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Original article

Molecular Detection of the Mexa Efflux Pump Gene in *Pseudomonas aeruginosa* Isolated from Diyala Province

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Abstract
This study was conducted at Baquba Teaching Hospital and Consulting Clinic from June 2024 to August 2024, due to the importance of Pseudomonas aeruginosa bacteria that are and cause various clinical infections The study included collecting 88 samples of burn infections, wounds, ear infections, and urinary tract infections from 54.54% males (48) and 45.45% females (40). Their ages ranged from 10 to 60 years including patients hospitalized and non-hospitalized. The disease models were grown on MacConkey solid medium, blood medium, and Pseudomonas solid medium, and the phenotypic characteristics of the colonies were observed. Standard bacterial and biochemical tests were performed to diagnose the bacterial isolates, which included oxidase and catalase tests. 10 bacterial isolates were isolated from both sexes and different ages. The percentage of isolates was of males, 54.54% and of females, 45.45%. The rate of diagnosis of Pseudomonas aeruginosa isolates using the VITEK2 device was 100% and the percentage of isolates from infections was Burns, ears, urinary tract, and wounds 35%, 10%, 20%,23%, respectively The isolates underwent identification and antimicrobial susceptibility testing employing VITEK® 2 compact system Efflux pumps have a significant function in mediating antibiotic resistance in P. aeruginosa isolates The correlation between phenotypic and genotypic results, particularly the prevalence of MexA gene, underscores the importance of efflux pumps in MDR in P.aeruginosa strains from this clinical setting P. aeruginosa presents a significant clinical issue owing to its multidrug-resistant (MDR) phenotypic, resulting in severe and potentially fatal infections.
Keywords. *Multi-drug resistance, Efflux pump, MexA gene, Pseudomonas aeruginosa.*

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Introduction

Pseudomonas aeruginosa is a type of opportunistic pathogen that induces many diseases and is frequently implicated in nosocomial infections [1]. *P. aeruginosa* was a Gram-negative bacterium commonly found in the environment. P. aeruginosa was implicated with various infections, both community- and hospital-acquired, such as pneumonia, cystic fibrosis, urinary tract infections, and burn infections. It's an aerobic, motile rod classified under the family Pseudomonadaceae [2]. *P. aeruginosa* exists in both water and soil and was a recognized pathogen responsible for infections in humans, plants, and animals. The pigment synthesis of pyoverdine, pyocyanin, and pyorubin by *P. aeruginosa* is readily observable on agar plates. Relative to other bacteria, the genome of *P. aeruginosa* has a notably large (5.5–7 Mbp) and encodes several regulatory proteins and enzymes essential for metabolic processes, growth, and efflux systems, consequently contributing to resistance to antibiotics. Owing to its extensive encoding capacity, *P. aeruginosa* exhibits enhanced stability and adaptability to many hostile conditions [3].

P. aeruginosa belongs to the multidrug-resistant (MDR) opportunistic pathogens that induce acute or chronic infections in immunocompromised patients suffering from chronic obstructive pulmonary disease (COPD), cystic fibrosis, cancer, trauma, burns, septicemia, and ventilator-associated pneumonia (VAP), including cases associated with COVID-19 [4]. *P. aeruginosa* exhibits a wide variety of pathogenic characteristics and changes its genetic makeup and physical characteristics to avoid being killed by drugs and the body's immune system involving resource competition, anaerobic conditions, elevated antibiotic concentrations, and immunological responses including neutrophil assaults are only a few of the substantial stresses that *P. aeruginosa* faces in the chronic infection setting within the CFlung [5].The capacity to acquire MDR represents a significant worry, since it endures a wide range of antibiotic classes, including aminoglycosides (Amikacin, Gentamicin, and Tobramycin) and fluoroquinolones (Ciprofloxacin, Norfloxacin, and Ofloxacin).

