

Antibiotic Resistance of *Pseudomonas aeruginosa* in Burns and Wounds in Baghdad and Al-Samawah City

Mohammed Ali Alaboudi¹, Seger Abdulkhadim Seger Aljwaid²

^{1,2} Department of Medical Laboratories, College of Health and Medical Technology, Sawa University, Almutana, Iraq

DOI:

<https://doi.org/10.47134/mpk.v1i2.3124>

*Correspondence:

¹ Mohammed Ali Alaboudi

² Seger Abdulkhadim Seger Aljwaid

Email:

¹ mohammedali@sawauniversity.edu.iq

² saqer.abd@sawauniversity.edu.iq

Received: 17-07-2024

Accepted: 24-07-2024

Published: 31-07-2024



Copyright: © 2024 by the authors. Submitted for open access publication under the terms and conditions of the Creative Commons Attribution (BY SA) license

(<http://creativecommons.org/licenses/by/4.0/>).

Abstract: The most prevalent pathogen in nosocomial situations remains to be *Pseudomonas aeruginosa*. High levels of resistance to several antibiotic classes are displayed by this bacterium. Thus, the purpose of this work is to examine the multidrug-resistant *P. aeruginosa* bacteria that have been isolated from wound and burn infections. From burn and wound, 69 *P. aeruginosa* isolates were obtained. Antibiotic susceptibility testing was carried out using the conventional Kirby-Bauer disk-diffusion test method. The antibiotic resistance rate ranged between (20.28–85.5) for 69 isolates of *P. aeruginosa* tested. The current study revealed that *P. aeruginosa* isolates that higher level of resistance to Gentamicin, Cefepime, Ceftazidime Ticarcillin, Aztreonam, and also appear high sensitive to Amikacin, Imipenem, Meropenem and Ciprofloxacin. Therefore, in order to implement successful empirical medicines throughout hospital settings, it is imperative to carry out molecular epidemiology research and antibiotic surveillance.

Keywords: *Pseudomonas aeruginosa*, antibiotic resistance, burns, wounds

Introduction

A common pathogenic bacterium called *Pseudomonas aeruginosa* can cause serious opportunistic infections, particularly in those with impaired immune systems. This organism is highly hazardous when it spreads across healthcare institutions because it penetrates the skin of humans and enters the body, leading to nosocomial infection, especially in hospital critical care units (ICUs).

Owing to the existence of many pathways of true resistance to the majority of antibiotics, *P. aeruginosa*'s pathogenesis is complex, leading to the development of a wide range of cellular structures and extracellular compounds that are essential in enhancing pathogenicity (Al-Mayyahi, 2018). GN bacterium that resembles a rod Hospital-acquired infections are frequently caused by *P. aeruginosa*. Although healthy individuals are often unaffected by HPV, the virus may colonize any area of the body with sufficient moisture to create a niche. About 8–10% of all infections linked to healthcare are caused by *P. aeruginosa*.

Infections in the US (51,000 cases in 2013). About 13% of these cases had resistant to several drugs strains, and a rising proportion of pan-drug-resistant specimen that were unresponsive to any anti-pseudomonal medications used in the medical center were found (Wagner et al., 2016).

One of the most prevalent forms of trauma degradation is burns (Atilla et al., 2015). Burns affect the skin's ability to withstand pathogen activity and the integrity of the skin's immune system (Aleksiewicz et al., 2015). For patients with burns, nosocomial infection is a serious risk. One of the main causes of morbidity and death among hospitalized burn victims is infection. Patients who have burns are more susceptible to nosocomial infections due to their compromised status and type of injury (Risan et al., 2020). By definition, a wound is a break in the integrity of the skin's epithelium and can result in additional disruptions to the physiology, anatomy, and functioning of the skin. Wounds can be classified as either acute or chronic (Che Soh et al., 2020).

Methodology

This progressive investigation, which involved 69 clinical samples taken from patients with burn (n = 53) and wound (n = 16) infections, was carried out between January 12, 2021, and January 4, 2022. 58.6% and 50.9%, respectively, of the male and female patients who attended Al Hussain Teaching Hospital and Burn Hospital in Medical City, Baghdad Teaching Hospital and had ages ranging from one month to 73 years, were included in the study.

