Vol. 06, No. 02, pp. 64 –69 (2025)
**ISSN: 2708-1818**

JOURNAL OF LIFE AND BIO-SCIENCES RESEARCH

www.jlbsr.org

# Antibiotic Resistance in *Klebsiella pneumoniae* in Iraq: A Narrative Review

Nawfal R Hussein[1,*], Alan A Muhammad[1], Halder J Abozait[2]

[1]*Department of Biomedical Sciences, College of Medicine, University of Zakho, Kurdistan Region, Iraq (nawfal.hussein@uoz.edu.krd, alan.20027033@stud.uoz.edu.krd)*
[2]*Department of Medicine, College of Medicine, University of Duhok, Duhok, Kurdistan Region, Iraq (halder01jamal@gmail.com)*
https://doi.org/10.38094/jlbsr601154

*Correspondence: nawfal.hussein@uoz.edu.krd

## *Abstract*

**Antibiotic resistance in *Klebsiella pneumoniae* (*K. pneumoniae*) poses a significant public health challenge globally, with Iraq experiencing a notable rise in multidrug-resistant strains. This narrative review evaluates current evidence on antibiotic resistance patterns and genetic mechanisms underlying resistance in *K. pneumoniae* isolates across Iraq. The review highlights a high prevalence of β-lactam resistance, particularly to penicillins and cephalosporins, largely driven by extended-spectrum β-lactamase (ESBL) genes such as *bla*$_{SHV}$, *bla*$_{TEM}$, and *bla*$_{CTX-M}$. Carbapenem resistance is alarmingly increasing and is mediated primarily by carbapenemase genes, including *bla*$_{OXA-48}$, *bla*$_{NDM}$, and *bla*$_{VIM}$, with the *bla*$_{KPC}$ gene emerging in select regions. Aminoglycoside resistance remains moderate, with amikacin retaining efficacy, though resistance genes like *strA* and *strB* contribute to variability. Quinolone resistance is attributed to chromosomal mutations, efflux pumps, and plasmid-mediated genes such as *qnrS* and *qnrB*, with resistance rates varying significantly by region and clinical setting. The dissemination of resistance is further facilitated by efflux pumps, including acrAB, mdtK, and tolC and porin loss (ompK35, ompK36). Despite increased research, gaps remain due to limited sample sizes and regional disparities in individual studies. To combat this escalating threat, the review advocates for a coordinated national surveillance system, stringent antibiotic stewardship, enhanced infection control measures, and wider implementation of molecular diagnostics. Addressing these challenges is crucial to controlling the spread of resistant *K. pneumoniae* and preserving the efficacy of existing antibiotics in Iraq.**

***Keywords*:** *Antibiotic resistance, Sensitivity, Klebsiella pneumoniae, β-lactamase, Carbapenemase, Iraq.*
Received: June 9th, 2025/ Revised: July 10th, 2025/ Accepted: July 15th, 2025/ Online: July 16th, 2025

## I. INTRODUCTION

The increase in antibiotic resistance is a public health issue worldwide (Naghavi *et al*., 2024). Previous reports showed a high prevalence of antibiotic resistance in Iraq (Abduljabar and Naqid, 2022; Abdulkareem *et al.*, 2020; Al-Brefkani *et al.*, 2023; Hussein *et al.*, 2025). *Klebsiella pneumoniae* (*K. pneumoniae*) is a Gram-negative microorganism that causes a wide range of infections, including skin and soft tissue infections and life-threatening sepsis (Huy, 2024; Naqid *et al*., 2020). This bacterium can survive on hospital surfaces and infect many body tissues (Huy, 2024). Consequently, the opportunistic infection can readily transmit among patients (Ristori *et al*., 2024). Multiple factors have enhanced the development of multidrug-resistant strains in healthcare environments, including inappropriate antibiotic use and horizontal transfer of resistance genes (Huy, 2024; Ristori *et al*., 2024). Several genes that encode unique virulence factors enable *K. pneumoniae* to evade the immune system and cause various diseases. Such virulence factors include iron acquisition systems and adhesins (Riwu *et al.*, 2022). The prevalence of antibiotic resistance among *K. pneumoniae* samples is increasing and has evolved through various strategies, including the acquisition and transference of resistance genes (Huy, 2024). Epidemiological studies indicate a notable level of resistance in *K. pneumoniae* strains derived from nosocomial infections to frequently prescribed categories of antibiotics, such beta-lactam, aminoglycosides,



*doi: 10.38094/jlbsr602162*

and quinolone (Huy, 2024). The patterns and distribution of antibiotic resistance differ from one country to another.
Regional data are crucial at both local and international levels to employ targeted strategies to tackle the growing problem (Hussein *et al.*, 2025; Oliveira *et al.*, 2024). A significant amount of research is taking place in Iraq to explore the genetic elements linked to the drug resistance of *K. pneumoniae* bacteria. Nonetheless, most of these studies utilized small sample sizes. This research seeks to gather all existing studies on antibiotic resistance patterns and their related genetic influences. It also aims to address the gaps and weaknesses in the existing studies.

