ORIGINAL ARTICLE

Study of Efflux Pump Genes (Ade A, Ade C Genes) in *Acinetobacter Baumannii* Isolated from Patients in National Liver Institute

Fatma O. Khalil, Tawfik M. Abd El Moteleb, Salma U.F. Elneanai*, Hanaa M. Elgazzar
Medical Microbiology & Immunology Department, National Liver Institute, Menoufia University

Key words:
Acinetobacter baumanii, efflux pump, antibiotic resistance, (AdeA,AdeC)

Corresponding Author:
Salma Usri Fawzi Elneanai
Menouf, Menoufia Governorate, Egypt
Tel.: 01064964800
salmausri@gmail.com

ABSTRACT

Background: *Acinetobacter baumannii is an opportunistic pathogen that has emerged as a major cause of hospital-acquired infections, particularly in intensive care units. It is known for its remarkable ability to survive in harsh environmental conditions and its resistance to a wide range of antibiotics, making it a significant threat to public health.* ***Objectives:*** *To study the relation of efflux pump genes (AdeA, AdeC) with the antibiotic resistant acinetobacter.* ***Methodology:*** *Bacteriological Samples were collected from 50 patients admitted to the National Liver Institute (NLI)with signs of infection, these samples were cultured on blood and MacConkey agar, identification of Acinetobacter was done by microscopy, colonies and biochemical tests and species was identified by using GN-ID cards from the VITEK-2 system. Antibiotic susceptibility testing was performed using VITEK2 AST-N73 cards, while the presence of AdeA and AdeC efflux pump genes was detected through Multiplex-PCR analysis.* ***Results:*** *Co- expression of the AdeA and AdeC efflux pump genes was detected in 68% of the bacterial isolates, a prevalence notably higher than the individual expression rates of AdeA (10%) and AdeC (4%). In contrast, 18% of isolates exhibited no detectable expression of either gene, A statistically significant correlation was observed between meropenem resistance and the presence of the AdeA gene. Similarly, a significant association was identified between levofloxacin resistance and the presence of the AdeC gene. Moreover, a significant difference in the distribution of AdeA and AdeC gene expression was observed between multidrug-resistant (MDR) isolates and nonMDR isolates.* ***Conclusion:*** *The presence of efflux pump genes AdeA and AdeC is strongly associated with high levels of antibiotic resistance in Acinetobacter spp., particularly among isolates obtained from intensive care unit (ICU) settings. Recommendation: Continuous surveillance of antimicrobial resistance patterns is important to guide the appropriate and effective use of antibiotics.*

INTRODUCTION

Acinetobacter baumannii is Gram-negative aerobic Coccobacilli and prefers humid environments for stay [1,2]. It belongs to the *Moraxellaceae* family that predominantly causes nosocomial infections. These infections are diverse and may include ventilator-associated pneumonia (VAP), urinary tract infections, meningitis, bacteremia, gastrointestinal and wound infections[3]. Regrettably, the number of multidrug-resistant (MDR) *A. baumannii* isolates has increased significantly [3].

Antimicrobial or antibiotic resistance (AMR) has emerged as a substantial and triggering phenomenon for healthcare systems worldwide. In recent years it has been related to significant morbidity, mortality, and increased cost due to both prolonged length of hospitalization and treatment [4].

Extensive drug resistant (XDR) *A. baumannii* is called an isolate resistant to three or more classes of antimicrobials (penicillins and cephalosporins—including inhibitor combinations, fluoroquinolones, and aminoglycosides, resistant to carbapenems in most of cases), while pandrug resistant (PDR) *A. baumannii* is an XDR isolate resistant to polymyxins and tigecycline. Lately, extensively drug-resistant isolates have been led to the discovery of novel antimicrobials and the introduction of new treatment approaches[5], one of the mechanisms that this bacterium uses to resist various antibiotics is the use of efflux pumps. By using efflux pumps, the *A. baumannii* can direct antibiotics outwards and prevent antibiotics from affecting *acinetobacter* [6]

World Health Organization (WHO) has mentioned *A. baumannii* in its priority list, under critical problem, for research and development of new antibiotics, Infection with *acinetobacter* is increasing in people with Immunodeficiency [7].