P. aeruginosa isolates displaying multidrug resistance are enduring nosocomial infections, with prevalence rates from 11.5% to 24.7% and 9.0% to 11.2%, respectively, presenting considerable therapeutic challenges [6]. Nosocomial pneumonia caused by *P. aeruginosa* has worse clinical outcomes in comparison to other conventional pathogens. The incidence of MDR *P. aeruginosa* VAP has been identified to be notably high at 33%. [7]. *P. aeruginosa* can exhibit intrinsic,

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acquired, and adaptive antibiotic resistance mechanisms; occasionally, all three can be seen in a single strain. Resulting in MDR *P. aeruginosa* infections, which are directly associated with increased rates of morbidity, mortality, lengthier hospital stays, and greater costs of medical care [8]. MDR is facilitated by the efflux pump mechanism, which eliminates a variety of compounds and medicines, as well as colors, organic solvents, detergents, molecules required for intercellular communication, and metabolic products. *P. aeruginosa* has an efflux pump transporter that is part of the resistance nodulation division family. It is made up of three components: the transporter, the linker, and the outer membrane pore. It inhibits the extruded chemical from remaining in the periplasm and re-entering the cytoplasm [9].
The RND efflux pumps are the most important of the several families of efflux pumps associated with MDR in P. aeruginosa (Figure 1-1).
Antibiotic resistance relies heavily on RND pumps because of their unusual structure and function [10]. These pumps reduce intracellular drug concentrations and impart resistance by effectively expelling a wide spectrum of antimicrobial drugs via the inner membrane (IM) and outer membrane (OM). Furthermore, RND pumps are resistant to several kinds of antibiotics all at once due to their wide substrate specificity. Their relationship with clinical isolates showing high-level resistance to several medicines highlights their role in P. aeruginosa MDR. A concerning feature of P. aeruginosa is its poor antibiotic sensitivity. This is due to the interaction of many efflux pumps and antibiotic resistance genes expressed on chromosomes, including mexA, mexB, and others. Additionally, *P. aeruginosa* exhibits low outer membrane permeability. In addition to this intrinsic resistance, *P. aeruginosa* develops acquired resistance either through mutations in chromosomally encoded genes or by horizontal gene transfer of antibiotic resistance determinants. The development of multidrug resistance (MDR) in *P. aeruginosa* involves multiple genetic mechanisms. Mutations are often selected for based on antibiotic pressure, especially in chronic infections, leading to resistant strains. Furthermore, the accumulation of diverse resistance genes, often facilitated by integrons, enables the bacterium to acquire a broad arsenal of resistance determinants. Recent studies have emphasized processes associated with the development of efflux pumps and the creation of tiny colony variations, both of which may significantly influence the adaptive antibiotic responses to *P. aeruginosa*.

Methodology

Isolation and identification of bacteria

An overall of 88 clinical samples were obtained from diverse sites, including the nasal cavity, blood, urinary tract infections, and wounds, from patients in Baqubah City between June 2024 and August 2024. Following the acquisition of a solitary colony of isolated bacteria, the isolates have been determined based on phenotypic colony features, different biochemical tests such as oxidase and catalase, and confirmed by Vitek 2 Compact. Resistance of P. aeruginosa isolates to different antibiotic types from various classes was studied. The antibiogram testing was performed on 10 isolates following the guidelines of the Clinical and Laboratory Standards Institute by using the Kirby-Bauer disk diffusion technique, isolates cultured on Mueller-Hinton agar, and disks from BioMérieux in Turkey. To compare the findings with the National Committee for CLSI [11], the inhibition zones were measured in millimeters using the zone inhibition ruler. To identify the genotype, the *P. aeruginosa* isolates were tested for the presence of MexA by using PCR. An anti-allergic test for the bacterium Pseudomonas aeruginosa is performed in the following way: Kiyby-bauer Disk Diffusion test, bacterial suspension prepared from McFarland in Nacl solution, and inoculate the bacteria on the Muller-Hinton agar medium using a swab, and put antibiotic tablets ex (Piperacillin, Tazobactam, Ceftazidime, Cefepime, Meropenem, Imipenem, Gentamicin, Ciprofloxacin, Colistin, Amikacin, Azetronam) and incubate for 16-18 hours at a temperature of 35-37 °C. The growth inhibition zones around the tablets show evidence of bacterial resistance to the antibiotic.
Phenotypic detection of Efflux pumps was done by activating 10 isolates and adding different concentrations ranging from 250, 200, 100, 25, 20 micrograms/ml of the ethidium bromide formula, proportions of this formula to sterile Tryptone soy (TsA agar), and cooled to 45. Prepare a bacterial suspension from the bacterial isolates to be tested by transferring a single colony aged 24 hours on the solid Muthent agar medium using the physiological adhesive solution and compare its turbidity with the standard turbidity constant solution withdraw 5 microliters of the above suspension using a fine pipette, each separately, and drop it as a single drop on the TsA medium containing the ethidium bromide dye. The plates were left for a period at room temperature until the drops dried before turning the plates over. The plates were incubated for 24 hours at 37 degrees. Examining the dishes using ultraviolet light U.V. light at a wavelength of 320 nanometers to observe the intensity of fluorescence. The bacterial colonies that give fluorescence at the current concentration of the dye pump the flow more effectively compared to the colonies that only fluoresce at low concentrations of the dye.