Bacterial isolation

On MacConkey, cetrimide, and blood agar, all clinical samples were infected. The common lab procedure for *P. aeruginosa* identification was applied to identify the isolates antibiotic susceptibility testing.

Assessment of antibiotic susceptibility

The Kirby-Bauer disc-diffusion method was used to test Mueller-Hinton agar (Neogen, USA) for antibiotic sensitivity against the most widely used antibiotics (Bioanyalyse, Mast group, Himedia). Eleven antimicrobial drugs comprising Ceftazidime (CAZ), Imipenem (IPM), Piperacillin-tazobactam (PRL), Levofloxacin (LEV), Meropenem (MEM), Gentamicin (GN), Amikacin (AK), Ticarcillin (TC), Cefepime (FEP), Aztreonam (ATM), and Ciprofloxacin (cip) were examined for susceptibility.

Table 1. Antibiotics Discs utilized in This Study

Antibiotic	Symbol	Content	Origin
Gentamicin	CN	10ug	Bioanyalyse
Aztreonam	ATM	30 ug	Bioanyalyse
AMIKACIN	AK	30ug	Mast group
Ciprofloxacin	CIP	5MCg	Himedia
LEVOFLOXACIN	LEV	5Ug	Bioanyalyse
Ticarcilin	Tc	75/10 ug	Bioanyalyse
Meropenem	MEM-10	10Ug	Himedia
PiPeracillin	PIT	100/10Ug	Bioanyalyse
Imipenem	IPM-10	10Ug	Bioanyalyse
Cefepime	FEP-10	10ug	Bioanyalyse
Ceftazidime	CAZ-30	30ug	Bioanyalyse

Result

The current study analyzed 69 isolates of *P. aeruginosa* strains that obtained from burn 53 (60.9) and wound 16 (42.1) infections the result of *P. aeruginosa* according gender in male high than female score 41 (58.6) while female 28 (50.9), the result of *P. aeruginosa* according location appear in Baghdad 36 (59) while in Muthanna city 33 (51.6), the result according age appear the age <24 score 41 (62.1), while age 25-50 score 17 (53.1) and age >50 sign 11 (40.7)

The current study analyzed 69 isolates of *P. aeruginosa* strains that obtained from burn 53 (60.9) and wound 16 (42.1) infections the result of *P. aeruginosa* according gender in male high than female score 41 (58.6) while female 28 (50.9), the result of *P. aeruginosa* according location appear in Baghdad 36 (59) while in Muthanna city 33 (51.6), the result according age appear the age <24 score 41 (62.1), while age 25-50 score 17 (53.1) and age >50 sign 11 (40.7).

Table 2. Frequency of General risk factors according to culture positivity

Positive Growth	Gender		Location		Age		
	Male	Female	Baghdad	Muthanna	<24	25-50	>50
<i>P. aeruginosa</i>	41 (58.6)	28 (50.9)	36 (59)	33 (51.6)	41 (62.1)	17 (53.1)	11 (40.7)
Other Bacteria	29 (41.4)	27 (49.1)	25 (41)	31 (48.4)	25 (37.9)	15 (46.9)	16 (59.3)
Total	70 (56)	55 (44)	61 (48.8)	64 (51.2)	66 (52.8)	32 (25.6)	27 (21.6)
P value	0.469 ^a		0.473 ^a			0.164 ^a	
	0.118 ^b		0.718 ^b			<0.001 ^{*b}	

* represent a significant difference at $p < 0.05$. a; the statistical analysis between positive growth culture (*pseudomonas* and other bacteria) and risk factors (Gender, Location and Age groups), b; the statistical analysis between culture results (*P. aeruginosa*) and risk factors (Gender, Location and Age groups).

Table 3 show percentage of *P. aeruginosa* in burn swab 53 (60.9) while in wound swab 16 (42.1).