## II. Methods

This review was conducted about *K. pneumoniae*, aiming at determining antibiotic resistance patterns and related genetic factors. To focus on relevant studies, "*Klebsiella pneumoniae*," "antibiotic," "Iraq," and "genes" were used as keywords for the search, and PubMed was utilized as a primary database. Our search was limited to articles published in English, and studies focused on *K. pneumoniae* in Iraq were included. Following the search, the titles and abstracts of the retrieved articles were first screened for relevance. Articles were then assessed for their suitability within the scope of this review. Studies unrelated to *K. pneumoniae* resistance in Iraq, review articles lacking primary data, and studies focused on other Klebsiella species were excluded.

## III. Beta-Lactam Resistance In *K. Pneumoniae*

β-lactamase enzymes are one of the mechanisms that enable *K. pneumoniae* to resist a wide range of β-lactam antibiotics, including penicillins, cephalosporins, and carbapenems (Santajit and Indrawattana, 2016; Singh *et al*., 2011). The impact of such enzymes will be discussed in this review.

### *A. Penicillin and cephalosporin resistance*

A study investigating the antibiotic sensitivity pattern of *K. pneumoniae* in a variety of clinical specimens in northern Iraq showed that resistance to penicillin such as ampicillin approaches 100% in most samples. Such a resistance was primarily due to the bacterial production of penicillinases (Naqid *et al*., 2020). In another study conducted in the Burn unit in Baghdad city, the tested isolates were resistant to penicillin such as piperacillin (Hammoudi and Hussein, 2018) (Table 1). In another study conducted in pediatric and gynecology hospitals in Babylon and Karbala Provinces, $bla_{\text{OXA-1}}$ gene, which encodes a penicillin-hydrolyzing beta-lactamase was found in 94% of the studied samples (Abbas, 2021). In a study conducted in Najaf in 2017, recruiting 43 *K. pneumoniae* isolates from hospitalized patients with urinary tract infections and burns, the vast majority of the strains (98%) were resistant to amoxicillin and amoxicillin / clavulanic acid. In the same study $bla_{\text{SHV}}$ was the most common gene responsible for resistance followed by $bla_{\text{IMP}}$ at a prevalence of 86.04% and 9.30%, respectively (Aljanaby and Alhasnawi, 2017) (Table 2).

### *B. Cephalosporin resistance*

While in a study conducted in Duhok 65.8% of *K. pneumoniae* isolates were resistant to ceftriaxone, 85% of the isolates were resistant to ceftriaxone in a study conducted on burn patients in Baghdad (Hammoudi and Hussein, 2018; Naqid *et al*., 2020). In a large comprehensive study involving 60 *K. pneumoniae* isolates from three hospitals in Baghdad in 2022, resistance was observed in 85% to cefazolin and ceftazidime, 80% to ceftriaxone and cefepime (48/60), and 57% to cefoxitin (34/60) (Muhsin *et al*., 2022) (Table 1). In the same study, *MFS* and *MdtK* efflux pump genes were found in 88% of cephalosporin-resistant microorganisms. Besides, *CfiaS* and *CfiaL* were found in 85.7% of the strains resistant to cephalosporin (Muhsin *et al*., 2022). Additionally, *OmpK-35* and *OmpK-36* were found in 85% and 25%, respectively (Muhsin *et al*., 2022) (Table 2). In another study conducted in different hospitals in Sulaimani in 2022, the vast majority of *K. pneumoniae* were resistant to cephalosporins, including ceftazidime, ceftriaxone, and cefotaxime (more than 90%). In the same study, $bla_{\text{SHV}}$ was detected in 92.85% of isolates, and $bla_{\text{TEM}}$, found in 53.57% (Mohammed and Anwar, 2022). Another study in Erbil investigated the genetic makeup of resistance strains in a thalassemia center, isolates from inpatients and outpatients carried $bla_{\text{TEM}}$ (64.7%), $bla_{\text{CTX-M}}$ (41.1%), and $bla_{\text{SHV}}$ (35.2%) genes (Pishtiwan and Khadija, 2019). In another study that was conducted in 2022 on a variety of inpatient and outpatient specimens, molecular genotyping of resistant microorganisms detected $bla_{\text{CTX-M}}$ in 64.81% of isolates, followed by $bla_{\text{TEM}}$ in 40.74% and $bla_{\text{SHV}}$ in 35.19% (Bakr *et al*., 2022) (Table 2).

