494/>
3. Jabbar AH, Alsailawi HA, Mudhafar M, Riyadh M, Alkenany HA, Ibrahim IH, *et al.* Antibiotic resistance of

- non-lactose-fermenting Gram-negative bacteria in urinary tract infection. *Asian Journal of Green Chemistry*. 2025; 9(4):404-414.
4. Nimer NA. Nosocomial infection and antibiotic-resistant threat in the Middle East. *Infection and Drug Resistance*. 2022; 1:631-9. DOI:10.2147/IDR.S351755.
 5. Li XY, Liu XG, Dong ZL, Chai LT, Liu YJ, Zhao J. The distribution, drug susceptibility, and dynamic trends of *Pseudomonas aeruginosa* infection in a tertiary hospital in China during 2016–2022. *Infection and Drug Resistance*. 2023; 31:3525-33. DOI:10.2147/IDR.S417819.
 6. Al-Dahmoshi H, Al-Obaidi RD, Al-Khafaji N. *Pseudomonas aeruginosa*: diseases, biofilm and antibiotic resistance. In *Pseudomonas aeruginosa*-biofilm formation, infections and treatments 2020. IntechOpen.
 7. Pachori P, Gothwal R, Gandhi P. Emergence of antibiotic resistance *Pseudomonas aeruginosa* in intensive care unit; a critical review. *Genes & diseases*. 2019;6(2):109-19. DOI:10.1016/j.gendis.2019.03.001.
 8. Masar AM, Hanoon A, Jasim A, Alattab A, Musafar K, Abdulzahraa Z. Bacterial profile and antibiotic susceptibility patterns of urinary tract infection among children in Karbala Teaching Hospital. *Journal of Tropical Life Science*. 2023;13(1):131-6. <https://jtrolis.ub.ac.id/index.php/jtrolis/article/download/2551/624>).
 9. ANJUM S. Characterization of β-lactamase in clinical isolates of *Pseudomonas aeruginosa*. Doctoral dissertation. Jain University. 2022. https://arkajainuniversity.ac.in/naac/Criteria%201/1.3.4/1_3_4_DOCUMENTS/BIOTECH/AJU190645.pdf.
 10. Serretiello E, Manente R, Dell'Annunziata F, Folliero V, Iervolino D, Casolaro V, et al. Antimicrobial resistance in *Pseudomonas aeruginosa* before and during the COVID-19 pandemic. *Microorganisms*. 2023;11(8):1918. DOI:10.3390/microorganisms11081918.
 11. Jamatia AR, Roy DE, Shil RU, Prabhakar PK. Bacteriological profile and antimicrobial resistance patterns isolates in pus samples at Agartala Government Medical College. *Asian J Pharm Clin Res*. 2017;10(1):335-7. DOI:10.22159/ajpcr.2017.v10i1.17462.
 12. Magiorakos AP, Srinivasan A, Carey RB, Carmeli Y, Falagas ME, Giske CG, et al. Multidrug-resistant, extensively drug-resistant and pandrug-resistant bacteria: an international expert proposal for interim standard definitions for acquired resistance. *Clinical Microbiology and Infection*. 2012;18 (3): 268-281. DOI:10.1111/j.1469-0691.2011.03570.x.
 13. Dahal P, Shrestha M, Maharjan M, Parajuli R. Multi-drug-resistant *Pseudomonas aeruginosa* and its coexistence with β-lactamases at a tertiary care hospital in a low-resource setting: a cross-sectional study with an association of risk factors. *Therapeutic advances in infectious disease*. 2025; 12, 20499361251345920. <https://doi.org/10.1177/20499361251345920>. DOI 10.1177/20499361251345920.
 14. Schwartz B, Klamer K, Zimmerman J, Kale-Pradhan PB, Bhargava A. Multidrug resistant *Pseudomonas aeruginosa* in clinical settings: a review of resistance mechanisms and treatment strategies. *Pathogens*. 2024;13(11):975. DOI:10.3390/pathogens13110975.
 15. Kang D, Zhang L, Kirienco NV. High-throughput approaches for the identification of *Pseudomonas aeruginosa* antivirulents. *MBio*. 2021;12(3):10-128. DOI:10.1128/mBio.02240-20.
 16. Bassetti M, Monti G, Henriksen AS, Longshaw C. Predicting early appropriate therapy for patients infected by carbapenem-resistant Gram-negative pathogens in intensive care units in Italy. *Antimicrobial Resistance & Infection Control*. 2024;13(1):91.