Several mechanisms contribute to *A. baumannii* strains resistance, such as *β-lactamases* expression, alteration of cell membrane permeability, increased expression of efflux pumps, mutations in DNA gyrases and topoisomerases encoding genes [8].

The AdeABC efflux pump, a chromosomally encoded tripartite system, belongs to the resistance-

nodulation-division (RND) superfamily of efflux transporters. This complex is composed of three key components: AdeA, a membrane fusion protein; AdeB, a multidrug transporter; and AdeC, an outer membrane channel protein. These genes are typically located in close proximity on the chromosome and are co-regulated by adjacent two- component regulatory systems [9].

The presence of the *adeABC* efflux pump genes is generally low in antibiotic-sensitive *A. baumannii* strains but is significantly more prevalent among drug-resistant isolates. This observation has led some researchers to propose that *adeABC* may serve as a molecular marker for resistance in *A. baumannii* [10]. In addition to efflux pump genes, other major resistance determinants commonly found in multidrug-resistant (MDR) *A. baumannii* strains include β-lactamase enzymes, integrons, and insertion sequence (IS) elements[11].

METHODOLOGY

Study participants:

This study was conducted in the Clinical Microbiology and Immunology Department of the National Liver Institute, Menoufia University Hospitals during the period from June 2022 to October 2023. A total of 50 patients with signs of infection were enrolled in the study. Ethical approval was obtained from the Ethical Committee Board of the National Liver Institute, Menoufia University, under the approval number NLI IRB 00014014/2024.

Samples collection:

Bacteriological samples were collected from patients exhibiting signs of active infection. The types of specimens included: urine (n = 3), blood (n = 20), ascitic fluid (n = 4), tracheal aspirates/endotracheal tube samples (n = 6), throat swabs (n = 2), nasal swabs (n = 6), drain fluid (n = 3), sputum (n= 3), and central venous catheter (CVC) tips (n = 3).In addition to microbiological analysis, a series of laboratory investigations were performed, including: Complete blood count (CBC), Liver function tests (AST, ALT, total bilirubin, serum albumin), Renal function tests (serum creatinine, urea), Inflammatory markers (C-reactive protein [CRP]), and coagulation profile (prothrombin time, INR).

Identification of the isolates:

Acinetobacter were identified by morphology of the colony, negative gram stain, culture on blood and maconkey agar and biochemical reactions (indole, urease agar, triple sugar iron tese and oxidase) then confirmed by VITEK-2 compact system GN-ID cards (bioMerieux, France).

Testing of antibiotic susceptibility:

The susceptibility of tested *Acinetobacter* l isolates to antibiotics was done using VITEK2 ASTN73 card following the manufacturer's.

Genotypic identification of virulence genes:

Detection of *AdeA* , *AdeC* genes in the *Acinetobacter baumanii* isolates was done using Multiplex-PCR by primers as shown in table1

Table 1: Primers used in the study:

Primer name	Sequence (5`-3`)	Product Size (bp)	Annealing temperature(C)	Reference
AdeA	**Forward primer GAAATCCGTCCGCAAGTC Reverse primer ACACGCACATACATACCC**	683 bp	55	**(9)**
AdeC	**Forward primer ATTTCAGGTCGTAGCATT Reverse primer CTTGATAAGTAGAGTAGGGATT**	370 bp	55	**(9)**

Extraction and purification of DNA:

Thermo Scientific gene JET™ genomic DNA Purification Kit was used for purification of DNA according to Manufacturers' instructions.

DNA amplification:

DNA amplification was done using the Primers of the genes (table 1) purchased from Thermo Fisher Scientific USA. Mixtures of PCR contain DreamTaq green PCR Master Mix (2x), 10 µl from DNA Extract, 0.25 µl from each gene forward primer and 0.25 µl from each gene reverse primer[9]. The condition for amplification of adeA,C genes was as follows: initial denaturation at 94 °C for 5 min, 30 cycles of 94 °C for 1 min, 55°C for 1 min, 72 °C for 1 min and a final amplification at 72 °C for 5 min[9]. Amplified products detection:

The amplified products size was visualized using (2%) agarose gels after ethidium bromide staining (Sigma, USA). have been determined in AdeC (370 bp) and AdeC (683bp) comparison to a DNA ladder (100-1000bp), the UV trans-illuminator and photographed by digital camera. Fig (1)