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DNA extraction and PCR

The Promega DNA extraction kit was utilized to extract the genomic DNA of the 10 isolates. The presence of the MexA gene PCR amplification with a set of primer pairs, as shown in (Table 1) and the amplification program is shown in (Table 2). In order to determine the sample quality for further uses, the concentration of extracted DNA was detected using a Quantus Fluorometer.

Table 1: Primer used in the study

Primer name	Vol of nuclease-free water (µl)	Concentration (pmol/ µl)	*Primer sequence*
mexA-F	320	100	*GCAGACGGTGACCCTGAATA*
mexA-R	300	100	*GTATTGGCTACCGTCCTCCA*

Agarose gel electrophoresis was employed post-PCR amplification to verify the existence of amplification. PCR was entirely reliant on the criteria of the retrieved DNA. The agarose solution was poured into the gel tray following the sealing of both ends with cellophane tape, and the agarose was allowed to harden at the ambient temperature for 30 minutes. The comb was meticulously extracted, and the gel was positioned in the gel tray. The tray was filled with IX TAE-electrophoresis buffer until it reached 3-5 mm above the gel surface. Two microliters of loading dye were applied for every 5 µL DNA sample, and the samples were meticulously loaded into the individual wells. PCR products were immediately loaded. The PCR product, Sul, was simply put into the well. Electrical power was activated at 100 volts per milliampere for 60 minutes. DNA migrates from the cathode to the anode. The bands stained with ethidium bromide in the gel were observed with gel imaging equipment.

Table 2: PCR Program

Steps	C°	M:s	Cycle
Initial Denaturation	95	05:00	1
Denaturation	95	00:30	30
Annealing	60	00:30	
Extension	72	00:30	
Final extension	72	07:00	1
Hold	10	10:00	

Statistical Analysis

The results were reported as mean values with standard error (SD). Statistical analysis was conducted using SPSS 26 (SPSS Inc., Chicago, USA). Significance was determined at a P-value below 0.05. The Chi-square test assessed significance when comparing percentages with probabilities of 0.05 and 0.01.

Results

This study was conducted in Baquba Teaching Hospital in Diyala governorate over a continuous two-month period from June 2024 to August 2024. It was a prospective, descriptive, and investigative study. The ten clinical isolates were identified as Pseudomonas aeruginosa using routine biochemical testing. All of the isolates were grown on primary isolation and selective media. In addition to other biochemical testing, Gram staining, catalase, and oxidase tests were performed on each of the outcomes of the Gram staining.

