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Review on the Imipenem Resistance in Patients with *E. Coli* and *K. Pneumoniae* Infection in Iraq

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A B S T R A C T
This article aimed to review the resistance mechanisms in Escherichia coli and Klebsiella pneumoniae and to conduct a comprehensive study of previous studies in all governorates of Iraq on the resistance of Escherichia coli and Klebsiella pneumoniae to imipenem using different clinical samples. The study demonstrated the prevalence of imipenem resistance in clinical isolates of *E. coli* and *K. pneumoniae* collected from different locations in Iraq. The results indicate an increase in carbapenem resistance rates in Iraq primarily driven by the production of carbapenemase enzymes, including New Delhi metallo-β-lactamase (NDM) and Klebsiella pneumoniae carbapenemase (KPC). The study emphasizes the urgent need for comprehensive strategies, including enhanced surveillance, judicious use of antibiotics, and implementation of infection control measures, to mitigate the growing threat of carbapenem resistance in Iraq.

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INTRODUCTION

Escherichia coli (E. coli) is a group of bacteria that can be found as a pathogenic strain (O157:H7) that causes foodborne illness, UTIs, and other diseases by producing Shiga toxins. This pathogenic strain can be transfer by undercooked ground beef, unpasteurized milk and contaminated water (1), these bacteria can be classified into numerous groups, one causes diarrheal in infants in developing countries that called Enteropathogenic *E. coli* (EPEC) by adhering to the intestinal wall and causing changes to the intestinal epithelium and leading to diarrhoea (2). Other groups of pathogenic bacteria can cause traveller's diarrhoea and diarrhoea in children in developing countries, called Enterotoxigenic E. coli (ETEC), which act by producing enterotoxins (heat-labile and heat-stable toxins) that stimulate the intestines and lead to watery diarrhea (3). Shiga Toxin-Producing E. coli (STEC) can produce Shiga toxins that damage blood vessels and lead to kidney failure (4). Enteroinvasive *E. coli* (EIEC) causes a severe form of diarrhea with blood and mucus called dysentery by invades the epithelial cells in the colon and leading to cell death and inflammation (5). Another strain called Enteroaggregative *E. coli* (EAEC) that associated with persistent diarrhea; this forms a characteristic "stacked brick" pattern of adherence to epithelial cells and produces toxins that cause inflammation (6). A final strain called Uropathogenic *E. coli* (UPEC) that adheres to the uroepithelial cells and causes MJI8 inflammation and infection, including cystitis and pyelonephritis (7).

Klebsiella pneumoniae is a Gram-negative bacterium found in the human respiratory tract and intestine as part of the normal flora and can cause opportunistic infections, causing a range of infections, especially in immunocompromised individuals, and also has a role in hospital-acquired infections including pneumonia, wound infections, urinary tract infections and bloodstream infections (8). Klebsiella pneumoniae infection results from the ability of the bacteria to adhere to host cells through villi, and can also adhere to epithelial cells in the lungs, urinary tract, and other tissues. This bacterium has several penetration mechanisms, such as its ability to form a thick capsule that helps it evade the immune system by preventing phagocytosis by host immune cells, producing a variety of toxins that damage host tissues, such as siderophores that capture iron from the host to aid bacterial growth and it can also form biofilms on medical devices, leading to persistent infections in urinary tract infections and catheter-associated infections (9).

Common infections associated with this bacterium include pneumonia, which is the most common and occurs especially in patients with chronic lung disease or those in hospitals, and can lead to severe necrotizing pneumonia with bloody sputum (referred to as "currant jelly sputum") (10). This bacterium can cause hospital- and community-acquired urinary tract infections, especially in patients with catheters or those with immunosuppression (11). This bacterium can also enter the bloodstream using various routes, leading to sepsis and septic shock (12). Klebsiella pneumoniae can also cause surgical wound infections, especially in immunocompromised individuals (13). In rare cases, Klebsiella pneumoniae can cause meningitis and liver abscesses, especially in individuals with diabetes or chronic alcohol abuse (14).

Mechanisms of Resistance

E. coli is a Gram-negative bacterium commonly found in the intestines of humans and other warm-blooded organisms. Some strains of E. coli are pathogenic, and antibiotic resistance in these strains poses a significant public health challenge. The mechanisms by which *E. coli*

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develops resistance to antibiotics are diverse and often involve several genetic and biochemical strategies:

Enzymatic Inactivation of Antibiotics
E. coli can produce many enzymes that inhibit the action of a wide range of antibiotics, as it has the ability to produce beta-lactamase enzymes that work to degrade the beta-lactam ring, making beta-lactam antibiotics (such as penicillins, cephalosporins, and carbapenems) ineffective, these enzymes are considered one of the main mechanisms for the resistance of these bacteria to antibiotics (15).