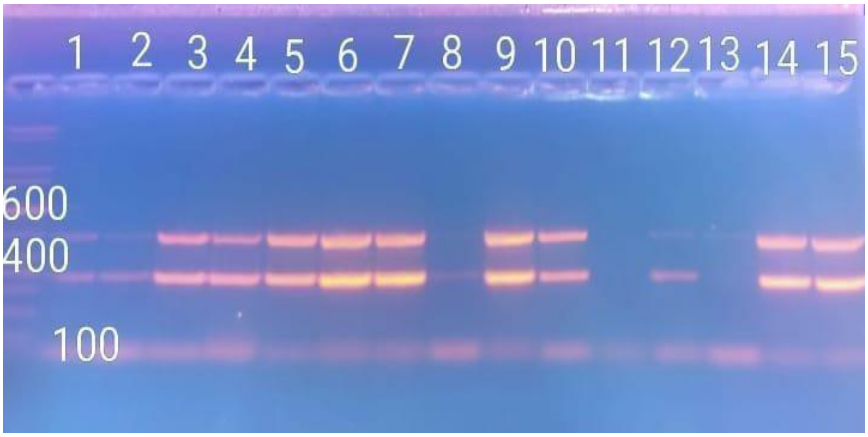

Fig. 1: Identification of AdeA , AdeC genes among *acinetobacter baumanii* isolates by using multiplex PCR.
Lanes 3,4,5,6,7,9,10,14,15 positive for gene AdeC (370 bp) and AdeA(683 bp). but lanes 1,2,8,11,13 are negative for the 2 genes. While lane 12 show positive band for Ade C gene only.

Statistical analysis:

Statistical analysis has been calculated by the SPSS - version 25. Quantitative variables were described as mean, SD, range with using Student t-test. Qualitative variables described as percentage, and Fisher's exact test or Chi-square test were used. Statistical significance was adjusted at p value
<0.05.

RESULTS

50 bacteriological samples were collected from hospitalized patients aged from 3 weeks to 80 years with Mean ± SD (53.14 ± 22.87) admitted to National Liver Institute. Males were the most participants by percentage 56.3%, but females by percentage of 43.7%.

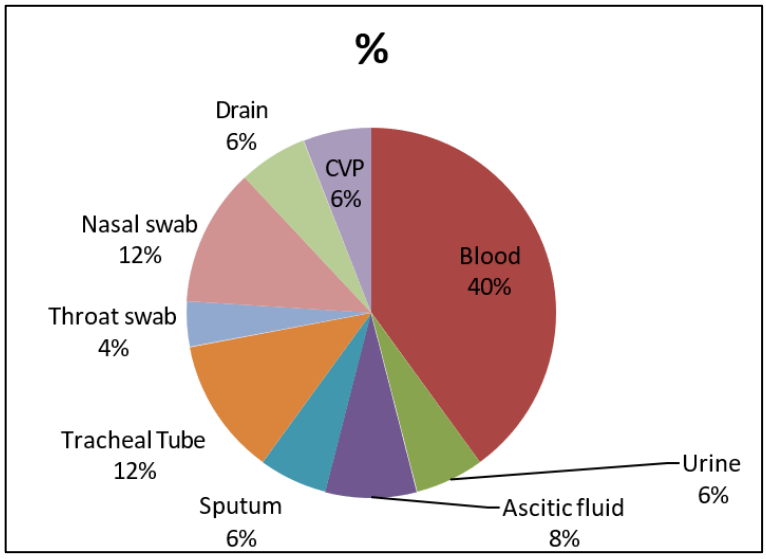

Fig. 2: Distribution of the Acinetobacter cases according to type of sample.

This figure shows that the highest percent of acinetobacter cases is blood (40%), then nasal swab and tracheal swab by (12%) for each, ascetic by (8%) But the least are urine, sputum and drain by (6%) for each.

As shown in table 3: Acinetobacter baumanii exhibited high resistance rates for most of the tested antibiotics. The highest resistance rates were for cefepime and Tetracyclin (78.0%) followed by Ceftazidime, Amikacin, Sulfatrimethoprim (70%) then Ciprofloxacine (66%), Impenem (64%), Meropenem (56%) and finally Gentamycin, Piperacillin–Tazobactam, Ampicillin- Sulbactam (54%) for each.