Table 3. Frequency of culture positivity according to Source of infection

Positive growth	Source	
	Burn Swab	Wound Swab
<i>P. aeruginosa</i>	53 (60.9)	16 (42.1)
Other bacteria	34 (39.1)	22 (57.9)
Total	87 (69.6)	38 (30.4)
P value	a- 0.078 b- <0.0001* c- 0.109	

* indicate a p-value of less than 0.05 substantial difference. The statistical analysis that is conducted in the following three cases: a) the positive growth culture (*pseudomonas* and other bacteria) and the source of infection (Burn and Wound); b) the culture results (*P. aeruginosa*) and the source of infection (Burn and Wound); c) the culture results (Other bacteria) and the source of infection (Burn and Wound).

As advised by CLSI (2021), *Pseudomonas aeruginosa* isolates from burn wounds were evaluated for antibiotic sensitivity using the Kirby Bauer Disk Diffusion technique. The antigram of the isolates revealed varying resistance to the antibiotics under study, as shown in table (2). Ceftazidime (CAZ), Imipenem (IPM), Piperacillin-tazobactam (PRL), Levofloxacin (LEV), Meropenem (MEM), Gentamicin (GN), Amikacin (AK), Ticarcillin (TC), Cefepime (FEP), Aztreonam (ATM), and Ciprofloxacin (cip) were among the eleven antimicrobial agents whose susceptibilities were evaluated for. All of the antibiotics we utilized in our investigation did not work on the isolates a rise of antibiotic-resistant

bacteria, which is thought to constitute a significant treatment issue. As per the findings, the greatest proportion of resistance to Ceftazidime and Gentamicin was 85.5%, while the lowest rate was observed for Ciprofloxacin 14 (20.28%).

The figure 1 show the percentage of that *P. aeruginosa* were higher resistant to ceftazidime score 85.50%, for Cefepime 73.91%. The resistant to Piperacillin was 56.52%, while for Aztreonam 65.21%, Imipenem (28.98) and Meropenem (26.0), levofloxacin score 57.97%, Ciprofloxacin reach (78.26%), Gentamycin (85.5%) and amikacin (AK) record (26.08%).