Alteration of Target Sites
E. coli has the ability to acquire mutations in genes encoding antibiotic targets that reduce the drug's ability to bind effectively to them. For example, reduced affinity of beta-lactam antibiotics can occur due to mutations in penicillin-binding proteins (such as PBP2), and reduced affinity of antibiotics such as macrolides, tetracyclines, and chloramphenicol for the ribosome can occur due to mutations in rRNA or ribosomal proteins (16).

Efflux Pumps
E. coli has an effective antibiotic resistance mechanism in the form of membrane-based efflux pumps that move antibiotics out of the bacterial cell, thereby reducing their intracellular concentrations and making them less effective. For example, the AcrAB-TolC efflux pump system prevents the accumulation of antibiotics inside the cell by expelling them outside and is responsible for multidrug resistance to tetracyclines, fluoroquinolones, and beta-lactam antibiotics (17).

Reduced Permeability
One of the mechanisms of *E. coli* resistance to antibiotics is through changes in the permeability of its outer membrane by causing mutations in the genes that encode for outer membrane proteins, such as porin mutations as OmpF and OmpC that leads to reduces the flow of antibiotics in to the cell, such beta-lactams and quinolones (18).

Horizontal Gene Transfer (HGT)
E. coli bacteria have the ability to horizontally transfer genes through the process of recombination by conjugation, transformation, and conversion, and through this process, bacteria can acquire antibiotic resistance genes (19).

Biofilm Formation
E. coli bacteria have the ability to form biofilms, which act as a physical barrier that reduces the penetration of antibiotics into them (20).

Target Overproduction
Bacteria can increase the production of the target for antibiotic action, for example, overproduction of folate pathway enzymes such as dihydrofolate (FolA) or dihydropteroate synthetase (FolP), by providing excess target molecules that can compete with the antibiotic and thus resist sulphonamides and trimethoprim (21).

Genetic Mutation and Selection
Antibiotic resistance can occur due to spontaneous mutations in the bacterial genome, allowing resistant strains to proliferate. For example, mutations in genes encoding DNA gyrase (gyrA) or topoisomerase IV (parC) cause resistance to quinolones such as ciprofloxacin (22).

Klebsiella pneumoniae
Klebsiella pneumoniae is becoming increasingly resistant to many antibiotics due to its ability to produce broad-spectrum beta-lactamases (ESBLs) and carbapenemases such as Klebsiella pneumoniae carbapenemase(KPC). This has made treatment more difficult, especially in the case of multidrug-resistant (MDR) strains (29).

Beta-lactamase Production
One of the most important resistance mechanisms in *K. pneumoniae* is the production of beta-lactamases that degrade beta-lactam antibiotics (such as penicillins, cephalosporins, and carbapenems), e.g., extended-spectrum beta-lactamases (ESBLs) that degrade cephalosporins (such as cefotaxime and ceftriaxone). Among the enzymes of K. pneumoniae bacteria are carbapenems such as Klebsiella pneumoniae carbapenemase(KPC) which works to inactivate carbapenems (30).

Alteration of Target Sites
K. pneumoniae has the ability to render antibiotics ineffective by reducing the binding of the antibiotic to the target on which it acts. Mutations in PBPs reduce the effectiveness of beta-lactam antibiotics. Bacteria also have the ability to resist antibiotics that target protein synthesis, such as macrolides and aminoglycosides by mutations in ribosomal DNA (31).

Efflux Pump Overexpression
One of the main resistance mechanisms in K. pneumoniae to reduced antibiotic efficacy is the ability to overexpress efflux pumps that flush antibiotics out of bacterial cells and thus reduce their intracellular concentration, for example, the AcrAB-TolC efflux pump, where this system functions. Helps repel a wide range of antibiotics, including fluoroquinolones, tetracyclines, and beta-lactams (18).

Porin Channel Mutations
K. pneumoniae can develop resistance to antibiotics through mutations in genes that encode the outer membrane and thus alter the permeability of cell membranes and restrict the entry of antibiotics into the cell. For example, mutations in genes that encode porins, such as OmpK35 and OmpK36, reduce the permeability of the outer membrane to some antibiotics, especially beta-lactams and carbapenems (32).

Horizontal Gene Transfer
K. pneumoniae bacteria can undergo horizontal gene transfer, such as conjugation, transformation, and genetic conversion, and thus, the ability to acquire resistance genes from other bacteria. Many of the resistance traits in K. pneumoniae are transferred on plasmids, which can be transferred between different types of bacteria. For example, there are Plasmids carrying carbapenemase genes (e.g., bla_KPC) that are commonly found in *K. pneumoniae* (33).

Biofilm Formation
Biofilms play an important role in antibiotic resistance of *K. pneumoniae* as the matrix prevents antibiotic penetration and enhances persistence in hostile environments (34).