 17. Yang J, Xu JF, Liang S. Antibiotic resistance in *Pseudomonas aeruginosa*: mechanisms and emerging treatment. *Critical Reviews in Microbiology*. 2024 ;18:1-9. DOI:10.1080/1040841X.2024.2429599
 18. Alkhulaifi ZM, Mohammed KA. Prevalence and molecular analysis of antibiotic resistance of *Pseudomonas aeruginosa* isolated from clinical and environmental specimens in Basra, Iraq. *Iranian Journal of Microbiology*. 2023; 15(1), 45–54. <https://doi.org/10.18502/ijm.v15i1.11917>.
 19. Okun KO, Osundi S, Dibal J, Ngbale T, Bello M, Akuhwa RT, et al. Bacterial contamination of operating theatre and other specialized care unit in a tertiary hospital in Northeastern Nigeria. *Afr J Microbiol Res*. 2012;6(13):3092-6. DOI:10.5897/AJMR12.1427.
 20. Wood SJ, Kuzel TM, Shafikhani SH. *Pseudomonas aeruginosa*: infections, animal modeling, and therapeutics. *Cells*. 2023;12(1):199. DOI:10.3390/cells12010199.
 21. Yang AF, Huang V, Samaroo-Campbell J, Augenbraun M. Multi-drug resistant *Pseudomonas aeruginosa*: a 2019-2020 single center retrospective case control study. *Infection Prevention in Practice*. 2023;5(3),100296. <https://doi.org/10.1016/j.infpip.2023.100296>.
 22. Jibril AH, Bawa H, Mohammed K, Nuhu A, Uhuami AO. High risk of *Pseudomonas aeruginosa* infection in patients attending public hospitals in Sokoto, Nigeria. *The Microbe*. 2025;6:100271.
 23. Hilal HA. Molecular and epidemiological study of *pseudomonas aeruginosa* isolated from burn patients in Baghdad city-Iraq. *Cardiometry*. 2023; (29):116-21.
 24. Kessel J, Bug G, Steffen B, Brunnberg U, Vehreschild MJ, Weber S, et al., Risk factors and outcome of *Pseudomonas aeruginosa* bloodstream infections (PABSI) in hematological patients: a single center retrospective cohort study. *Infection*. 2024 .19:1-0. DOI 10.1007/s15010-024-02453-0.
 25. Awoke A, Gudeshe G, Chane K, Siyum Y, Tilahun W, Gebremedhin H. Traditionally used phytomedicines and their associated threats in Bitu district, southwestern Ethiopia. *Journal of Ethnobiology and Ethnomedicine*. 2025;21(1):8.
 26. Umar AI, Garba I, Ganau AM, Bunza NM, Ashcroft OF, Habeeb YD. Vancomycin resistance among clinical isolates of *Staphylococcus aureus* obtained from selected hospitals in Sokoto Metropolis. *UMYU Journal of Microbiology Research*. 2020;5(2):111-6.
 27. Asamenew T, Worku S, Motbainor H, Mekonnen D, Deribe A. Antimicrobial resistance profile of *Pseudomonas aeruginosa* from different clinical samples in Debre Tabor comprehensive specialized hospital, Northwest Ethiopia. *Ethiopian Journal of Health Sciences*. 2023 ;33(3). DOI:10.4314/ejhs.v33i3.5.
 28. Abdulmutallib S, Muntari B, Bunza NM, Ganau AM. Antibigram profile of *Pseudomonas aeruginosa* isolated from wounds of patients attending some selected hospitals in Sokoto metropolis, Nigeria. *GSB Biological and Pharmaceutical Sciences*. 2019;9(2):32-43.
 29. Stracciolini A, Casciano R, Levey Friedman H, Stein CJ, Meehan III WP, Micheli LJ. Pediatric sports injuries: a

- comparison of males versus females. The American Journal of Sports Medicine. 2014;42(4):965-72. DOI:10.1177/0363546513519770.
30. Oliveira A, de Souza CR, de Araújo RS, Vasconcelos MA, da Silva VL, Diniz CG, et al., Prevalence of antimicrobial resistance in *Pseudomonas aeruginosa* and characterization of metallo- β -lactamases genes in isolates from hospitals in Recife, Pernambuco, Brazil. Revista da Sociedade Brasileira de Medicina Tropical. 2017;50(2):179-83. DOI:10.1590/0037-8682-0036-2017.
 31. Whelan S, Lucey B, Finn K. Uropathogenic *Escherichia coli* (UPEC)-associated urinary tract infections: the molecular basis for challenges to effective treatment. Microorganisms. 2023;11(9):2169. DOI:10.3390/microorganisms11092169.