Table 3: Antibiotic sensitivity of *Acinetobacter baumanii* isolates (N= 50) by VITEK 2 compact system

Antimicrobial agent	Acinetobacter baumanni		
	Sensitive	Intermediate	Resistant
Impenem	6(12.0%)	12(24.0%)	32(64.0%)
Meropenem	5(10.0%)	17(34.0%)	28(56.0%)
Gentamycin	7(14.0%)	16(32.0%)	27(54.0%)
Piperacillin –Tazobactam	2(4.0%)	21(42.0%)	27(54.0%)
Ampicillin	5(10.0%)	21(42.0%)	27(54.0%)
Ampicillin Sulbactam	4(8.0%)	21(42.0%)	25(50.0%)
Ceftazidime	3(6.0%)	12(24.0%)	35(70.0%)
Cefepime	4(8.0%)	7(14.0%)	39(78.0%)
Amikacin	3(6.0%)	12(24.0%)	35(70.0%)
Tetracycln	3(6.0%)	8(16.0%)	39(78.0%)
Ciprofloxacine	2(4.0%)	15(30.0%)	33(66.0%)
Sulfatrimethoprim	1(2.0%)	14(28.0%)	35(70.0%)
Levofloxacine	7(14.0%)	23(46.0%)	20(40.0%)

As shown in table 4: AdeA and AdeC genes were significally higher in MDR group than Non MDR group.

Table 4: **Comparison between the different groups of *Acinetobacter baumannii* regarding the presence of adeA and adeC genes:**

		Non MDR		MD	R	Test of significance	P value
		Count	%	Count	%		
AdeA	Present	5	45.5%	31	79.5%	4.929	**0.026***
	Absent	6	54.5%	8	20.5%		
Ade.C	Present	5	45.5%	34	87.2%	8.705	**0.003***
	Absent	6	54.5%	5	12.8%		

As shown in table 5: AdeA gene was significally higher in meropenam resistant strains.

Table 5: Relation between presence of adeA gene and resistance to different antibiotics

		AdeA				Test of significance	P value
		Present		Absent			
		Count	%	Count	%		
Imipenem	Sensitive	5	13.9%	1	7.1%	0.43	0.51
	Resistant	31	86.1%	13	92.9%		
Meropenem	Sensitive	1	2.8%	4	28.6%	7.5	**0.006***
	Resistant	35	97.2%	10	71.4%		
Gentamycin	Sensitive	4	11.1%	3	21.4%	0.9	0.35
	Resistant	32	88.9%	11	78.6%		
Pipra	Sensitive	2	5.6%	0	0.0%	0.81	0.37
	Resistant	34	94.4%	14	100.0%		
Ampicillin	Sensitive	3	8.3%	2	14.3%	0.4	0.53
	Resistant	33	91.7%	12	85.7%		
Salbactam	Sensitive	3	8.3%	1	7.1%	0.02	0.89
	Resistant	33	91.7%	13	92.9%		
Ceftazedim	Sensitive	2	5.6%	1	7.1%	0.045	0.83
	Resistant	34	94.4%	13	92.9%		
Cefepime	Sensitive	2	5.6%	2	14.3%	1.04	0.3
	Resistant	34	94.4%	12	85.7%		
Amikacin	Sensitive	2	5.6%	1	7.1%	0.045	0.83
	Resistant	34	94.4%	13	92.9%		
Tetracyclin	Sensitive	2	5.6%	1	7.1%	0.045	0.83
	Resistant	34	94.4%	13	92.9%		
Ciprofloxacin	Sensitive	2	5.6%	0	0.0%	0.81	0.37
	Resistant	34	94.4%	14	100.0%		
Salfamethoprim	Sensitive	1	2.8%	0	0.0%	0.4	0.53
	Resistant	35	97.2%	14	100.0%		
Levofloxacin	Sensitive	4	11.1%	3	21.4%	0.89	0.35
	Resistant	32	88.9%	11	78.6%		

As shown in table 6: AdeC gene was significally higher in levofloxacine resistant strains.