In a recent study conducted on inpatients in Al-Anbar Governorate in 2024, 20% of the isolates were metallo-lactamase producers, and 41.81% tested positive for extended-spectrum beta-lactamases (ESBLs). The same study found efflux pump genes, *acrAB*, *mdtK*, and *tolC* in 94.54 %, 14.54 %, and 89.09 % of the isolates, respectively, while the porin-encoding genes, *ompK35* and *ompK36*, were found in 96.36 % and 98.18 % of the isolates (Hussein *et al*., 2024). In a study conducted on wound samples in AL-Diwaniyah province, Iraq in 2022, Ceftazidime resistance was 59.7% and Cefotaxime resistance was 50.5%. Additionally, the *acrA* and *acrB* genes were both carried in 100% of the isolates (Abid Fazaa, 2023). In a study conducted in Iraq in 2022 investigating ESBLs among *K. pneumoniae* in patients with community-acquired pneumonia, 47.4% and 15.8% of the isolates carried $bla_{\text{CTX-M}}$ and $bla_{\text{SHV}}$, respectively (Raouf *et al*., 2022). In another study conducted in Najaf in 2022 investigating the genetic makeup of resistance using isolates from urine samples, 32% of samples carried the $bla_{\text{SHV-1}}$ gene (Mansor *et al*., 2022) (Tables 1 and 2).

### *C. Carbapenem resistance*

Carbapenem resistance in *K. pneumoniae* mostly arises via acquired carbapenem-hydrolyzing β-lactamases, with *K. pneumoniae* carbapenemases (KPCs) being the most prevalent. The production of certain enzymes, such as KPC

enzymes, has enabled *K. pneumoniae* to acquire resistance to beta-lactam antibiotics. These enzymes are frequently seen in *Klebsiella* species. ESBLs, especially those belonging to the CTX-M family, are prevalent in *K. pneumoniae*, resulting in resistance to extended-spectrum beta-lactams. There are variations in resistance mechanisms across Enterobacteriaceae, particularly *K. pneumoniae*.

The emergence of Class B carbapenemases, including VIM, IMP, and NDM types, facilitates the proliferation of pan-resistant strains. These enzymes are progressively recognized in *K. pneumoniae* isolates (Abduljabar and Naqid, 2022). A 2019 study in Erbil found no carbapenem resistance isolates, however, a more recent 2023 study on inpatients with respiratory and urinary tract infections revealed a 7.5% rate of resistance to imipenem and 12.2% to meropenem (Mohammed *et al*., 2023; Pishtiwan and Khadija, 2019). Furthermore, a 2020 study from Duhok indicated carbapenem resistance rates as high as 90% (Naqid *et al*., 2020). In contrast, the imipenem resistance rate was 26% in Ramadi province in 2022 (Mohammed *et al*., 2023) (Table 1).

Table 1. Summary of beta-lactam resistance patterns in *K. pneumoniae* in Iraq

| Resistance Type | Resistance Rate | Location | Year |
|---|---|---|---|
| Penicillin (ampicillin) | 100% | Iraq (general) | 2020 |
| Penicillin (piperacillin) | High | Baghdad | 2018 |
| Penicillin (amoxicillin and amoxicillin / clavulanic acid) | 98% | Najaf | 2017 |
| Cephalosporin (ceftriaxone) | 65.8% | Duhok | 2020 |
| Cephalosporin (ceftriaxone) | 85% | Baghdad | 2018 |
| Cephalosporin (cefazolin, ceftazidime) | 85% | Baghdad | 2022 |
| Cephalosporin (ceftriaxone, cefepime) | 80% | Baghdad | 2022 |
| Cephalosporin (cefoxitin) | 57% | Baghdad | 2022 |
| Cephalosporin (general resistance) | 90% | Sulaimani | 2022 |
| Ceftazidime resistance | 59.7% | AL-Diwaniyah | 2022 |
| Cefotaxime resistance | 50.5% | AL-Diwaniyah | 2022 |
| Carbapenem (imipenem) | 0% | Erbil | 2019 |
| Carbapenem (meropenem) | 0% | Erbil | 2019 |
| Carbapenem (general) | 90% | Duhok | 2020 |
| Carbapenem (imipenem) | 26% | Ramadi | 2022 |
| Carbapenem (imipenem) | 7.5% | Erbil | 2022 |
| Carbapenem (meropenem) | 12.2% | Erbil | 2022 |