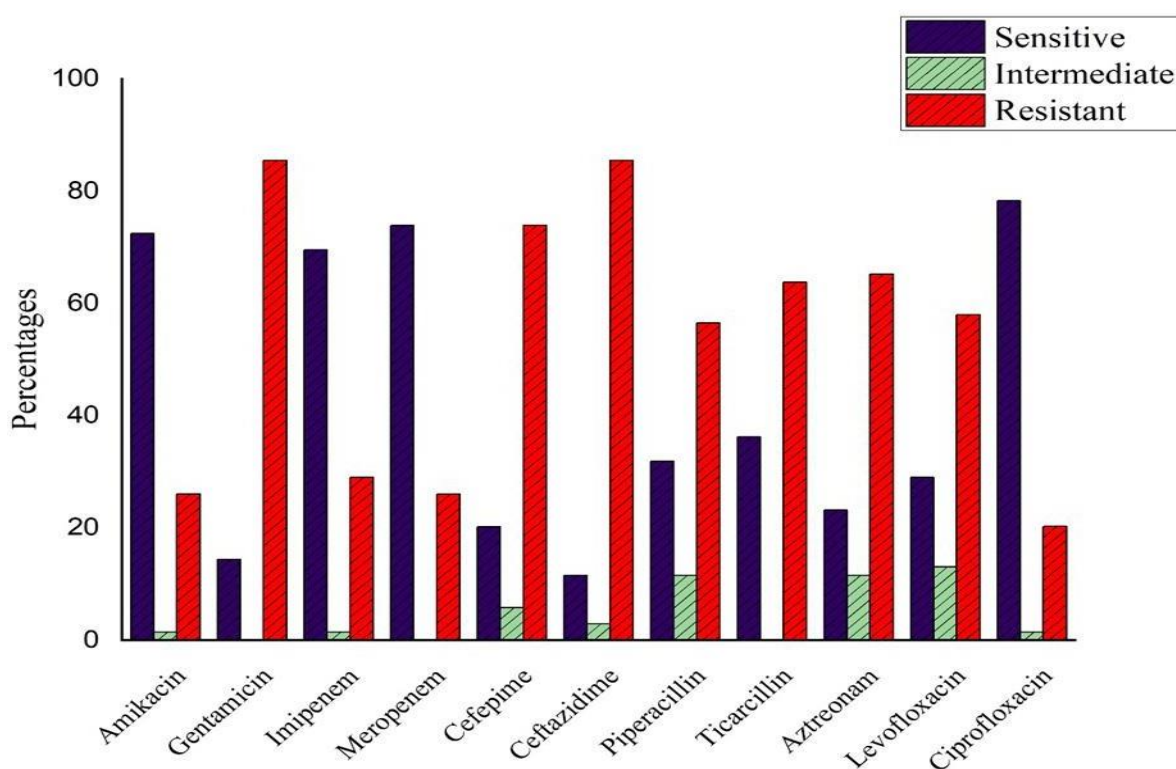


Figure 1. Antibiotic Resistance ratios among *P. aeruginosa* isolates

Discussion

The present study shows of *P. aeruginosa* resistance to ceftazidime (85.50%) that agreed with the results of previous studies (Alhamdani & Al-Luaibi, 2020) the recorded 100% resistance for ceftazidime and agree with (Abdul-Wahid et al., 2015) and (Al-Janahi et al., 2020) where record (79.3%) and (69.3) respectively in other hand disagreed with Al-Mayyahi, (2018) were recorded at 31.7%. Ceftazidime is an antibiotic that is part of the cephalosporin category. The beta-lactam ring present in cephalosporin antibiotics prevents the production of the bacterial cell wall. The reason for higher susceptibility to this class of antibiotics could be attributed to the non-permeability of these drugs through the plasma membrane (Brooks et al., 2007), the presence of the extended-spectrum β -lactam enzyme, which is often expressed as a plasmid and disrupts the β -lactam ring, preventing it from

binding to the cell wall protein (Bush & Jacoby, 2010), or the secretion of chromosomally expressed enzymes (Lopez-Yeste et al., 1996).

The another member of Cephem the Cefepime score resistance in our study (73.91%) compatible with (11) indicated that the rate of antibiotic cefepime resistance (74.3%) The current results obtained from resistance of *Pseudomonas aeruginosa* towards piperacillin (56.52%) was agreed with Al-Saeedi & Raheema (2019) were recorded 59.7% for resistance of (PA) to piperacillin. While the results obtained by Ahmed, Z. (2015), Abdel et al (2016), and Kamal et al (2018). *P. aeruginosa* appears to be more resistant to piperacillin, with a 100% resistance rate. *P. aeruginosa* appears to be very resistant to these antibiotics when they are used excessively, despite their therapeutic significance. Frequent overuse and irregular usage will lead to an increase in bacterial resistance and the emergence of numerous bacterial isolates that are identified by their multidrug resistance (Ali et al., 2015). Ticarcillin is another member of β -Lactams show high resistance in our study reach to (63.76%) close rate to Qader et al (2021) were score (71%).

As for the antibiotic aztreonam, our local isolates showed (65.21%) resistance which agree with the study of Alhamdani & Al-Luaibi (2020) which recorded resistance to this antibiotic (65.38%) While the study of Naqvi et al (2005) disagreed with the current result who recorded the resistance of bacteria to this antibiotics in a percentage (6.8%) meropenem and imipenem appear sensitive score in current study (73.9%) and (69.56) respectively agreed with Al-Kazrage was score (65%)for meropenem and (%70) for Imipenem while disagree with Hu et al (2021) was resistance recorded (75%) for both antibiotic. Tested for all bacterial isolates under investigation, levofloxacin, one of the six medicines in the fluoroquinolones category, can be utilized towards *P. aeruginosa*. As for levofloxacin, our results appear (57.97%) were similar to resistant of levofloxacin with Abdul-Wahid & Almohana (2015) was score (62%) while the result of Oumeri & Yassin (2021) in Duhok city, Iraq disagree with current study score low resistance 13.3%. The present study recorded a high sensitivity of the *P. aeruginosa* to Ciprofloxacin antibiotic belonging to the Fluoroquinolones group at the current results (78.26%). It was consistent with the study of Al-Mayyahi (2018) who recorded the percentage of bacteria sensitivity from patients with burns and wound to Ciprofloxacin antibiotic rate (69.84%).

