INTERNATIONAL JOURNAL OF MEDICAL SCIENCE AND DENTAL HEALTH
(ISSN: 2454-4191)
www.ijmsdh.org (2026)

Received: 06 January 2026
Revised: 22 January 2026
Accepted: 19 February 2026
Published: 28 February 2026

Page No - 121-127
DOI - 10.55640/ijmsdh-12-02-15

Article Citation: Witwit , L. J., Al-Ardawi , E. J. R. ., & Altabtabaai, T. A. . (2026). Detection of Blandm And Blaoxa-23 Genes in Acinetobacter Baumannii Isolated from Sputum Samples of Iraqi Patients. International Journal of Medical Science and Dental Health, 12(02), 121-127. https://doi.org/10.55640/ijmsdh-12-02-15

Detection of Blandm And Blaoxa-23 Genes in Acinetobacter Baumannii Isolated from Sputum Samples of Iraqi Patients

Luma Jasim Witwit
Department of Microbiology, College of Dentistry, University of Babylon, Hilla, Iraq

Eman Jaber Raheem Al-Ardawi
Ministry of Education, General Directorate of Education in Holy Karbala

Teeb Ahmed Altabtabaai
Department of Field Crops, College of Agriculture, University of Al-Qasim Green, Babylon, Iraq

Abstract

Acinetobacter baumannii is a pivotal pathogenic bacterium causing hospital-acquired respiratory infections, particularly in critically ill patients, and its prevalence of multidrug-resistant strains has increased globally. Two of the most relevant carbapenemase genes from a clinical standpoint is blaOXA-23 and blaNDM which are highly involved in conferring resistance to carbapenems in *A. baumannii.* The aim of this study was to investigate the prevalence of the blaOXA-23 and blaNDM genes in A. baumannii isolates isolated from sputum samples obtained from Iraqi patients, and whether these genes were associated with patterns of resistance to specific antibiotics. This is a cross-sectional study performed in the Teaching Hospital in Al-Diwaniyah City, Iraq during March 2025 to August 2025. A total of 48 patients with a clinically suspected lower RTI were recruited. Sputum aspirates were obtained under aseptic conditions and processed by standard microbiological procedures. *A. baumannii* was identified by colony morphology, Gram staining and standard biochemical testing. The susceptibility testing for imipenem, meropenem, amikacin gentamicin ciprofloxacin ceftazidime and piperacillin–tazobactam (p-t) were performed by the Kirby–Bauer disk diffusion according to the CLSI. Molecular identification of blaOXA-23 and blaNDM genes was done by conventional

polymerase chain reaction (PCR). High resistance rates were also noted for carbapenems, imipenem (87.5%) and meropenem (85.4%). Resistance was also high for ceftazidime (81.3%) and piperacillin/tazobactam (79.2%), which were much lower in case of aminoglycosides. Molecular investigation indicated that blaOXA-23 and blaNDM genes were observed in 83.3% and 37.5% of the isolates, respectively. The link between resistance to carbapenems and the simultaneous presence of blaOXA-23 and blaNDM genes was strongly significant. These observations show high rates of blaOXA-23 but still considerable rates of blaNDM enzymes among CRAB isolates in Al-Diwaniyah, and highlight the necessity for regular molecular surveillance, proper infection control measures and improved antimicrobial stewardship policies to prevent expansion of MDR *A. baumannii* strains.

Keywords: blaOXA-23, blaNDM, Acinetobacter baumannii, pneumonia, Carbapenemase

Introduction

Acinetobacter baumannii is a Gram-negative pathogenic bacterium that cause nosocomial infections and has growing global importance as one of the most problematic agent accountable for healthcare-related infections in patients, mainly induced in intensive care units (ICUs) and among those having the ventilator-associated pneumonia beside developing other life-threatening nosocomial infections (Thacharodi et al., 2024). This bacterium is one of the members of the ESKAPE group (six highly virulent and multidrug-resistant bacterial pathogens), which is a pool of the bacterial pathogens that are found to have antibiotic resistance mechanism against multiple antimicrobial agents and causes noxious infections having high morbidity and mortality rate. Before, *A. baumannii* was described as an 'inherently low pathogenomic' environmental bacterium, but its astonishing genome plasticity would lead it to acquire and disseminate a cargo of antimicrobial genes seriously hampering clinical practices (de Souza et al., 2025).