Carbapenemases are classified into two types based on their amino acid sequences: metallo-β-lactamases (Class B), which contain zinc in the active site, and serine-β-lactamases (Classes A, C, and D), which contain serine in the active site. Molecular investigations confirm that *K. pneumoniae* isolates in Iraq produce a range of carbapenemases, including OXA-48 family (class D carbapenemase), NDM (New Delhi metallo-β-lactamase) and VIM (Verona Integron-encoded Metallo-β-lactamase) enzymes being the most prominent. In 2022, a study conducted in Karbala found that $bla_{\text{OXA-48}}$ gene was present in 45% of the isolates, $bla_{\text{NDM}}$ gene in 30% of isolates and $bla_{\text{VIM}}$ in 60% of isolates (Ibraheim *et al*., 2024). Conversely, the KPC class A enzyme remains relatively rare in Iraq and only found in 2% of the isolates (Ibraheim *et al*., 2024). In another study conducted on different clinical samples of hospitalized patients in Baghdad in 2022, all the isolates were resistant to ertapenem, meropenem and imipenem. In the same study, it was shown that 80.9% carbapenem-resistant *K. pneumoniae* isolates harbored $bla_{\text{KPC}}$ gene, while none carried other carbapenemase genes including $bla_{\text{NDM}}$, $bla_{\text{VIM}}$ and $bla_{\text{IMP}}$ (Hussein *et al*., 2022) (Table 2).

Table 2. Summary of beta-lactam resistance genes in *K. pneumoniae* in Iraq

| Resistance Gene | Resistance Rate | Location | Year |
|---|---|---|---|
| Penicillinase gene - $bla_{\text{OXA-1}}$ | 94% | Babylon and Karbala | 2021 |
| Carbapenemase gene - $bla_{\text{OXA-48}}$ | 45% | Karbala | 2022 |
| Carbapenemase gene - $bla_{\text{NDM}}$ | 30% | Karbala | 2022 |
| Carbapenemase gene - $bla_{\text{VIM}}$ | 60% | Karbala | 2022 |
| Carbapenemase gene – $bla_{\text{KPC}}$ | 2% | Karbala | 2022 |
| Carbapenemase gene - $bla_{\text{KPC}}$ | 80.9% | Baghdad | 2022 |
| ESBL genes – $bla_{\text{TEM}}$ | 64.7% | Erbil | 2016 |
| ESBL genes - $bla_{\text{CTX-M}}$ | 41.1% | Erbil | 2016 |
| ESBL genes – $bla_{\text{SHV}}$ | 35.2% | Erbil | 2016 |
| ESBL gene - $bla_{\text{SHV}}$ | 32% | Najaf | 2022 |
| ESBL genes - $bla_{\text{CTX-M}}$ | 47.4% | Iraq (CAP) | 2022 |
| ESBL genes – $bla_{\text{SHV}}$ | 15.8% | Iraq (CAP) | 2022 |
| Porin genes - *ompK35* | 96.36% | Iraq (general) | 2024 |
| Porin genes - *ompK36* | 98.18% | Iraq (general) | 2024 |
| Efflux pump gene - *acrA* | 100% | Iraq (general) | 2024 |
| Efflux pump gene - *acrB* | 100% | Iraq (general) | 2024 |
| Efflux pump gene - *tolC* | 89.09% | Iraq (general) | 2024 |
| Efflux pump gene - *MFS* | 88% | Baghdad | 2022 |
| Efflux pump gene - *MdtK* | 14.54% | Baghdad | 2022 |
| Cephalosporin resistance genes - *CfiaS* | 85.7% | Baghdad | 2022 |
| Cephalosporin resistance genes - *CfiaL* | 85.7% | Baghdad | 2022 |