The two primary methods by which *Pseudomonas aeruginosa* resists fluoroquinolones are the efflux pump and structural alteration of target enzymes (Hooper, 2001). Point mutations in the gyrA/gyrB genes in the QRDR (quinolone-resistant- determinative region) pattern, which is thought to be the active site of the enzyme, can modify the principal target for fluoroquinolones (DNA gyrase, sometimes referred to as topoisomerase II). Due to these mutations, the amino acid sequences of subunits A and B are changed, which results in the production of a modified form of topoisomerase II that has a decreased affinity for binding quinolone molecules. Point mutations in the parC and parE genes, which encode the ParC and ParE enzyme subunits, respectively, can modify the secondary target (topoisomerase IV) (Masuda et al., 2000). The results in the Table (2) show that resistance to amikacin (AK) was (26.08%), this result agree with Hassan et al (2012) was recorded (25%) while show more

resistance in score obtained by Al-Kaisse et al (2015) and Abdel et al (2016) that showed that the percentage to amikacin resistance was 79% and 82%. Respectively resistance for gentamycin in our study (85.5%) full compatible to study in Sulaimaniyah, Iraq (Othman et al., 2014) score (85.3%) but in study previous, score less in resistance (50%) by Attiah et al (2021).

Some genetically expressed enzymes, such as N-acetyl-transferases, adenyl-transferases, and phosphosphor-transferases, may be the cause of the bacterial resistance to this family of antibiotics. These enzymes have been recognized as particular classes of AMEs (aminoglycoside-modifying enzymes) (Aghazadeh et al., 2014). Such enzymes may have chromosomal or portable plasmid-encoded genes (Cox et al., 2015).

Conclusion

The results of this investigation showed that *P. aeruginosa* isolates have increased resistance to piperacillin, ticarcillin, azatreonam, cefepime, and gentamicin. On the other hand, its resistance to imipenem, meropenem, and amikacin is minimal. Consequently, doing antibiotic monitoring is crucial.

References

- Abdel, F. R., Abbas, M. K., Jaafar, F. N., & Mukhlif, M. (2016). Detection of Genome Content of *Pseudomonas Aeruginosa* Biofilm Formation and Resistance to Some Disinfectants and Antibiotics. *Engineering and Technology Journal*, 34(1 Part (B) Scientific).
- Abdul-Wahid, A., & Almohana, A. (2015). Dissemination of Aminoglycosides Resistance in *Pseudomonas Aeruginosa* Isolates in Al-Nasiriya Hospitals. *Kufa Journal for Nursing Sciences*, 5(1), 126-136.
- Aghazadeh, M., Hojabri, Z., Mahdian, R., Nahaei, M. R., Rahmati, M., Hojabri, T., & Pajand, O. (2014). Role of Efflux Pumps: MexAB-OprM and MexXY(-OprA), AmpC Cephalosporinase and OprD Porin in Non-Metallo- β -Lactamase Producing *Pseudomonas Aeruginosa* Isolated From Cystic Fibrosis and Burn Patients. *Infection, Genetics and Evolution*, 24, 187-192.
- Ahmed, Z. (2015). Detection of Inducible Betalactamase in *Pseudomonas Aeruginosa* Isolated From Different Clinical Samples in Kirkuk City. *Kirkuk University Journal-Scientific Studies*, 10(4), 71-92.
- Al-Doory, I. A. H. (2012). A Diagnostic Study of *Pseudomonas Aeruginosa* Isolated From Contaminated Burns and Wounds Using Cultural and Molecular Methods (Doctoral dissertation, M. Sc. Thesis, College of Science for Women, University of Baghdad, Iraq).