Table 1 : Mechanisms of Antibiotic Resistance in *Escherichia coli*

Mechanism	Description	Example of Antibiotics Affected	Reference
Production of β-lactamases	Enzymes that degrade β-lactam antibiotics (e.g., penicillins and cephalosporins) by hydrolyzing the β-lactam ring.	Penicillin, Cephalosporins, Carbapenems	23
Efflux pumps	Transport proteins that expel antibiotics from the bacterial cell, decreasing drug concentration inside.	Tetracyclines, Fluoroquinolones, Macrolides	24
Target modification	Alteration of the antibiotic's target site so that the antibiotic can no longer bind effectively.	Methicillin, Vancomycin	25
Decreased permeability	Changes in the bacterial outer membrane or cell wall that prevent the antibiotic from entering the cell.	Penicillin, Carbapenems, Tetracyclines	26
Enzymatic inactivation of aminoglycosides	Aminoglycoside-modifying enzymes (AMEs) chemically modify antibiotics to deactivate them.	Gentamicin, Tobramycin, Amikacin	27
Modification of antibiotic targets (ribosome)	Modification of the ribosomal binding site to prevent antibiotics from binding.	Streptomycin, Tetracycline	28

Table 2: Mechanisms of Antibiotic Resistance in *Klebsiella pneumoniae*

Mechanism	Description	Example of Antibiotics Affected	Reference
Production of β-lactamases	Klebsiella pneumoniae produces extended-spectrum β-lactamases (ESBLs) and carbapenemases that degrade β-lactam antibiotics.	Penicillins, Cephalosporins, Carbapenems	36
Carbapenemase production (e.g., KPC)	Carbapenem-hydrolyzing enzymes (e.g., KPC) render carbapenems ineffective, which are often considered the last-resort antibiotics.	Carbapenems (Imipenem, Meropenem)	37
Efflux pumps	Active transport systems that expel antibiotics from the cell, reducing intracellular antibiotic concentrations.	Tetracyclines, Fluoroquinolones, Aminoglycosides	38
Target modification	Modification of antibiotic binding targets (e.g., ribosomes or cell wall precursors), preventing effective binding of antibiotics.	Aminoglycosides, Macrolides	25
Reduced permeability	Alterations in the outer membrane or porin channels reduce the influx of antibiotics into the bacterial cell.	Penicillins, Carbapenems	39
Modification of outer membrane proteins	Alteration of porins (e.g., OmpK36) that reduce antibiotic entry, contributing to resistance.	Carbapenems, Cephalosporins	40
Aminoglycoside-modifying enzymes	Enzymes that chemically modify aminoglycosides, preventing them from binding to their target on the ribosome.	Gentamicin, Tobramycin, Amikacin	27
Modification of peptidoglycan synthesis	Alteration in cell wall biosynthesis enzymes to evade the action of antibiotics like vancomycin.	Vancomycin, Beta-lactams	29

Target Overproduction

K. pneumoniae can develop resistance by overproducing enzymes targeted by antibiotics, which may reduce the effectiveness of the drug. For example, K. pneumoniae can overproduce enzymes such as dihydropyridine synthetase or dihydrofolate reductase, leading to resistance to sulphonamides and trimethoprim (21).

Mutation in DNA Gyrase and Topoisomerase

Mutations in the genes encoding DNA gyrase and topoisomerase IV (such as gyrA and parC) can lead to resistance to fluoroquinolones like ciprofloxacin by preventing the drugs from binding effectively to their targets, impairing DNA replication and repair (35).

Epidemiology of Resistance

E. coli and **K. pneumoniae** are major pathogens responsible for various infections such as pneumonia, bloodstream infections, and urinary tract infections (UTIs). The resistance of these pathogens to Imipenem in Iraq is rising due to factors like overuse and misuse of antibiotics, insufficient infection control measures, and limited access to new antimicrobial agents. This study reviewed previous studies about imipenem resistance in patients with *E. coli* and *K. pneumoniae* infections in Iraq, as shown in Table 3 below. The results of previous studies in Iraq showed that resistance has recently begun to increase due to the frequent use of the antibiotic in a random manner, and thus, bacteria develop resistance mechanisms against it, as shown in Table 4.