## IV. Aminoglycoside Resistance

Aminoglycosides, such as gentamicin, tobramycin, and amikacin, are frequently utilized in combination therapy for *K. pneumoniae* infections (Sajerli *et al.*, 2025). Despite significant resistance that appears to vary by region and climate, amikacin and gentamicin have mostly maintained moderate efficacy against *K. pneumoniae* in Iraq over the past decade. In a study conducted in Baghdad recruiting samples from burn unit, 19% of isolates exhibited resistance to amikacin, compared to 26% which exhibited resistance to gentamicin (Hammoudi and Hussein, 2018). In another study conducted in Ramadi province, it was found that only 16% of *K. pneumoniae* isolates were resistant to amikacin, highlighting it as one of the most effective treatment options (Mohammed *et al*., 2023). Regarding gentamicin, the resistance rates varied from 19% in Duhok city to more than 50% in Baghdad (Mustafa and Abdullah, 2020; Naqid *et al*., 2020). In the 2016 Baghdad burn unit study, gentamicin demonstrated good activity, with 74% of isolates remaining susceptible (Hammoudi and Hussein, 2018). The data show that while community-acquired or non-critical isolates may remain sensitive, aminoglycoside resistance is context-dependent with higher resistance shown in ICU and invasive infections. In a study conducted in AL-Diwaniyah province, gentamicin resistant rate was 18.3% (Abid Fazaa, 2023) (Table 3).

One study aimed to investigate the genetic basis of the resistance to aminoglycoside among hospitals and burn centers in Najaf, Iraq. The *strA* and *strB* genes encode aminoglycoside-modifying enzymes, specifically streptomycin phosphotransferases. These enzymes confer resistance specifically to streptomycin, which is an older aminoglycoside. There was a high rate of *strA* and *strB* genes

among *K. pneumoniae* isolates, reaching 71.4% compared with 7.14% and 3.57% for *aacC-1* and *aac(6')/aph(2')* genes respectively. In the same study, *aacC-2*, *aph(3')-IIIa* and *ant(4')-Ia* were not identified in the isolates (Tuwaij *et al*., 2019) (Table 3).

Table 3. Summary of aminoglycoside resistance in *K. pneumoniae* in Iraq

| Resistance Type | Resistance Rate | Location | Year |
|---|---|---|---|
| Amikacin resistance | 19% | Baghdad | 2016 |
| Gentamicin resistance | 26% | Baghdad | 2016 |
| Amikacin resistance | 16% | Ramadi | 2022 |
| Gentamicin resistance | 19% | Duhok | 2020 |
| Gentamicin resistance | >50% | Baghdad | 2020 |
| Gentamicin resistance | 26% | Baghdad | 2016 |
| Gentamicin resistance | 18.3% | AL-Diwaniyah | 2022 |
| Gene - *strA/B* | 71.4% | Najaf | 2022 |
| Gene - *aacC-1* | 7.1% | Najaf | 2022 |
| Gene - *aac(6')/aph(2')* | 3.6% | Najaf | 2022 |
| Genes - *aacC-2*, *aph(3')-IIIa*, *ant(4')-Ia* | 0% | Najaf | 2022 |

## V. Quinolone Resistance

There are four main mechanisms of quinolone resistance that are employed by *K. pneumoniae*, and these are summarized in Figure 1 (Abbas, 2021; Muhsin *et al.*, 2022).

In a study conducted in Iraq, collecting isolates from many centers, 19-46% of the isolates showed resistance to ciprofloxacin across different hospitals (Mustafa and Abdullah, 2020). In AL-Diwaniyah province, Iraq, resistance to ciprofloxacin was 25.2% (Aljanaby and Alhasnawi, 2017) (Table 4).

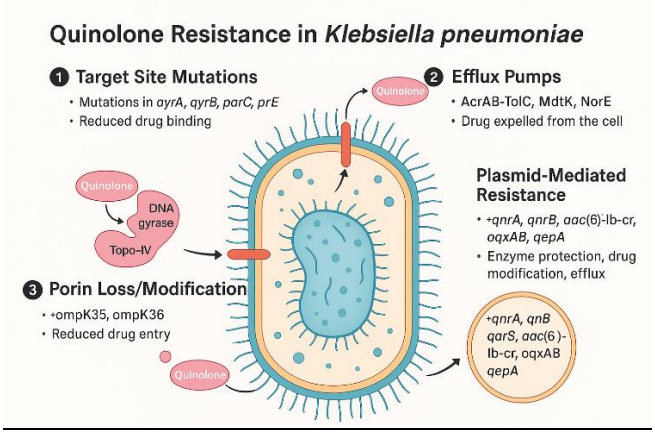


Figure 1. Mechanisms of quinolone resistance in K. pneumoniae. First, target site mutations in gyrA, gyrB, parC, and parE that reduce drug binding to DNA gyrase and topoisomerase IV; second, active efflux of quinolones via pumps such as AcrAB-TolC, MdtK, and NorE; third, decreased permeability due to loss or modification of porin proteins OmpK35 and OmpK36; Finally, plasmid-mediated resistance involving genes like qnrA, qnrB, aac(6')-Ib-cr, oqxAB, and qepA, which confer protection to target enzymes, modify drugs, or enhance efflux of antibiotics.