- Aleksiewicz, R., Kostro, K., Kostrzewski, M., Lisiecka, B., Bojarski, M., & Mucha, P. A. (2015). Percentage of CD4+, CD8+, and CD25+ T Lymphocytes in Peripheral Blood of Pigs in the Course of Experimental Burns and Necrectomy. *Journal of Veterinary Research*, 59(3), 401-410.
- Alhamdani, R. J. M., & Al-Luaibi, Y. Y. (2020). Detection of *exoA*, *nan1* Genes, the Biofilm Production with the Effect of Oyster Shell and Two Plant Extracts on *Pseudomonas Aeruginosa* Isolated From Burn Patients and Their Surrounding Environment. *Systematic Reviews in Pharmacy*, 11(11).
- Alhamdani, R. J. M., & Al-Luaibi, Y. Y. (2020). Detection of *exoA*, *nan1* Genes, the Biofilm Production with the Effect of Oyster Shell and Two Plant Extracts on *Pseudomonas Aeruginosa* Isolated From Burn Patients and Their Surrounding Environment. *Systematic Reviews in Pharmacy*, 11(11).
- Ali, Z., Mumtaz, N., Naz, S. A., Jabeen, N., & Shafique, M. (2015). Multi-Drug Resistant *Pseudomonas Aeruginosa*: A Threat of Nosocomial Infections in Tertiary Care Hospitals. *Journal of Pakistan Medical Association*, 65(12), 12-16.
- Al-Kaisse, A. A., Al-Thwani, A. N., & Al-Segar, A. N. (2015). Incidence and Antibiotics Sensitivity of Multidrug-Resistance of *Pseudomonas Aeruginosa* Isolated From Burn Patients and Environmental Samples From Three Hospitals in Baghdad. *Journal of Biotechnology Research Center*, 9(2), 67-73.
- Al-Kazrage, H. A. (In press). Inhibition of Virulence Factors in *Pseudomonas Aeruginosa* Isolated From Clinical Samples Using Galardin Loaded AgPEG Nanocomposite.
- Al-Mayyahi, A. W. (2018). Detection of (*exoT*, *exoY*, *exoS* and *exoU*) Genes in *Pseudomonas Aeruginosa* Isolate From Different Clinical Sources (Doctoral dissertation, M. Sc. Thesis Submitted to the Council of the Institute of Genetic Engineering and Biotechnology for Post Graduate Studies, University of Baghdad. 60-63).
- Al-Saeedi, R. H. A., & Raheema, R. H. (2019). Molecular Diagnosis of Some Virulence Genes in *Pseudomonas Aeruginosa* Clinical Isolates in Wasit Province. *Indian Journal of Public Health*, 10(04).
- Atila, A., Tomak, L., Katrancı, A. O., Ceylan, A., & Kılıç, S. S. (2015). Mortality Risk Factors in Burn Care Units Considering the Clinical Significance of *Acinetobacter* Infections. *Ulus Travma Acil Cerrahi Derg*, 21(1), 34-38.
- Attiah, S. A., Majeed, G. H., & Mohammed, T. K. (2021). Molecular Detection of the *exoU* and *toxA* Genes Among *Pseudomonas Aeruginosa* of Patients With Burn and Wound Infection in Baghdad City. *Annals of the Romanian Society for Cell Biology*, 25(6), 109-122.
- Brooks, G. F., Carroll, K. C., Butel, J. S., Morse, S. A., Mietzner, T. A., & Jawetz, M. (2007). *Adelberg's Medical Microbiology*. *Sultan Qaboos University Medical Journal*, 7, 273.
- Bush, K., & Jacoby, G. A. (2010). Updated Functional Classification of β -lactamases. *Antimicrobial Agents and Chemotherapy*, 54(3), 969-976.