Table 3: Study Groups in Iraq

N.	Province	Number of studies	Bacteria	Resistance range
1	Baghdad	22	E. coli & K. pneumoniae	0 - 61.6 %
2	Najaf	11	E. coli & K. pneumoniae	0 - 100%
3	Wasit	7	E. coli & K. pneumoniae	0 - 63 %
4	Anbar	7	E. coli & K. pneumoniae	0 - 64.4%
5	Erbil	6	E. coli & K. pneumoniae	0 - 45%
6	Duhok	10	E. coli & K. pneumoniae	0 - 12.2%
7	Sulaymaniyah	1	E. coli	4.7
8	Salah al-Din	1	E. coli & K. pneumoniae	0
9	Thi-Qar	2	E. coli & K. pneumoniae	3.3-10
10	Babylon	5	E. coli & K. pneumoniae	0-14.2
11	Diyala	2	*K. pneumoniae*	0-52.6
12	Maysan	1	E. coli & K. pneumoniae	0
13	Diwaniyah	2	E. coli & K. pneumoniae	2.7- 41.4
14	Basrah	1	E. coli & K. pneumoniae	2.4 & 13.2
15	Karbala	1	E. coli & K. pneumoniae	0 & 10
16	Ninewa	1	E. coli & K. pneumoniae	0 & 0
17	Kirkuk	2	E. coli & K. pneumoniae	1.5 & 88 %

Table 4: Imipenem resistance in patients with *E. Coli* and *K. Pneumoniae* infection in Iraq

N.	Y published	Province	Bacterium	Source of infection	No. of isolates	Resistance status	Reference
1	2010	Baghdad	*E. coli*	Urine	10	0 R	41
2	2010	Najaf	*K. pneumoniae*	Urine	25	0 R	42
3	2011	Wasit	*E. coli*	Stool	145	0 R	43
4	2011	Baghdad	*E. coli*	Urine	75	0 R	44
5	2012	Wasit	*E. coli*	Stool	325	0 R	45
6	2012	Anbar	*E. coli*	UTIs	289	4.5R	46
7	2013	Erbil	*E. coli*	UTIs	50	4.7R	47
8	2013	Baghdad	*E. coli* *K.pneumoniae*	Wound Burn	67	0R 6.66R	48
9	2014	Erbil	*E. coli*	Water	36	31 R	49
10	2015	Duhok	*E. coli* *K. pneumoniae*	UTIs	141	0R 0R	50
11	2015	Sulemania	*E. coli*	UTIs	150	4.7R	51
12	2015	Najaf	*K. pneumoniae*	Urine Burn Genital tract Wound, Environmental hospital	89	0 R	52
13	2015	Tikrit	*E. coli* *K. pneumoniae*	Urine	100	0 R 0 R	53
14	2015	Baghdad	*E. coli* *K. pneumoniae*	Urine Burn swabs, wound swab, Sputum Blood	70 14	4 R 8 R	54
15	2015	Baghdad	*K. pneumoniae*	Burns	45	9.3 R	55
16	2015	Baghdad	*K. pneumoniae*	Urine	80	57.2 R	56
17	2015	Thi-Qar	*E. coli*	Burn swabs, wound swab, vaginal swabs urine Diarrheal	90	3.33 R	57
18	2016	Wasit	*E. coli*	UTIs	91	0 R	58
19	2016	Duhok	*E. coli*	UTIs	106	0 R	59
20	2016	Kirkuk	*E. coli*	Urine	234	1.5R	60

21	2016	Baghdad	*K. pneumoniae*	Urine Bacteraemia Wound Burn Sputum Ear Pus Stool	24	29.17R	61
22	2016	Baghdad	*E. coli*	Seminal fluid	4	0 R	62
			K. pneumoniae		7	0 R	62
23	2016	Babylon	*K. pneumoniae*	Sputum	15	14.28 R	63
24	2016	Baghdad	*K. pneumoniae*	Burn	210	21.42 R	64
25	2016	Najaf	*K. pneumoniae*	Burn	23	52.38 R	65
26	2016	Diyala	*K. pneumoniae*	Urine Wounds Burns	18	0 R	66
27	2017	Duhok	*E. coli*	UTIs	276	2.1R	67
28	2017	Najaf	*K. pneumoniae*	Urine Burn	43	9.30 R	68
29	2017	Erbil	*K. pneumoniae*	Sputum Urine Wound	84	4.76 R	69
30	2017	Duhok	*K. pneumoniae*	CSF	33	0 R	70
31	2017	Baghdad	*K. pneumoniae*	Burn Wound	11	18 R	71
32	2018	Baghdad	*K. pneumoniae*	Sputum Blood Burns Wound Urine Ear	39	58.9 R	72
33	2018	Baghdad	*K. pneumoniae*	Burn Wound	36	21.42 R	73
34	2018	Najaf	*K. pneumoniae*	Urine Burn Seminal fluid	69	20 R	74
35	2018	Erbil	*E. coli*	Sputum Urine Wound	60	5 R	75
36	2018	Baghdad	*E. coli*	Urine	30	0 R	76
			K. pneumoniae		11	0 R	
37	2018	Najaf	*E. coli*	Burns	51	0 R	77
			K. pneumoniae		93	3.2 R	
38	2018	Baghdad	*E. coli*	Urine	121	4.1 R	78
39	2019	Duhok	*K. pneumoniae*	Urine Blood Wound swab Vaginal swab Sputum CSF	281	12.2 R	79
40	2019	Babylon	*E. coli*	Urinary tract infection (UTI)	42	11.9 R	80
41	2019	Anbar	*E. coli*	Burns Wounds Otitis media, Urinary tract infection (UTI)	7	25 R	81
42	2019	Erbil	*E. coli*	Urine Blood	24	45.8R	82 82
			K. pneumoniae	Sputum	74	37.5R	
43	2019	Erbil	*E. coli*	Urine	20	0 R	