Imipenem, meropenem and doripenem are important carbapenems used for serious gram-negative infections including those due to the multidrug-resistant A. baumannii. Nevertheless, the dissemination of Carbapenem-Resistant Acinetobacter baumannii (CRAB) at a worldwide level has considerably reduced their usefulness, causing treatment failures and an increase in mortality in patients with severe underlying disease (Thacharodi et al., 2024; Muller, 2023). CRAB is a priority pathogen to global health organizations and has been correlated with outbreaks, endemic resistance in the hospital. Carbapenem-resistant *A. baumannii* has been designated as a critical priority pathogen by the World Health Organization (WHO) that urgently requires development of new antimicrobials to combat the emerging public health risk posed by antimicrobial resistance (Muller, et al., 2023 ; de Souza et al., 2025).

Resistance of A. baumannii to carbapenems is mainly due to expression of genes encoding for carbapenem-hydrolysing enzymes, especially class D β-lactamases (oxacillinases) and metallo-β-lactamases. The blaOXA-23 gene, which encodes OXA-23 type carbapenemase, is one of the most frequently documented determinants worldwide. OXA-23 imparts high-level resistance to the carbapenems and is commonly linked to epidemic international clones, which disseminate in hospital settings (Papadopoulou et al., 2024). Its spread is frequently promoted by mobile genetic elements including transposons that mediate rapid dissemination between isolates, which supports the maintenance and transmission of CRAB genera (Odhafa et al., 2024).

The blaNDM gene types specify for New Delhi metallo-β-lactamases (NDMs) that are zinc-dependent β-lactamases with activity for nearly all β-lactams (including carbapenems) and no such effect on monobactams. blaNDM was originally found in Enterobacterales and has spread to *A. baumannii* and other gram-negative strains, representing an unprecedented expansion of carbapenemase-driven resistance beyond class D serine Î²-lactamases (Rahbarnia et al., 2020). The combination of blaNDM and blaOXA-23 into a single isolate is less frequent however, it has been reported in several countries from different geography including North Africa, Europe and Asia most are associated with extensively drug-resistant phenotypes against which clinicians have limited therapeutic options. These double carbapenem-resistant genotypes would restrict therapeutic options and lower the activity of last-resort antibiotics, and highlight an increasing complexity in resistance profiles in CRAB strains (Ramoul et al., 2016).

High prevalence of carbapenem resistance genes in A. baumannii clinical strains isolated from the Middle East, North America and Central Asia/Asia-Pacific conducted by others including Iraq regional, has been previously reported. Recent Iraqi surveillance informs us of the prevalence of blaOXA-23 in clinical and environmental hospital isolates with frequently association with global clone lineages GC1 (Global Clone 1) and GC2 (Global Clone 2) known to be major drivers for international CRAB spread (Odhafa et al., 2024). Simultaneous studies on Babylon and Al-Najaf areas of Iraq also described the very high rate of blaNDM and other OXA-type carbapenemase genes in clinical isolates, which marked a complicated pattern of resistance genes that can promote the emergence of strains

resistant to most antibiotics with untreatable features in hospital environment (Alkhawaja & Hadi, 2025). blaOXA-23 is almost always detected in the molecular epidemiology studies of infections caused by CRAB, while blaNDM is identified with variable frequency, thereby underscoring the importance of region-specific surveillance and molecular typing to guide antibiotic stewardship and infection control practices (Kilbas et al., 2023).

This study aims to investigate the molecular prevalence of carbapenem resistance genes blaNDM and blaOXA-23 in clinical Acinetobacter baumannii isolates from sputum specimens of Iraqi patients. They aim at studying the spread of these resistance determinants and assess their implication in the acquisition of carbapenem resistance. The study also aims to generate knowledge to help toward explaining the molecular mechanisms by which multidrug resistance is mediated in respiratory A. baumannii infections in Iraq.

Patients and Methods

Study Design and Setting

A cross-sectional hospital-based study was carried out in the Teaching Hospital of Al-Diwaniya City, Iraq, during a period of six months (March 2025 and August 2025). In total, 48 patients habitually diagnosed as having lower respiratory infections and admitted to respiratory or internal medicine wards during the period of study were included. Diagnosis relied on clinical, radiological and laboratory routine findings. Inclusion criteria All adults, both adult male and female patients whose sputum specimens have confirmed bacterial isolation of *Acinetobacter baumannii*. Patients who were treated with systemic antimicrobial therapy within 72 h of specimens collection were excluded to reduce the effect on bacterial isolation and resistance patterns. Cases where complete clinical data was not available were also removed.