In another study conducted in burn unit in 2018, 59.5% of *K. pneumoniae* isolates were resistant to ciprofloxacin, indicating high nosocomial resistance (Hammoudi and Hussein, 2018). However, a study from Duhok in 2020 showed a 100% sensitivity to both ciprofloxacin and levofloxacin (Naqid *et al*., 2020). In a study conducted in Iraq investigating plasmid-mediated quinolone resistance (PMQR) in 50 isolates *of K. pneumoniae*, it was found that 76% of isolates carried *qnrS* and 36% carried *qnrB*, with some harboring both genes, while *qnrA*, *qnrC*, and *qnrD* were not detected. Chromosomal mutations in *gyrA* and *parC* have not been specifically reported in these Iraqi studies (Mustafa and Abdullah, 2020). Moreover, genes of plasmid-mediated quinolone resistance (PMQR) such as *qnr* help to lower *K. pneumonia'*s sensitivity to ciprofloxacin by shielding target enzymes from fluoroquinolone binding (Table 4).

Table 4. Summary of quinolone resistance in *K. pneumoniae* in Iraq

| Antibiotic/Gene | Resistance Rate | Location | Year |
|---|---|---|---|
| Ciprofloxacin | 19–46% | Iraq (multiple centers) | 2020 |
| Ciprofloxacin | 59.5% | Baghdad | 2018 |
| Ciprofloxacin | 0% | Duhok | 2020 |
| Levofloxacin | 0% | Duhok | 2020 |
| Gene *qnrS* | 76% | Baghdad | 2020 |
| Gene *qnrB* | 36% | Baghdad | 2020 |
| Gene *qnrA* | 0% | Baghdad | 2020 |
| Gene *qnrC* | 0% | Baghdad | 2020 |
| Gene *qnrD* | 0% | Baghdad | 2020 |

## VI. Conclusions And Recommendations

The findings in this review clearly indicate that *K. pneumoniae* has established complex and multifaceted methods to resist various antibiotics in Iraq. This is a worldwide concern, though the prevalence varies significantly from one region to another. A major concern is that β-lactam antibiotics, such as penicillins, cephalosporins, and carbapenems, are becoming increasingly ineffective against numerous bacterial strains. Ampicillin resistance remains highly prevalent, and ESBL producers are frequently encountered in healthcare facilities across the nation. The alarming rise in resistance to ceftriaxone, ceftazidime, and cefepime in certain provinces is particularly disturbing. The identification of ESBL-associated genes like $bla_{SHV}$, $bla_{TEM}$, and $bla_{CTX-M}$ indicate that these resistance traits can be inherited. While carbapenem resistance was previously rare, it is now on the rise. The detection of carbapenemase genes such as $bla_{OXA-48}$, $bla_{NDM}$, and $bla_{VIM}$ in isolates from Iraq highlights the rapid spread of pan-resistance strains. Although KPC-type carbapenemases remain uncommon, their emergence in Baghdad suggests possible future dissemination. The situation is exacerbated by the loss of porins and the overexpression of efflux pumps (for instance, *ompK35*, *ompK36*, *acrAB*, and *tolC*), which facilitate both innate and acquired resistance mechanisms. Aminoglycoside resistance appears to be relatively mild and is influenced by geographic location and the type of infection present.

Amikacin still ranks as one of the most effective options, though gentamicin resistance shows significant variability depending on usage patterns and the severity of infections. Genetic studies reveal that changes in enzymes such as *strA*

and *strB* and *aac* genes can confer resistance to bacteria. Moreover, the rise in quinolone resistance is attributed to mutations in the target area, efflux mechanisms, and plasmid-mediated factors. Some regions, such as Duhok, still show sustained susceptibility, whereas in hospitals, resistance levels have escalated to as much as 60%. The common occurrence of *qnrS* and *qnrB* genes highlights the potential for resistance traits to be transmitted across species and areas via horizontal gene transfer.