			K. pneumoniae		48	0 R	83
44	2019	Baghdad	*K. pneumoniae*	Wound Urine Ear	39	58.9 R	84
45	2019	Baghdad	*K. pneumoniae*	Urine Blood Sputum Wounds Burns	50	36 R	85
46	2019	Anbar	*K. pneumoniae*	Burned Wounds Sputum C.S.F Blood	50	22 R	86
47	2020	Najaf	*E. coli*	Urine	46	0 R	87
			K. pneumoniae		29	1 R	
48	2020	Najaf	*E. coli*	Urine	76	0 R	88
49	2020	Duhok	*E. coli*	Urine, Wounds Cervical blood semen Ascitic fluid Cerebral spinal fluid	454	3.52 R	89
50	2020	Baghdad	*E. coli*	Blood	11	21.43 R	90
			K. pneumoniae		14	18.18 R	
51	2020	Duhok	*K. pneumoniae*	urine	282	0 R	91
52	2021	Misan	*E. coli*	Burn	8	0 R	92
			K. pneumoniae	Wound	7	33.3 R	
53	2021	Anbar	*E. coli*	Urine	33	12.12 R	93
			K. pneumoniae	Faces	54	7.41 R	
54	2021	Duhok	*E. coli*	Wound	13	9 R	94
			K. pneumoniae		25	4 R	
55	2021	Najaf	*K. pneumoniae*	Urine	207	2.7R	95
56	2021	Anbar	*K. pneumoniae*	Urine Sputum Wound	45	64.44R	96
57	2021	Baghdad	*K. pneumoniae*	Urine	150	2.6 R	97
58	2021	Diwaniyah	*K. pneumoniae*	Sputum Burn Urine	70	41.42 R	98
59	2022	Wasit	*K. pneumoniae*	Urine	77	19.48 R	99
60	2022	Diwaniyah	*E. coli*	Urine	256	2.7R	100
61	2022	Basrah	*E. coli*	Urine	82	2.4 R	101
			K. pneumoniae		38	13.2 R	
62	2022	Baghdad	*E. coli*	Blood	25	12R	102
			K. pneumoniae		20	55R	
63	2022	Najaf	*K. pneumoniae*	Sputum	163	0 R	103
64	2022	Babylon	*E. coli*	Stools	100	0 R	104
			K. pneumoniae	Urine		0 R	
65	2022	Duhok	*E. coli*	Urine	166	7.83 R	105
			K. pneumoniae		65	0 R	
66	2023	Babylon	*E. coli*	Urine	231	4.5 R	106
67	2023	Karbala	*E. coli*	Urine	63	0 R	107
			K. pneumoniae		30	10 R	
68	2023	Anbar	*K. pneumoniae*	Urine Blood Wound Sputum	29	27.6R	108

69	2023	Baghdad	*K. pneumoniae*	Urine Wound Blood Sputum Pus Endotracheal	35	52.8 R	109
70	2023	Wasit	*E. coli*	Urine	284	5 R	110
71	2023	Diyala	*K. pneumoniae*	Wounds Burns Sputum	29	58.62 R	111
72	2024	Baghdad	*K. pneumoniae*	Blood Urine wounds Burns Sputum	61	19.67 R	112
73	2024	Babylon	*E. coli*	Urine	71	1.5 R	113
			K. pneumoniae		6	0 R	
74	2024	Anbar	*K. pneumoniae*	Urine Wound Blood Sputum	55	13.8R	114
75	2024	Mosul	*E. coli*	Urine	62	0 R	115
			K. pneumoniae		32	0 R	
76	2024	Wasit	*K. pneumoniae*	Urine	16	63R	116
77	2024	Wasit	*K. pneumoniae*	Urine	212	2R	117
78	2024	Baghdad	*K. pneumoniae*	Urine Blood	60	61.6 R	118
79	2024	Kirkuk	*K. pneumoniae*	Urine	17	88 R	119
80	2024	Najaf	*K. pneumoniae*	Wound Urine Burn Blood Semen Ear Sputum	27	100 R	120
81	2024	Thi-Qar	*E. coli*	Uremia	40	5 R	121
			K. pneumoniae		30	10 R	
82	2024	Duhok	*E. coli*	Urine	265	0 R	122

Risk Factors for Resistance Development

Many healthcare-associated factors play an important role in the emergence and spread of carbapenem-resistant strains of bacteria, including prolonged hospitalizations, surgical procedures, frequent use of broad-spectrum antibiotics, and inadequate infection control protocols in hospitals. In Iraq, crowded health environments and widespread use of antibiotics in both health and community settings increase the likelihood of resistance developing (123).