 32. Kothari A, Kherdekar R, Mago V, Uniyal M, Mamgain G, Kalia RB, et al. Age of antibiotic resistance in MDR/XDR clinical pathogen of *Pseudomonas aeruginosa*. Pharmaceuticals. 2023;16(9):1230. DOI:10.3390/ph16091230.
 33. Santerre Henriksen A, Jeannot K, Oliver A, Perry JD, Pletz MW, Stefani S, et al., ARTEMIS Study Investigators. In vitro activity of cefiderocol against European *Pseudomonas aeruginosa* and *Acinetobacter* spp., including isolates resistant to meropenem and recent β -lactam/ β -lactamase inhibitor combinations. Microbiology Spectrum. 2024;12(4):e03836-23. DOI:10.1128/spectrum.03836-23.
 34. Rodríguez-Martínez JM, Poirel L, Nordmann P. Molecular epidemiology and mechanisms of carbapenem resistance in *Pseudomonas aeruginosa*. Antimicrobial Agents and Chemotherapy. 2009;53(11):4783-8. DOI:10.1128/AAC.00574-09.
 35. Khan F, Khan A, Kazmi SU. Prevalence and susceptibility pattern of multi drug resistant clinical isolates of *Pseudomonas aeruginosa* in Karachi. Pakistan Journal of Medical Sciences. 2014;30(5):951. DOI:10.12669/pjms.305.5687.
 36. Fadhil MM, Hadi OM. Phylogeny of antibiotic resistance genes of *Escherichia coli* B2 isolated from urinary tract infection patients. Hilla University College Journal for Medical Science. 2024;2(3):40-7.
 37. Mohamed A, Abdelhamid F. Antibiotic susceptibility of *Pseudomonas aeruginosa* isolated from different clinical sources. Zagazig Journal of Pharmaceutical Sciences. 2020;28(2):10-7.
 38. Ogbolu DO, Ogunledun A, Adebisi OE, Daini OA, Alli A. Antibiotic susceptibility patterns of *Pseudomonas aeruginosa* to available antipseudomonal drugs in Ibadan, Nigeria. African Journal of Medicine and Medical Sciences. 2008; 37(4), 339–344.
 39. Tuon FF, Dantas LR, Suss PH, Tasca Ribeiro VS. Pathogenesis of the *Pseudomonas aeruginosa* biofilm: a review. Pathogens (Basel, Switzerland). 2022; 11(3), 300. <https://doi.org/10.3390/pathogens11030300>.
 40. Frigui S, Messadi AA, Thabet L. Colonisation Et infection a *Pseudomonas aeruginosa* Dans Un Service De Réanimation Des Brûlés: Étude Sur 8 Ans. Annals of burns and fire disasters. 2020; 33(4), 304–311. DOI:10.3205/0002855.
 41. Narten M, Rosin N, Schobert M, Tielen P. Susceptibility of *Pseudomonas aeruginosa* urinary tract isolates and influence of urinary tract conditions on antibiotic tolerance. Current Microbiology. 2012;64:7-16. DOI:10.1007/s00284-011-0026-y.
 42. Dou Y, Zhang X, Zhang Q, Shi Y. Analysis of the drug-resistance of *Pseudomonas aeruginosa* and the use of antibiotics in burn wards. Zhonghua Shao Shang za zhi= Zhonghua Shaoshang Zazhi. Chinese Journal of Burns. 2011;27(2):109-13. DOI: 10.1177/0300060517703573.
 43. Pang Z, Raudonis R, Glick BR, Lin TJ, Cheng Z. Antibiotic resistance in *Pseudomonas aeruginosa*: mechanisms and alternative therapeutic strategies. Biotechnology Advances. 2019;37(1):177-92. DOI:10.1016/j.biotechadv.2018.11.013.
 44. Bidell MR, Opraseuth MP, Yoon M, Mohr J, Lodise TP. Effect of prior receipt of antibiotics on the pathogen distribution and antibiotic resistance profile of key Gram-negative pathogens among patients with hospital-onset urinary tract infections. BMC infectious Diseases. 2017;17:1-7. DOI:10.1186/s12879-017-2541-4.
 45. Qin S, Xiao W, Zhou C, Pu Q, Deng X, Lan L, et al. *Pseudomonas aeruginosa*: pathogenesis, virulence factors, antibiotic resistance, interaction with host, technology advances and emerging therapeutics. Signal transduction and targeted therapy. 2022;25;7(1):199. DOI:10.1038/s41392-022-01056-1.
 46. Holt JG, Krieg NR, Sneath HA, Stanley JT, Williams ST. Bergeys Manual of Determinative. 1994.
 47. MacFaddin JF. Biochemical tests for identification of medical bacteria. 3rd ed. Lippincott Williams and Wilkins, USA. 2000.