Sample Collection and Bacterial Identification

Aseptic sputum specimens were obtained in the early morning from all patients before initiation of antibiotic therapy. All the samples were collected in sterile, wide mouthed containers using standard sputum collection techniques for minimizing oral flora contamination. Negative high-quality specimens (low numbers of epithelial cells and large amounts of polymorphonuclear leukocytes after microscopic exam) were the only ones that processed. The specimens were spread on blood agar plates (Oxoid Ltd., UK) and MacConkey agar which were then incubated aerobically at 37°C for 24-48 hours. Presumptive identification of A. baumannii was done according to the colony morphology, Gram staining (Gram-negative coccobacilli), non-lactose fermentation on MacConkey Agar, oxidase negative nature and standard biochemical tests. The results of these experiments were confirmed by standard biochemical procedures. The proved isolates were stored in the brain heart infusion broth containing glycerol at –80°C, and stored for molecular analyses. ATCC® 19606™ was included as quality control.

Antimicrobial Susceptibility Testing

Antibiotic susceptibility profile was specified by Kirby–Bauer disk diffusion method on Mueller–Hinton agar, according to Clinical and Laboratory Standards Institute (CLSI) recommendations (2024). Antimicrobial agents that were chosen belonged to various classes of drugs for the antimicrobial therapy of bacterial infections caused by bacteria that react negatively to gram staining:

Imipenem (10 µg)

Meropenem (10 µg)

Amikacin (30 µg)

Gentamicin (10 µg)

Ciprofloxacin (5 µg)

Ceftazidime (30 µg)

Piperacillin–tazobactam (100/10 µg)

The diameter of inhibition zone was read after incubation for 18–24 h at 37°C and explained as intermediate, susceptible, or resistant by the CLSI breakpoints. MDR was considered as non-susceptibility acquired to at least one antimicrobial from three or more classes (Magiorakos et al., 2012). A. baumannii ATCC® 19606™ was used for quality control testing.

DNA Extraction

Genomic DNA of all confirmed *A. baumannii* isolates was extracted with GeneJET Genomic DNA Purification Kit (Thermo Fisher Scientific, Lithuania) based on the manufacturer's instructions. The concentration and purity of DNA were evaluated by utilizing a spectrophotometer, and DNA samples were stored frozen at –20°C until molecular testing.

Molecular Identification of blaNDM and blaOXA-23 Genes

PCR method (not real time) was utilized to ascertain the existence of carbapenemase-encoding genes, blaNDM, and blaoxa-23 among the isolates. Gene amplification was performed with previously reported specific primers. For PCR amplification, the final reaction volume was 25 µL which included: 12.5 µL of 2X PCR Master Mix (Promega USA), 1 µL (10 pmol/µL) of each forward and reverse primer, 3 µL template DNA, and water free of nuclease to make up the volume.

The reactions have been repeated in a thermal cycler (Eppendorf Mastercycler, Germany) as follows: An initial denaturation of 94°C for 5 min. 35 cycles of:Denaturation was performed at 94°C for 30 s . Annealing (40 s at 55 or 58°C depending on primer specificity) on at 72°C for 45 sec. 7 min at 72°C for last extension

The PCR products were separated in 1.5% agarose gels stained with ethidium bromide (0.5 µg/mL) and images taken under ultraviolet light using a gel documentation system (Bio-Rad, USA). The detection of blaNDM and blaOXA-23 genes was verified through amplicon sizes obtained that matched those of the targeted fragments (621 bp and 501 bp). The positive control strains, which were confirmed to contain these gene sequences, were also included in each run; water free of nuclease was used as the negative control.

Ethical Considerations

The study was recommended by the Scientific and Ethical Committee at Teaching Hospital in Al-Diwaniya City. All the participants signed written informed consent before entering this study. Patient anonymity was preserved and all information was collected for research purpose only.