Prevention and Control Measures

Effective infection control practices, such as isolation of infected patients, proper sterilization of medical equipment, and regular hand hygiene, are essential to limit the spread of resistant strains. Antibiotic stewardship programs that aim to rationalize the use of imipenem and other antibiotics can help slow the development of resistance (124).

Conclusions

The increasing resistance of *E. coli* and *K. pneumoniae* to imipenem in Iraq poses a serious threat to public health, limiting treatment options for severe bacterial infections. The study highlights a review of previous studies in Iraq that have shown that resistance to imipenem is increasingly prevalent, and that factors such as antibiotic misuse, inadequate infection control protocols, and ease of horizontal gene transfer are exacerbating the problem. Addressing this problem requires a multifaceted approach, including robust antimicrobial stewardship programs, regular monitoring of resistance patterns, and stringent infection prevention measures in health care settings. Immediate action is essential to limit the spread of imipenem-resistant strains and maintain the effectiveness of carbapenems as a treatment of last resort.

Reference

1. Kaper JB, Nataro JP, Mobley HL. Pathogenic *Escherichia coli*. Nat Rev Microbiol. 2004;2(2):123-40.
2. Nataro JP, Kaper JB. Diarrheagenic *Escherichia coli*. Clin Microbiol Rev. 1998;11(1):142-201.
3. Nataro JP, Kaper JB. Enterotoxigenic *Escherichia coli* (ETEC): pathogenesis, epidemiology, and prevention. Clin Microbiol Rev. 2004;17(4):597-620.
4. Rivas M, Becerra M. Shiga toxin-producing *Escherichia coli* (STEC) infection: pathogenesis and epidemiology. Front Cell Infect Microbiol. 2017;7:2.