The high resistance rates of *K. pneumoniae* in Iraq are not only a national concern but also a serious global health threat. Resistant strains and resistance genes can disseminate internationally through travel, medical tourism, migration, and trade. Given Iraq's strategic geographic location and regional conflicts, the spread of multidrug-resistant organisms from the region may significantly affect neighboring countries and beyond. As such, combating this issue requires immediate international cooperation, including shared surveillance data, coordinated infection control policies, and collaborative research on novel diagnostics and therapeutics. Only through a global, unified approach can we hope to mitigate the growing threat posed by resistant pathogens like *K. pneumoniae*. It is crucial to enhance infection prevention strategies, particularly in intensive care units and other high-risk areas, while also increasing the adoption of molecular diagnostics to enable rapid detection of resistance genes.

## REFERENCES


Abbas, F.M. (2021). Prevalence of transferable OXA-1 β-lactamase associated with carbapenem-resistant *Klebsiella pneumoniae* isolates in Iraq. *J Pure Appl Microbiol*, 15(2), 877-882.

Abduljabar, S.S., Naqid, I.A. (2022). *Staphylococcus aureus* among athletes in Zakho City, Kurdistan Region, Iraq: Nasal carriage rate, risk factors, and antibiotic sensitivity profile. *Science Journal of University of Zakho*, 10(3), 112-118.

Abdulkareem, W.L., Hussein, N.R., Mohammed, A.A., Arif, S.H., Naqid, I.A. (2020). Risk factors association for MRSA nasal colonization in preoperative patients in Azadi Teaching Hospital-Duhok, Kurdistan Region, Iraq. *Science Journal of University of Zakho*, 8(3), 88-91.

Abid Fazaa, A.S. (2023). Detection of *AcrA* and *AcrB* efflux pumps in multidrug-resistant *Klebsiella pneumonia* that isolated from wounds infection patients in Al-Diwaniyah province. *Arch Razi Inst*, 78(1), 269-276.

Al-Brefkani, A.M., Rasheed, N.A., Hussein, N.R. (2023). Virulotyping of *Listeria monocytogenes* isolated from human and food products samples using multiplex PCR in Duhok Province, Iraq. *Science Journal of University of Zakho*, 11(2), 215-219.

Aljanaby, A.A.J., Alhasnawi, H. (2017). Phenotypic and molecular characterization of multidrug resistant *Klebsiella pneumoniae* isolated from different clinical sources in Al-Najaf Province-Iraq. *Pak J Biol Sci*, 20(5), 217-232.

Bakr, K.I., Abdul-Rahman, S.M., Hamasalih, R.M. (2022). Molecular detection of β-lactamase genes in *Klebsiella pneumoniae* and *Escherichia coli* isolated from different clinical sources. *Cell Mol Biol (Noisy-le-grand)*, 67(4), 170-180.

Hammoudi, A.A., Hussein, A.N. (2018). Antibiotic resistance of *Klebsiella pneumoniae* isolates from in patients with burn infections. *Journal of Wasit for Science and Medicine*, 11(1), 133-145.

Hussein, N., Abozait, H., Naqid, I., Yasen, R. (2025). Antibiotic Resistance in *Pseudomonas aeruginosa* in Iraq: A Narrative Review. *Journal of Life and Bio Sciences Research*, 6(01), 55-63.

Hussein, N.H., Kareem, S.M., Al-Kakei, S. N.H., Taha, B.M. (2022). The predominance of *Klebsiella pneumoniae* carbapenemase (KPC-type) gene among high-level carbapenem-resistant *Klebsiella pneumoniae* isolates in Baghdad, Iraq. *Mol Biol Rep*, 49(6), 4653-4658.

Hussein, R.A., Al-Kubaisy, S.H., Al-Ouqaili, M.T.S. (2024). The influence of efflux pump, outer membrane permeability and β-lactamase production on the resistance profile of multi, extensively and pandrug resistant *Klebsiella pneumoniae*. *J Infect Public Health*, 17(11), 102544.

Huy, T.X.N. (2024). Overcoming *Klebsiella pneumoniae* antibiotic resistance: new insights into mechanisms and drug discovery. *Beni-Suef University Journal of Basic and Applied Sciences*, 13(1), 13.

Ibraheim, H.K., Madhi, K.S., Jasim, A.S., Gharban, H.A. (2024). Molecular identification of Klebsiella species from pneumonic goats, Iraq. *Open Veterinary Journal*, 14(11), 2980.

Mansor, M.R., Kashkool, A.H., D., Al-Ameer, S.S., Al-Hasan, B.K., Almulla, A.F. (2022). Molecular detection of *bla*(SHV-la) Gene in *Klebsiella pneumonia* isolated from urinary tract infections, Najaf, Iraq. *Arch Razi Inst*, 77(3), 1181-1184.