(Table 3) shows the percentages of clinical samples included in this study and their relationship to hospitalized and non-hospitalized patients, their genders, and ages. In general, the percentage of pathological samples collected from hospitalized patients was higher than that from non-hospitalized patients. Regarding the percentages of pathological samples and their distribution by gender, they were higher among males, with the number of samples reaching compared to females, with the number of samples collected reaching Ear swabs had the among both males and females, with 10 samples, followed by urine samples, with 20 samples collected, followed by wound samples, with 23 samples, and the number was from burn samples, with 35 samples collected from both males and females.

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Table 3: percentages of clinical samples

Sample Type	Inpatients	Number of females	Number of males
Burn	35	18	17
wound	23	9	14
urine	20	7	13
ear	10	6	4
Total	88	40	48

The results of the susceptibility test obtained from 10 isolates showed different antibiograms. The summary of multiple antibiotic resistance profiles for isolate identification is shown in a diagram.

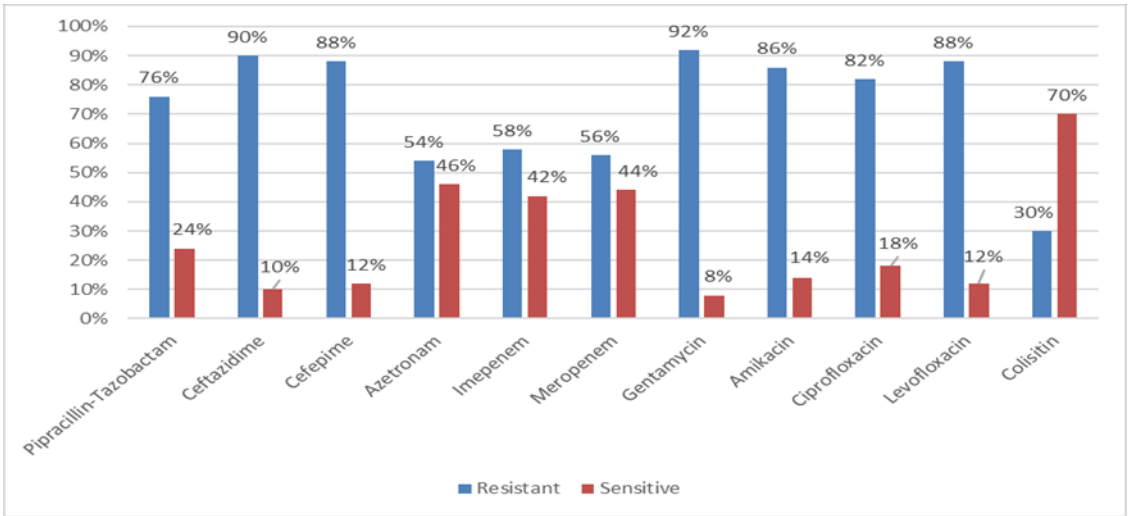

Figure 1: Antimicrobial susceptibility testing for isolated P. aeruginosa strains

The Tryptose Soy agar cartwheel method was used, as bromide dye was used to detect drug-resistant flow pumps, and this dye does not collect inside the germ cell. The bacterial isolates included in the study tested for having flow pumps, as they used a source of U.V light for the phenotypic detection of flow pumps, the percentage of isolates that have flow pumps with high efficiency was 70%, and 1020 of the isolates had flow pumps with medium efficiency in 10% of isolates did not have flow pumps, as shown in (Table 4).

Table 4: Percentages of Pseudomonas aeruginosa produced and unproductive Efflux pumps

Efficiency of flow pumps	Number of isolates produced for flow pumps
Higheffux Pumps	70 %
Inter mediat effux pumps	20 %
Low-effux Pumps	01 %