5. Reeves PR, Clegg S. The biology of *Escherichia coli*. Microbiol Monogr. 2002;1.
6. Nataro JP, Kaper JB. Enteroaggregative *Escherichia coli*: a new category of enteric pathogens. Emerg Infect Dis. 2006;12(8):1296-305.
7. Mobley HLT, Chippendale K. Pathogenesis of *Escherichia coli* urinary tract infection. Infect Immun. 2001;69(1):72-81.
8. Abbas R, Chakkour M, Zein El Dine H, et al. General overview of *Klebsiella pneumoniae*: epidemiology and the role of siderophores in its pathogenicity. Biology. 2024;13(2):78.
9. Guerra MES, Destro G, Vieira B, et al. *Klebsiella pneumoniae* biofilms and their role in disease pathogenesis. Front Cell Infect Microbiol. 2022;12:877995.
10. Wright SD, et al. *Klebsiella pneumoniae* pneumonia: pathogenesis and prevention. Am Rev Respir Dis. 1990;141(2):470-3.
11. Patel R. *Klebsiella pneumoniae*: the protean pathogen. Clin Infect Dis. 2013;56(10):1555-6.
12. Tumbarello M, et al. *Klebsiella pneumoniae* bacteremia: risk factors for resistance to third-generation cephalosporins and outcome. Antimicrob Agents Chemother. 2007;51(10):3399-405.
13. Han J, et al. *Klebsiella pneumoniae* infection and its impact on clinical outcomes in patients with burns. Burns. 2013;39(4):727-31.
14. Shon AS, et al. *Klebsiella pneumoniae* liver abscess: pathogenesis and clinical outcome. Infect Immun. 2013;81(3):907-12.
15. Livermore DM. The role of beta-lactamases in resistance to beta-lactam antibiotics in Gram-negative bacteria. Clin Infect Dis. 2008;47(S1):S88-S95.
16. Jacoby GA, Munoz-Price LS. The new beta-lactamases. N Engl J Med. 2005;352(4):380-91.
17. Poole K. Efflux-mediated antimicrobial resistance. J Antimicrob Chemother. 2005;56(1):11-9.
18. Nikaido H. Multidrug efflux pumps of Gram-negative bacteria. J Bacteriol. 2009;191(3):443-50.
19. Zhang Y, Nikaido H. Efflux pumps of Gram-negative bacteria. Annu Rev Microbiol. 2004;58:10.1-10.20.
20. Mah TF, O'Toole GA. Mechanisms of biofilm resistance to antimicrobial agents. Trends Microbiol. 2001;9(1):34-9.
21. Pletzer D, Mansour SC. Target overproduction as a mechanism of bacterial resistance to antibiotics. Antimicrob Agents Chemother. 2018;62(6):e02255-17.
22. Chopra I, Roberts M. Tetracycline antibiotics: mode of action, applications, molecular biology, and epidemiology of bacterial resistance. Microbiol Mol Biol Rev. 2001;65(2):232-60.
23. Bush K, Jacoby GA. Updated functional classification of β-lactamases. Antimicrob Agents Chemother. 2010;54(3):969-76.
24. Poole K. Efflux pumps as antimicrobial resistance mechanisms. Ann Med. 2007;39(3):162-76.
25. Boucher HW, Talbot GH. Advances in the management of antibiotic-resistant infections. Clin Infect Dis. 2007;45(Suppl 2):S89-S95.
26. Lister PD, et al. Antimicrobial resistance in *E. coli* and other Enterobacteriaceae. Int J Antimicrob Agents. 2009;34(1):1-9.
27. Ryu S. Aminoglycoside resistance in *Escherichia coli*. Microb Drug Resist. 2012;18(2):128-35.
28. Møller JB, et al. Mechanisms of resistance to antibiotics in *E. coli*. Nat Rev Microbiol. 2015;13(1):42-52.
29. Giske CG, et al. Global epidemiology of *Klebsiella pneumoniae* resistance to carbapenems. Clin Infect Dis. 2015;60(8):1289-97.
30. Bradford PA. Extended-spectrum beta-lactamases in the 21st century: characterization, epidemiology, and detection of this important resistance threat. Clin Microbiol Rev. 2001;14(4):933-51.
31. Drieux L, Bénézech J, Aubert D. Clinical and laboratory issues of carbapenem resistance in *Klebsiella pneumoniae*. Clin Infect Dis. 2008;47(4):49-52.
32. Lavigne JP, Goussard S. Mechanisms of carbapenem resistance in *Klebsiella pneumoniae* clinical isolates from a French hospital. J Antimicrob Chemother. 2009;63(2):196-8.
33. Chen L, Huang Y. Plasmid-mediated antibiotic resistance in *Klebsiella pneumoniae*. Curr Opin Microbiol. 2017;39:98-103.
34. Tumbarello M, Spanu T, Di Carlo D. Biofilm formation in *Klebsiella pneumoniae*: role in persistence and resistance to therapy. J Antimicrob Chemother. 2010;65(4):711-6.
35. Hooper DC. Mechanisms of action and resistance of older and newer fluoroquinolones. Clin Infect Dis. 2001;32(3):639-45.
36. Queenan AM, Bush K. Carbapenemases: the versatile β-lactamases. Clin Microbiol Rev. 2007;20(3):440-58.
37. Nordmann P, et al. Emergence of Enterobacteriaceae producing extended-spectrum β-lactamases. Clin Microbiol Infect. 2011;17(4):682-7.
38. Li XZ, et al. Efflux pumps and antibiotic resistance in *Klebsiella pneumoniae*. Int J Antimicrob Agents. 2015;45(4):344-55.
39. Giamarellou H, et al. Antibiotic resistance in *Klebsiella pneumoniae*. Clin Microbiol Infect. 2011;17(6):812-20.
40. Zhang R, et al. Porin mutations in *Klebsiella pneumoniae* conferring resistance to carbapenems. Antimicrob Agents Chemother. 2014;58(7):4056-63.
41. Shaokat S, Hameed H. Evaluation of stability of cefamandol and ceftazidime with clavulanic acid against extended-spectrum β-lactamase. Iraqi J Pharm Sci. 2010;19(2):1-6.
42. Almohana A, Hadi Z, AbdulHadi S. Extended-spectrum β-lactamase (ESBL) mediated resistance to third-generation cephalosporins in urinary tract infection isolates of *Klebsiella pneumoniae*. QMJ. 2010;6(9):1-17.
43. Al-Charrakh A, Al-Khafaji J, Shamkhi J. Detection of ESBLs in enteropathogenic (EPEC) isolates associated with infantile diarrhea in Wasit city, Iraq. Asian J Pharm Biol Res. 2011;1:1-16.
44. Khaleq M, Abd A, Dhahi M. Efficacy of combination of meropenem with gentamicin and amikacin against resistant *E. coli* isolated from