Table 6: Relation between presence of adeC gene and resistance to different antibiotics

		Ade.C				Test of significance	P value
		Present		Absent			
		Count	%	Count	%		
Imipenem	Sensitive	5	12.8%	1	9.1%	0.11	0.74
	Resistant	34	87.2%	10	90.9%		
Meropenem	Sensitive	3	7.7%	2	18.2%	1.05	0.31
	Resistant	36	92.3%	9	81.8%		
Gentamycin	Sensitive	6	15.4%	1	9.1%	0.28	0.6
	Resistant	33	84.6%	10	90.9%		
Pipra	Sensitive	2	5.1%	0	0.0%	0.59	0.44
	Resistant	37	94.9%	11	100.0%		
Ampicillin	Sensitive	3	7.7%	2	18.2%	1.05	0.31
	Resistant	36	92.3%	9	81.8%		
Salbactam	Sensitive	3	7.7%	1	9.1%	0.02	0.88
	Resistant	36	92.3%	10	90.9%		
Ceftazedim	Sensitive	2	5.1%	1	9.1%	0.24	0.63
	Resistant	37	94.9%	10	90.9%		
Cefepime	Sensitive	2	5.1%	2	18.2%	1.99	0.16
	Resistant	37	94.9%	9	81.8%		
Amikacin	Sensitive	1	2.6%	2	18.2%	3.7	0.054
	Resistant	38	97.4%	9	81.8%		
Tetracyclin	Sensitive	3	7.7%	0	0.0%	0.9	0.34
	Resistant	36	92.3%	11	100.0%		
Ciprofloxacin	Sensitive	2	5.1%	0	0.0%	0.59	0.44
	Resistant	37	94.9%	11	100.0%		
Salfamethoprim	Sensitive	1	2.6%	0	0.0%	0.29	0.6
	Resistant	38	97.4%	11	100.0%		
Levofloxacin	Sensitive	3	7.7%	4	36.4%	5.9	**0.016***
	Resistant	36	92.3%	7	63.6%		

DISCUSSION

The combination of high levels of resistance and the ability to acquire new resistance mechanisms makes *Acinetobacter* challenging to treat, the emergence of extensively drug-resistant strains adds another layer of complexity, limiting treatment options and increasing the risk of treatment failure [12]. In this study we observed that both AdeA and AdeC shows high percent than each gene only (68%) in relation to (10%,4%) of every gene alone, In the other hand acinetobacter isolates that not express the both genes were (18%). Which was on agreement with the study of Zong et al and Terkuran et al [13,14] And in contrary with Santos et al. noted that there was a "weak role" for AdeABC in resistance[15].

The present study showd that Ade A gene had the highest percent in the acinetobacter tracheal tube isolates, ascetic fluid, Drain, CVC (100%) and AdeC gene had the heighist percent in the acinetobacter tracheal tube isolates, ascetic fluid, Drain, CVC, throat swab (100%) for each followed by acinetobacter blood samples (70%). In agree with us the study of Ranjbar R et al[9]. AdeA gene showed a 100% prevalence in *Acinetobacter* isolates from tracheal tubes, ascitic fluid, drains, and central venous catheters (CVCs) in agree with Azab et al. [10].

In our study, *A. bumani* was the highest rate of resistance to Ceftazidime, cefepime and tetracycline (78% for each) % followed by Amikacin, Sulfatrimethoprim (70%) then Ciprofloxacine (66%), Impenem (64%), Meropenem (56%) and finally Gentamycin, Piperacillin –Tazobactam, Ampicillin-Sulbactam (54%) for each., this result was agreed by another research papers Azab et al.[10].

In the present study, *Acinetobacter baumannii* isolates demonstrated significant resistance to tetracycline (71%) and meropenem (74.2%). These results are consistent with previous research, which has identified key resistance mechanisms in *A. baumannii*, including the production of beta-lactamases and the

activity of efflux pumps, both of which contribute substantially to antimicrobial resistance, in agree with Rumbo et al [16].

Tashkan et al. observed resistance rates in Acinetobacter baumannii isolates, with Ciprofloxacin showing 95% resistance, Imipenem at 82%, and Gentamicin at 35%. Furthermore, the prevalence of Multidrug Resistance (MDR) and Extensively Drug Resistance (XDR) in the studied strains were found to be 76% and 30%, respectively. This was compared with similar findings from the present study, where higher resistance rates for Ciprofloxacin and Imipenem were reported [17].