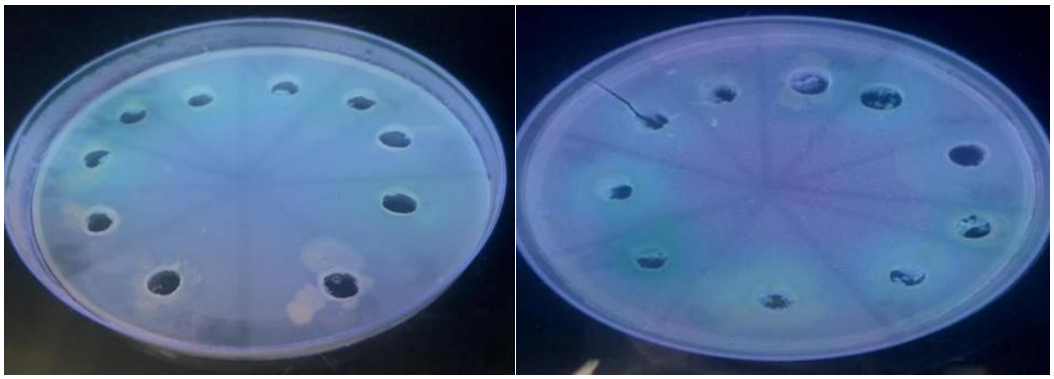

Figure 2: The Tryptose Soy agar cartwheel method applied to Pseudomonas aeruginosa cultures was swabbed on TSA plates containing incubation at 37 ◦C for 18 hours, fluorescence was detected under UV light.

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The gene Mex A are detected in 10 isolates (100%) of the isolates included in the study, as the results of the detection were that 8 isolates were carriers of the gene Mex A with a significant statistical difference and the results of the polymerase chain reaction of the Mex A gene showed that it has 605 base pairs when comparing the beams with the volumetric index DNA I adder as shown in (Figure 2).

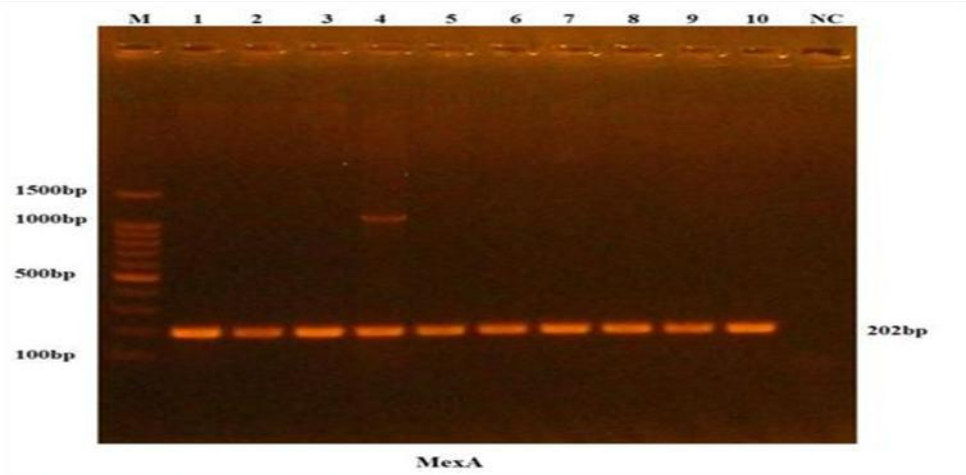

Figure 3: PCR amplification of the MexA gene from P. aeruginosa, with the amplicon size 202bp. DNA amplification products were electrophoresed in 2% agarose gel. Electrophoresis took 1.5 hours at 70 volts. Lanes 1-18 represent.