Table1: List of primers applied for amplification of blaNDM and blaOXA-23 genes

Gene	Amplicon size (bp)	Annealing temp (°C)	Primer sequence (5′–3′)
blaNDM	621 bp	52–55°C	F: GGTTTGGCGATCTGGTTTTC R: CGGAATGGCTCATCACGATC
blaOXA-23	501 bp	52–55°C	F: GATCGGATTGGAGAACCAGA R: ATTTCTGACCGCATTTCCAT

Results

The results show a very high resistance to carbapenem: 87.5% and 85.4% for imipenem and meropenem, respectively which confirms the wide spread diffusion of *A. baumannii* producing carbapenemasas strains . Rates of β-lactam/β-lactamase inhibitor combinations and resistance to third-generation cephalosporins were also high, effectively restricting the armamentarium of available therapies. There was relatively lower level of resistance, especially to amikacin, indicating partial activity still retained to some isolates. High resistance against fluoroquinolones was preserved, representing the multiresistant phenotype which was frequently described in hospital- related *A. baumannii* (Table 2).

Table 2. Rates of antibiotic resistance reported in the *Acinetobacter baumannii* isolates

Groups	Resistant Isolates No. (%)	Susceptible Isolates No. (%)
Imipenem	42 (87.5%)	6 (12.5%)
Meropenem	41 (85.4%)	7 (14.6%)
Amikacin	30 (62.5%)	18 (37.5%)
Gentamicin	33 (68.8%)	15 (31.2%)
Ciprofloxacin	36 (75.0%)	12 (25.0%)
Ceftazidime	39 (81.3%)	9 (18.7%)
Piperacillin–tazobactam	38 (79.2%)	10 (20.8%)

The blaOXA-23 was the predominant gene found (n = 5/6; 83.3%) through molecular characterization, suggesting that an OXA-23 carbapenemase is the prevalent mechanism of resistance to carbapenems in this population. blaNDM was however identified in 37.5% of isolates indicating a high yet relatively less prevalence of metallo-β-lactamase resistance (Table 3).

Table 3. Frequency and percentage of blaNDM and blaOXA-23 genes in *Acinetobacter baumannii* isolates (n=48)

Groups	Positive No. (%)	Negative No. (%)
blaNDM	**18 (37.5%)**	**30 (62.5%)**
blaOXA-23	**40 (83.3%)**	**8 (16.7%)**

There was a strong correlation of carbapenem resistance (imipenem and meropenem) with blaNDM as well as blaOXA-23 genes ($P < 0.01$), substantiating their importance in the hydrolysis of carbapenems. It was particularly strong for blaOXA-23, which was more common than the other genes among isolates. Resistance to aminoglycosides and fluoroquinolones, however, was not significantly associated with blaNDM and only weak or borderline for blaOXA-23, suggesting that these phenotypes are driven by other mechanisms. Moderate but significant association was also observed between ceftazidime and piperacillin–tazobactam resistance, possible suggestion co-carriage of resistance determinants in the same mobile genetic elements with blaOXA-23. These results, in general, support a biologically based pattern of antibiotic resistance that is consistent with the molecular epidemiology of carbapenem-resistant A. baumannii.

Table 4. Association between antibiotic resistance and presence of blaNDM and blaOXA-23 genes in *Acinetobacter baumannii* isolates

Groups	blaNDM Chi Square (P value)	blaOXA-23 Chi Square (P value)
Imipenem	**9.82 (0.002)****	**22.41 (0.0000)****
Meropenem	**8.74 (0.003)****	**20.95 (0.0000)****
Amikacin	**1.94 (0.163)**	**2.76 (0.096)**
Gentamicin	**2.48 (0.115)**	**3.91 (0.048)***
Ciprofloxacin	**0.89 (0.345)**	**2.11 (0.146)**
Ceftazidime	**3.87 (0.049)***	**7.52 (0.006)***
Piperacillin–tazobactam	**3.12 (0.077)**	**6.48 (0.011)***

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

Discussion

The degree of antimicrobial resistance in *A. baumannii*' isolated from sputum samples of patients referred to Al-Diwaniyah Teaching Hospital was relatively high in this study. The proportion of resistance to carbapenems was statistically higher, 87.5% and 85.4% for imipenem and meropenem, respectively. These results agree with international publications which have identified CRAB as an important nosocomial pathogen representing a major threat and a cause of increased mortality due to the limited range of available therapeutic strategies (Tacconelli et al., 2018; Wong et al., 2017). CRAB has been listed by the WHO among priority research and development of new antimicrobials due to it rapid spread and resistance potential (Tacconelli et al., 2018).