- Che Soh, N. A., Rapi, H. S., Mohd Azam, N. S., Santhanam, R. K., Assaw, S., Haron, M. N., & Ismail, W. I. W. (2020). Acute Wound Healing Potential of Marine Worm, *Diopatra Claparedii* Grube, 1878 Aqueous Extract on Sprague Dawley Rats. *Evidence-Based Complementary and Alternative Medicine*, 2020.
- Cox, G., Stogios, P. J., Savchenko, A., & Wright, G. D. (2015). Structural and Molecular Basis for Resistance to Aminoglycoside Antibiotics by the Adenylyltransferase ANT(2'')-Ia. *MBio*, 6(1), e02180-14.
- Hassan, K. I., Rafik, S. A., & Mussum, K. (2012). Molecular Identification of *Pseudomonas Aeruginosa* Isolated From Hospitals in Kurdistan Region. *Journal of Advanced Medical Research*, 2(3), 90-98.
- Hooper, D. C. (2001). Emerging Mechanisms of Fluoroquinolone Resistance. *Emerging Infectious Diseases*, 7(2), 337.
- Hu, Y., Li, D., Xu, L., Hu, Y., Sang, Y., Zhang, G., & Dai, H. (2021). Epidemiology and Outcomes of Bloodstream Infections in Severe Burn Patients: A Six-Year Retrospective Study. *Antimicrobial Resistance and Infection Control*, 10(1), 1-8.
- Kamal, M. A., Aldin, C. I., & Husein, A. S. (2018). Prevalence Study of *Pseudomonas Aeruginosa* in Teaching Tikrit Hospital From Different Sources. *Tikrit Journal of Pure Science*, 20(4), 55-59.
- Lopez-Yeste, M., Xercavins, M., Lite, J., Cuchi, E., & Garau, J. (1996). Fluoroquinolone and Aminoglycoside Resistance in Chromosomal Cephalosporinase-Overproducing Gram-Negative Bacilli Strains with Inducible Beta-lactamase. *Enfermedades Infecciosas Y Microbiologia Clinica*, 14(4), 211-214.
- Masuda, N., Sakagawa, E., Ohya, S., Gotoh, N., Tsujimoto, H., & Nishino, T. (2000). Substrate Specificities of MexAB-OprM, MexCD-OprJ, and MexXY-OprM Efflux Pumps in *Pseudomonas Aeruginosa*. *Antimicrobial Agents and Chemotherapy*, 44(12), 3322-3327.
- Naqvi, Z. A., Hashmi, K. H., Rizwan, Q. M., & Kharal, S. A. (2005). Multidrug Resistant *Pseudomonas Aeruginosa*: A Nosocomial Infection Threat in Burn Patients. *Pakistan Journal of Pharmacology*, 22(2), 9-15.
- Othman, N., Babakir-Mina, M., Noori, C. K., & Rashid, P. Y. (2014). *Pseudomonas Aeruginosa* Infection in Burn Patients in Sulaimaniyah, Iraq: Risk Factors and Antibiotic Resistance Rates. *The Journal of Infection in Developing Countries*, 8(11), 1498-1502.
- Oumeri, M. M. Q., & Yassin, N. A. (2021). Molecular Characterization of Some Carbapenem-Resistance Genes Among *Pseudomonas Aeruginosa* Isolated From Wound and Burn Infections in Duhok City, Iraq. *Journal of Duhok University*, 24(1), 136-144.
- Qader, M. K., Solmaz, H., & Merza, N. S. (2021). Molecular Characterization of Virulence Factors Among Antibacterial Resistant *Pseudomonas Aeruginosa* Isolated From Burn Infections From Duhok and Erbil Hospitals, Iraq. *Journal of Duhok University*, 24(1), 1-9.

-
- Risan, F. A., Salih, M. K., & Salih, T. A. (2020). Estimation of IL-17A and IFN- γ in the Burn of Patients that Afflicted by Different Bacterial Types. *International Journal of Psychosocial Rehabilitation*, 24(05).
- Wagner, S., Sommer, R., Hinsberger, S., Lu, C., Hartmann, R. W., Empting, M., & Titz, A. (2016). Novel Strategies for the Treatment of Pseudomonas Aeruginosa Infections. *Journal of Medicinal Chemistry*, 59(13), 5929-5969.