This study showed that For the MDR group all ascetic, drain, cvp isolates are 100% MDR, but tracheal and nasal swabs show 83.3% resistance, blood 75%, urine 766.7%, throat swab 50% and sputum 33.3%, In agreement with this study was Abd-Elsalam, Fetal and Alelign,Detal [18,19] and with disagreement with Smith et al and Patel, R. et al studies [20,21]

There was asignificant relationship between meropenam resistance and the prescence of A deA genes in *acinetobacter baumanii* isolates. As (p value <0.05). and asignificant relationship between levofloxacine resistance and the prescence of A deC genes in *acinetobacter baumanii* isolates. As (p value <0.05) in agree with us Zong et al and Zhang et al[14,22] but in disagreement were poliou, M. et al and Ali, A., et al studies[23,24]. This difference could be explained as Poliou et al. and Ali et al. had smaller sample sizes.

The present study found that *Acinetobacter baumannii* isolates with multidrug resistance (MDR) frequently harbor the adeA and adeC genes. This is consistent with the role of the adeABC efflux pump system, which play a significant role in the resistance of *A. baumannii* to multiple antibiotics by actively transporting the drugs out of bacterial cells, thus reducing their intracellular concentrations and diminishing their efficacy. Studies by Zong, Z. et al. and Zhang, X. et al align with your results [14,22].

CONCLUSION

Our study focused on virulence genes and antibiotic susceptibility pattern of *Acinetobacter baumaii* among NLI patients. As there was a relationship between efflux pump genes AdeA, AdeC and MDRO *Acinetobacter baumanii*. Recommendation: Surveillance of drug resistance should be done regularly for proper antibiotics selection.

Conflict of interest
There is no conflict of interest.
Fund: no fund was received.

REFERENCES

1. Bartal C, Rolsto KV, Nesher L. Carbapenem-resistant Acinetobacter baumannii: Colonization, Infection and Current Treatment Options. Infect Dis Ther, 2022 .11, 683–694.
2. Ahuatzin-Flores OE, Torres E, Chávez-Bravo E. Acinetobacter baumannii, a Multidrug-Resistant Opportunistic Pathogen in New Habitats: A Systematic Review. Microorganisms, 2022 12(4), 644.
3. Nasr P. Genetics**.** Epidemiology and clinical manifestations of multidrugresistant Acinetobacter baumannii. J. Hosp. Infection, 2020. 104, 4–11.
4. Ayoub Moubareck, C, Hammoudi Halat. Insights into Acinetobacter baumannii: A Review of Microbiological, Virulence, and Resistance Traits in a Threatening Nosocomial Pathogen, Antibiotics. 2020. 9(3):119
5. Karakonstantis S, Kritsotakis EI, Gikas A. Treatment options for K. pneumoniae, P. aeruginosa and A. baumannii coresistant to carbapenems, aminoglycosides, polymyxins and tigecycline: an approach based on the mechanisms of resistance to carbapenems. Infection, 2020. 48, 835–851
6. Ming-Feng Lin, Yun-You Lin, Chi-Chao Tu, Chung-Yu Lan. Distribution of different efflux pump genes in clinical isolates of multidrug-resistant Acinetobacter baumannii and their correlation with antimicrobial resistance. Journal of Microbiology, Immunology and Infection ,2020. 50, 2, 224-231
7. Lee C-R, Lee JH, Park M, Park KS, Bae IK, Kim YB, Cha CJ, Jeong BC, Lee SH. Biology of Acinetobacter baumannii: Pathogenesis, antibiotic resistance mechanisms, and prospective treatment options. Frontiers in Cellular and Infection Microbiology, 2017; 7, 55.
8. Vrancianu CO, Gheorghe I, Czobor IB, Chifiriuc MC. Antibiotic resistance profiles, molecular mechanisms and innovative treatment strategies of acinetobacter baumannii. Microorganisms; 2020 .8(6):935
9. Ranjbar R, Zayeri S, Afshar D. High Frequency of AdeA, AdeB and AdeC Genes among Acinetobacter baumannii Isolates. Iran J Public Health. 2020 .49(8):1539-1545.
10. Azab H, Askar AM, El-Fadeal N. Detection of AdeAB, TetA, and TetB efflux pump genes in clinical isolates of tetracyclineresistant Acinetobacter baumannii from patients of Suez Canal University Hospitals. BMC Microbiol 2025.25, 63