The molecular data of all isolates demonstrated the detection of blaOXA-23 in 83.3 % of *A. baumannii* isolates as the most abundant carbapenemase gene. This is consistent with many both local and foreign studies which have demonstrated that OXA-23 is the most common carbapenem-hydrolyzing enzyme in *A. baumannii* (Poirel & Nordmann, 2006; Evans & Amyes, 2014). BlaOXA-23 has also been frequently detected among CRAB isolates in other Middle Eastern countries such as Iraq, which is usually accompanied by international clones such as GC1 and GC2 enhancing its hospital spread (Alkhawaja & Hadi, 2025). The dominance of blaOXA-23 in the present work accounts for this significant statistical correlation between carbapenem resistance and gene carriage, especially against imipenem and meropenem (P 40%) several folds among different region and hospital settings observed from regional studies. The recovery of

blaNDM in over one-third of isolates described here reflects the continued spread of this resistance gene among Iraqi healthcare institutions (Alkhawaja & Hadi, 2025; Poirel et al., 2011).

In addition to carbapenems, they showed high resistance rates for ceftazidime (81.3%), piperacillin–tazobactam (79.2%) and ciprofloxacin (75%). These results are consistent with the multidrug-resistant (MDR) phenotype frequently observed in CRAB strains. These resistance profiles are also common globally, with CRAB isolates often being resistant to cephalosporins, fluoroquinolones and aminoglycosides as a result of acquisition of multiple resistance mechanisms (Wong et al., 2017). Resistance to aminoglycosides in this research was relatively lower (62.5% towards amikacin, 68.8% for gentamicin), Signifying partial activity retention; nevertheless, resistance is still significant. In *A. baumannii*, the resistance to aminoglycosides is frequently associated with both genes encoding aminoglycoside-modifying enzymes and 16S rRNA methylases other than carbapenemase production (Poirel et al., 2011).

The polymorphism-based association test between the carbapenem resistance, and blaOXA-23 and blaNDM genes were highly significant. This is biologically feasible since they are the genes encoding the enzymes responsible for hydrolysis of carbapenems. By contrast, the weak or steady associations found in flow tests between aminoglycosides/fluoroquinolones and either blaNDM or blaOXA-23 showed that under non-β-lactam agents' pressures resistance was accomplished through other pathways than β-lactamases; namely efflux transporters, target modification mutants and permeability defects. This pattern is in line with the multifactorial basis of multidrug resistance in *A. baumannii* (Evans & Amyes, 2014). The high incidence of carbapenemase genes in this study highlights the urgent requirement for improved antimicrobial stewardship and infection control practices. CRAB infections are related to longer hospitalisation, higher cost of care and increased mortality. Ongoing molecular surveillance will be crucial to track the dissemination of resistance genes and aid clinicians in making empiric therapy decisions. In addition, early identification of blaOXA-23 and blaNDM by the molecular approach could contribute to prompt infection control practices and diminish hospital outbreak (Wong et al., 2017).

Conclusion

This study demonstrates the predominance of blaOXA-23 and high prevalence of blaNDM among carbapenem-resistant *A. baumannii* isolates in Al-Diwaniyah. The high correlation of carbapenem resistance with presence of carbapenemase-encoding genes that we observed underlines the molecular mechanism of resistance and mirrors regional epidemiology. These data provide additional evidence that CRAB remains a major public health problem which requires unified local and global efforts for control.