Discussion

P. aeruginosa is a vastly adaptive pathogen, acknowledged for its inherent and acquired resistance to a broad spectrum of antibiotics, rendering it a significant barrier in medical therapy. This bacterium possesses various mechanisms for resistance to antibiotics, including decreased membrane permeability, alteration of antibiotic targets, production of beta-lactamases, and overexpression of efflux pumps like MexAB-OprM, that actively expel multiple classes of antibiotics from the cell. Those pathways are enhanced in biofilm-forming populations, contributing to chronic infections and therapy failures [12]. *P. aeruginosa* represents a principal opportunistic bacterium linked to a diverse array of hospital-acquired infections, and its MDR strains become increasingly concerning in clinical environments [13]. The primary mechanism driving this resistance is the continuous expression of the MexAB-OprM efflux pump, which may eliminate a wide range of antibiotics, including β-lactams, quinolones, and macrolides. [14]. 70% of the 180 clinical isolates tested positive for MDR, whereas 5.5% tested positive for pandrug resistance (PDR). Of the bacteria tested, 88.3% had the mexA gene and 69.4% carried the mexB gene [15]. In addition, MexAB-OprM was confirmed to be functionally involved when carbonyl cyanide m-chlorophenyl hydrazone (CCCP) was used to suppress efflux activity, leading to considerable MIC decreases [15]. In Iran, overexpression of mexB was observed in 29% of MDR clinical isolates compared to a susceptible control strain, highlighting differential gene expression as a resistance mechanism [16]). Another Iranian study in pediatric patients found overexpression in MexA (55.5%), MexB (53.3%), and OprM (35.5%) among resistant isolates, with 62% exhibiting upregulation of at least one component of the efflux system [17]. In surgical ICU samples, 44.8% of isolates were meropenem-resistant, and efflux activity was implicated in 60% of these via CCCP testing [18].

Overexpression of the pump is frequently linked to mutations in regulatory genes such as mexR, nalC, and nalD, which disrupt normal repression of the efflux operon [19]. Specific point mutations, like Gly71→Glu or Ser209→Arg in NalC, have been shown to directly affect MexAB-OprM expression [19]. In a Pakistani study, clinical isolates consistently harbored mexA, mexB, and oprM, and mutant docking simulations showed stronger efflux-related drug binding, supporting enhanced antibiotic expulsion [20]. In biofilm-forming *P. aeruginosa*, MexAB-OprM is further upregulated, while OprD is downregulated, creating a dual barrier to antibiotic penetration and action [21]. The pump also contributes to bacterial virulence; knockout strains lacking MexAB-OprM demonstrated significantly reduced epithelial cell invasion and colonization capacity [22]. Given its high prevalence and regulatory complexity, localized molecular surveillance of MexAB-OprM is essential for effective infection control and treatment planning [16]. Encouragingly, efflux pump inhibitors like CCCP have been shown to restore susceptibility in MDR strains, supporting their role as

adjunctive therapies [15]. Ultimately, integrating efflux gene profiling with biofilm characterization can inform antibiotic stewardship and lead to better clinical outcomes [21].

Several studies conducted in Iraq have highlighted the significant role of the MexAB-OprM efflux pump system in multidrug-resistant (MDR) P. aeruginosa clinical isolates. A study in Al-Qadisiyah province reported that 64% of isolates exhibited efflux pump activity, with PCR results showing mexA gene presence in 93.75% and oprM in 43.75% of tested strains, confirming the widespread dissemination of the MexAB-OprM system in clinical settings [22]. In Baghdad, among 120 isolates, 27 (22.5%) were resistant to colistin and showed high resistance to other antibiotics like gentamicin and lomefloxacin, suggesting a possible contribution of efflux mechanisms, including MexAB-OprM, though molecular confirmation was not conducted [23]. Another recent study from Basra identified a high prevalence of MDR and extensively drug-resistant (XDR) P. aeruginosa (66.3%), particularly in burn wound infections, and emphasized the likely contribution of efflux systems such as MexAB-OprM in driving resistance, alongside carbapenemase genes [24,25]. Collectively, these findings underscore the clinical importance of efflux pump–mediated resistance in Iraqi healthcare settings and the need for targeted molecular surveillance.

Conclusion

All *P.aeruginosa* that carry the three genes of efflux pump MexA gene in the MexABOprM operon in our research, can lead to increase the expel of antibiotic and increase the antibiotic resistance in *P.aeruginosa*, and that is make a health problem with patients especially those with impaired immune systems, such as those with severe burns and newborn as well as those with malignancy. On another hand, detection of efflux pump genes can be used to give an idea about a possible emergence of antibiotic resistance. PCR can reduce the identification process of bacteria with antibiotic resistance to a few hours.

Conflict of interest. Nil

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