Mohammed, A.B., Anwar, K.A. (2022). Phenotypic and genotypic detection of extended spectrum beta lactamase enzyme in *Klebsiella pneumoniae*. *PLoS One*, 17(9), e0267221.

Mohammed, A.N., Al-Rawi, D.F., Buniya, H.K. (2023). Evaluation of antibiotic resistance of *Klebsiella pneumoniae* isolated from patients in hospitals in Iraq. *Acta Microbiologica Bulgarica* 39(4), 411-417.

Muhsin, E.A., Sajid Al-Jubori, S., Abdulhemid Said, L. (2022). Prevalence of efflux pump and porin-related antimicrobial resistance in clinical *Klebsiella pneumoniae* in Baghdad, Iraq. *Arch Razi Inst*, 77(2), 785-798.

Mustafa, S.M., Abdullah, R.M. (2020). Prevalence of quinolones resistance proteins encoding genes (qnr genes) and co-resistance with β-lactams among *Klebsiella pneumoniae* isolates from Iraqi patients. *Baghdad Science Journal*, 17(2), 0406-0406.

Naghavi, M., Vollset, S.E., Ikuta, K.S., Swetschinski, L.R., Gray, A.P., Wool, E.E., Aguilar, G.R., Mestrovic, T., Smith, G., Han, C., Hsu, R.L., *et al.* (2024). Global burden of bacterial antimicrobial resistance 1990–2021: a systematic analysis with forecasts to 2050. *The Lancet*, 404(10459), 1199-1226.

Naqid, I.A., Hussein, N.R., Balatay, A.A., Saeed, K.A., Ahmed, H.A. (2020). The antimicrobial resistance pattern of *Klebsiella pneumonia* isolated from the clinical specimens in Duhok City in Kurdistan Region of Iraq. *J Kermanshah Univ Med Sci*, 24(2), e106135.

Oliveira, M., Antunes, W., Mota, S., Madureira-Carvalho, Á., Dinis-Oliveira, R. J., Dias da Silva, D. (2024). An overview of the recent advances in antimicrobial resistance. *Microorganisms*, 12(9).

Pishtiwan, A.H., Khadija, K.M. (2019). Prevalence of *bla*TEM, *bla*SHV, and *bla*CTX-M genes among ESBL-producing *Klebsiella pneumoniae* and *Escherichia coli* isolated from thalassemia patients in Erbil, Iraq. *Mediterranean journal of hematology and infectious diseases*, 11(1), e2019041.

Raouf, F.E.A., Benyagoub, E., Alkhudhairy, M.K., Akrami, S., Saki, M. (2022). Extended-spectrum beta-lactamases among *Klebsiella pneumoniae* from Iraqi patients with community-acquired pneumonia. *Rev Assoc Med Bras (1992)*, 68(6), 833-837.

Ristori, M.V., Scarpa, F., Sanna, D., Casu, M., Petrosillo, N., Longo, U.G., Lucia, D., Spoto, S., Chiantia, R.M., Caserta, A. *et al.* (2024). Multidrug-Resistant *Klebsiella pneumoniae* strains in a Hospital: Phylogenetic analysis to investigate local epidemiology. *Microorganisms*, 12(12).

Riwu, K.H.P., Effendi, M.H., Rantam, F.A., Khairullah, A.R., Widodo, A. (2022). A review: Virulence factors of *Klebsiella pneumonia* as emerging infection on the food chain. *Vet World*, 15(9), 2172-2179.

Sajerli, B., Makai, K., Lakatos, L., Sarkadi-Nagy, Á., Burián, K., Orosz, L. (2025). Aminoglycoside resistance dynamics and its predictive value for carbapenem resistance in multidrug-resistant

Acinetobacter baumannii and *Klebsiella pneumoniae*. *Journal of Global Antimicrobial Resistance*, 43, 309-318.

Santajit, S., Indrawattana, N. (2016). Mechanisms of antimicrobial resistance in ESKAPE pathogens. *BioMed research international*, 2016.

Singh, R.E., Veena, M., Raghukumar, K., Vishwanath, G., Rao, P.S., Murlimanju, B. (2011). ESBL production: Resistance pattern in Escherichia coli and *Klebsiella pneumoniae*, a study by DDST method. *IJABPT*, 2(4), 415-422.

Tuwaij, N., Al-Khilkhali, H., Al-Ghazaly, N. (2019). Genetic detection of aminoglycoside resistance genes among *Klebsiella pneumoniae* isolated from environment of Al-Najaf Hospitals, Iraq. *International Journal of Advances in Science Engineering and Technology*, 7(3), 55-60.