Reference

1. Alkhawaja, A. A. K., & Hadi, Z. J. (2025). High Prevalence of blaNDM and blaOXA Carbapenemases in Multiple Antibiotic Resistant Acinetobacter baumannii from Iraqi Hospital. *Kufa Medical Journal*, *21*(2), 30-41. https://doi.org/10.36330/kmj.v21.i2.19906
2. de Souza, J., D'Espindula, H. R. S., Ribeiro, I. d. F., Gonçalves, G. A., Pillonetto, M., & Faoro, H. (2025). Carbapenem Resistance in Acinetobacter baumannii: Mechanisms, Therapeutics, and Innovations. Microorganisms, 13(7), 1501. https://doi.org/10.3390/microorganisms13071501
3. Evans, B. A., & Amyes, S. G. (2014). OXA β-lactamases. *Clinical microbiology reviews*, *27*(2), 241–263. https://doi.org/10.1128/CMR.00117-13
4. Kilbas, E. P. K., Kilbas, I., & Ciftci, I. H. (2023). Molecular epidemiology of carbapenem-resistant *Acinetobacter baumannii* isolates in Turkiye: Systematic review. *Northern clinics of Istanbul*, *10*(4), 531–539. https://doi.org/10.14744/nci.2022.17003
5. Magiorakos, A. P., Srinivasan, A., Carey, R. B., Carmeli, Y., Falagas, M. E., Giske, C. G., Harbarth, S., Hindler, J. F., Kahlmeter, G., Olsson-Liljequist, B., Paterson, D. L., Rice, L. B., Stelling, J., Struelens, M. J., Vatopoulos, A., Weber, J. T., & Monnet, D. L. (2012). Multidrug-resistant, extensively drug-resistant and pandrug-resistant bacteria: an international expert proposal for interim standard definitions for acquired resistance. *Clinical microbiology and infection : the official publication of the European Society of Clinical Microbiology and Infectious Diseases*, *18*(3), 268–281. https://doi.org/10.1111/j.1469-0691.2011.03570.x
6. Müller, C., Reuter, S., Wille, J., Xanthopoulou, K., Stefanik, D., Grundmann, H., Higgins, P. G., & Seifert, H. (2023). A global view on carbapenem-resistant *Acinetobacter baumannii*. *mBio*, *14*(6), e0226023. https://doi.org/10.1128/mbio.02260-23
7. Odhafa, M. W., Al-Kadmy, I., Pourmand, M. R., Naderi, G., Asadian, M., Ghourchian, S., & Douraghi, M. (2024). The context of bla_{OXA-23} gene in Iraqi carbapenem-resistant Acinetobacter baumannii isolates belonging to global clone

1 and global clone 2. *BMC research notes*, *17*(1), 300. https://doi.org/10.1186/s13104-024-06890-w

8. Papadopoulou, M., Deliolanis, I., Polemis, M., Vatopoulos, A., Psichogiou, M., & Giakkoupi, P. (2024). Characteristics of the Genetic Spread of Carbapenem-Resistant Acinetobacter baumannii in a Tertiary Greek Hospital. Genes, 15(4), 458. https://doi.org/10.3390/genes15040458
9. Poirel, L., Bonnin, R. A., & Nordmann, P. (2011). Genetic basis of antibiotic resistance in pathogenic Acinetobacter species. *IUBMB life*, *63*(12), 1061–1067. https://doi.org/10.1002/iub.532
10. Rahbarnia, L., Farajnia, S., Khaneshi, H., Farajnia, H., Naghili, B., & Tanomand, A. (2020). Detection of blaOXA-23 and blaNDM-1 carbapenemase among clinical isolates of A. baumannii in Tabriz, north-west of Iran. *Gene Reports*, *18*, 100555.
11. Ramoul, A., Loucif, L., Bakour, S., Amiri, S., Dekhil, M., & Rolain, J. M. (2016). Co-occurrence of blaNDM-1 with blaOXA-23 or blaOXA-58 in clinical multidrug-resistant Acinetobacter baumannii isolates in Algeria. *Journal of global antimicrobial resistance*, *6*, 136–141. https://doi.org/10.1016/j.jgar.2016.05.003
12. Tacconelli, E., Carrara, E., Savoldi, A., Harbarth, S., Mendelson, M., Monnet, D. L., Pulcini, C., Kahlmeter, G., Kluytmans, J., Carmeli, Y., Ouellette, M., Outterson, K., Patel, J., Cavaleri, M., Cox, E. M., Houchens, C. R., Grayson, M. L., Hansen, P., Singh, N., Theuretzbacher, U., … WHO Pathogens Priority List Working Group (2018). Discovery, research, and development of new antibiotics: the WHO priority list of antibiotic-resistant bacteria and tuberculosis. The Lancet. Infectious diseases, 18(3), 318–327. https://doi.org/10.1016/S1473-3099(17)30753-3
13. Thacharodi, A., Vithlani, A., Hassan, S., Alqahtani, A., & Pugazhendhi, A. (2024). Carbapenem-resistant *Acinetobacter baumannii* raises global alarm for new antibiotic regimens. *iScience*, *27*(12), 111367. https://doi.org/10.1016/j.isci.2024.111367
14. Wong, D., Nielsen, T. B., Bonomo, R. A., Pantapalangkoor, P., Luna, B., & Spellberg, B. (2017). Clinical and Pathophysiological Overview of Acinetobacter Infections: a Century of Challenges. *Clinical microbiology reviews*, *30*(1), 409–447. https://doi.org/10.1128/CMR.00058-16