11. Lukovic B, Kabic J, Dragicevic M. et al. Genetic basis of antimicrobial resistance, virulence features and phylogenomics of carbapenem-resistant Acinetobacter baumannii clinical isolates. Infection ,2025 .53, 39–50
12. Mukhopadhyay R, Bairagi A, Mukherjee A, Prasad AK, Roy D, Nayak A. Multidrug resistant Acinetobacter baumannii: A study on its pathogenesis and therapeutics. Current Research in Microbial Sciences,2025. 8, 100331.
13. Terkuran M, Erginkaya Z, Konuray G, Meral M, Ünal N, Yaşar S, Köksal F. Evaluation of antibiotic resistance and adeABC, adeR, adeS efflux pump genes among foodborne and clinical Acinetobacter spp. in Türkiye. Iranian Journal of Public Health, 2022. 51(12), 2753–2763.
14. Zong Z, Lin D, Zhang Y. Role of efflux pumps in the multidrug resistance of Acinetobacter baumannii. Antimicrobial Agents and Chemotherapy, 2020.64(10),
15. Santos A, Silva A, Figueiredo S. Acinetobacter baumannii: Clinical significance and resistance mechanisms. Journal of Antimicrobial Chemotherapy, 2021.76(5), 1230-1239
16. Rumbo, C., Gato, E., López, M., Ruiz de Alegría, C., Fernández-Cuenca, F., Martínez-Martínez, L., Vila, J., Pachón, J., Cisneros, J. M., Rodríguez-Baño, J., Pascual, A., Bou, G., & Tomás, M ,Contribution of efflux pumps, porins, and β-lactamases to multidrug resistance in clinical isolates of *Acinetobacter baumannii*. *Antimicrobial Agents and Chemotherapy*, . 2013 *57*(11), 5247–5257
17. Basatian-Tashkan, B, Niakan M, Khaledi M, Afkhami H., Sameni F, Bakhti S, Mirnejad R. Antibiotic resistance assessment of Acinetobacter baumannii isolates from Tehran hospitals due to the presence of efflux pumps encoding genes (adeA and adeS genes) by molecular method. BMC Research Notes, 2020. 13(1), 543
18. Abd-Elsalam, F., Zeinelabedin, M., Abdelrahman, S., & Gabal, H. ,Bacteriological Profile and Antimicrobial Resistance in Ascitic Fluid of Patients with Community Acquired and Nosocomial Spontaneous Bacterial Peritonitis. Benha Medical Journal ,2022. 39(2), 647-665. doi: 10.21608/bmfj.2022.97727.1490
19. Alelign, D., Ameya, G., & Siraj, M. Bacterial pathogens, drug-resistance profile, and its associated factors from patients with suspected peritonitis in Southern Ethiopia. Infectious Diseases and Therapy, 2021. 14, 4107-4117. https://doi.org/10.2147/IDR.S335103
20. Smith J, Wang L, Chen G. Biofilm formation on central venous catheters: Implications for catheter-related infections. American Journal of Infection Control, 2024.52(5), 567-573.
21. Patel R, Desai H, Shah A. Drain fluid cultures in postoperative infections: A study of microbial flora and resistance patterns. Infection Control & Hospital Epidemiology, 2024.45(7), 789-795
22. Zhang X, Liu J, Wang J. Contribution of the AdeABC efflux pump to carbapenem resistance in Acinetobacter baumannii isolates. Microbial Drug Resistance,2023. 29(1), 11-19.
23. Ali A, Mahmood SS. Role of RND-efflux pump in levofloxacin resistance among Acinetobacter baumannii. Iraqi Journal of Agricultural Sciences, 2020.53(3), 542–550.
24. Poliou M, Markou A, Pournaras S. Mechanisms of carbapenem resistance in Acinetobacter baumannii: A review. Microbial Drug Resistance2020.6(4), 424-434.