patients with UTIs: *in vitro* study. Iraqi J Pharm Sci. 2011;20(2):1-9.
45. Shamki J, Al-Charrakh A, Al-Khafaji J. Detection of ESBLs in enteropathogenic *E. coli* (EPEC) isolates associated with infantile diarrhea in Wasit city. Med J Babylon. 2012;9(2):1-10.
46. Lafi SA, Alkarboly AA, Lafi MS. Bacterial urinary tract infection in adults, Hit district Anbar governorate, west of Iraq. Egypt Acad J Biol Sci G Microbiol. 2012;4(1):21-6.
47. Al-Jebouri MM, Mdish SA. Antibiotic resistance pattern of bacteria isolated from patients of urinary tract infections in Iraq. Open J Urol. 2013;3(2):124-31.
48. Alkaabi S. Bacterial isolates and their antibiograms of burn wound infections in Burns Specialist Hospital in Baghdad. Baghdad Sci J. 2013;10(2):1-10.
49. Aziz R, Al-Zubaidy F, Al-Mathkhury H, et al. Antibiogram of *Escherichia coli* isolated from different hospitals wastewater in Erbil City, Iraq. Iraqi J Sci. 2014;55(2):341-51.
50. Assafi MS, Ibrahim NM, Hussein NR, et al. Urinary bacterial profile and antibiotic susceptibility pattern among patients with urinary tract infection in Duhok city, Kurdistan region, Iraq. Int J Pure Appl Sci Technol. 2015;30(2):54.
51. Merza NS, Jubrael JM. The prevalence of virulence factors among uropathogenic *Escherichia coli* strains isolated from different hospitals in Kurdistan Region-Iraq. Int J Bioinform Biomed Eng. 2015;1(4):338-43.
52. Alshammari M, Al-Skhattat H. Detection of plasmid-mediated quinolone resistance genes in clinical and environmental hospital isolates of *Klebsiella pneumoniae* in Al-Najaf City. Kufa J Nurs Sci. 2015;5(2):1-9.
53. Al-Jebouri M, Al-Alwani H. Antibiotic resistance patterns of bacterial types isolated from urine of Iraqi patients with renal failure. World J Pharm Res. 2015;4(4):217-33.
54. Jabbar Ibrahim I, Kareem Hameed T. Isolation, characterization and antimicrobial resistance patterns of lactose-fermenter Enterobacteriaceae isolates from clinical and environmental samples. Open J Med Microbiol. 2015;5:169-76.
55. Albassam W. Detection of antibiotics resistance genes in clinical isolates of *Klebsiella pneumoniae*. Iraqi J Sci. 2015;56(1):407-16.
56. Abdulhasan G, Jasim S, Fadhil H. Detection of carbapenem-resistant *Klebsiella pneumoniae* using two sensitivity methods. AJPS. 2015;15(2):1-7.
57. Abd ALameer R, Abbas Y, Khudaier B. Genotyping of *Escherichia coli* isolated from clinical and hospitals environment. J Thi-Qar Sci. 2015;5(3):1-10.
58. Al-Mayahie S, Al Kuriashy JJ. Distribution of ESBLs among *Escherichia coli* isolates from outpatients with recurrent UTIs and their antimicrobial resistance. J Infect Dev Ctries. 2016;10(6):575-83.
59. Polse R, Yousif S, Assafi M. Prevalence and antimicrobial susceptibility patterns of uropathogenic *E. coli* among people in Zakho, Iraq. Int J Res Med Sci. 2016;4(4):1219-23.
60. Alsamarai AG, Ali S, Shareef H. Urinary tract infection in female in Kirkuk City, Iraq: causative agents and antibiogram. 2016;5:261-73.
61. Aziz R, Al-Jubori S. Genetic elements responsible for extreme drug resistance (XDR) in *Klebsiella pneumoniae* var *pneumoniae* isolated from clinical samples of Iraqi patients. World J Pharm Res. 2016;5(5):1-23.
62. Al-Hashimi A, Abdu-Allah S, Al-Rrubaa G, Mansur R. The effect of microalgae extraction on bacterial species isolated from seminal fluid of sexually-active males in Baghdad. J Genet Environ Res Conserv. 2016;4(2):171-7.
63. Abbas F. Prevalence of SHV-extended spectrum β-lactamase producing carbapenem-resistant *Klebsiella pneumoniae* among patients with lower respiratory tract infections in Babylon Province-Iraq. Int J ChemTech Res. 2016;10(7):645-9.
64. Hammoudi A, Hussein A, Jebur M. Detection of blaNDM-Metallo-β-lactamase genes in *Klebsiella pneumoniae* strains isolated from burn patients in Baghdad hospitals. Med J Babylon. 2016;13(4):904-13.
65. Tuwaij N. Molecular study of quinolone resistance in *Klebsiella pneumoniae* and *Citrobacter freundii* isolates. Al-Kufa Univ J Biol. 2016;8(3):1-14.
66. AL-Taai H. Bacteriological comparison between *Pseudomonas aeruginosa* and *Klebsiella pneumoniae* isolated from different infectious sources. Diyala J Pure Sci. 2016;12(1):162-82.
67. Hussein NR, Daniel S, Salim K, Assafi MS. Urinary tract infections and antibiotic sensitivity patterns among women referred to Azadi Teaching Hospital, Duhok, Iraq. Avicenna J Clin Microbiol Infect. 2017;5(2):27-30.
68. Aljanaby A, Alhasnawi H. Phenotypic and molecular characterization of multidrug resistant *Klebsiella pneumoniae* isolated from different clinical sources in Al-Najaf Province-Iraq. Pak J Biol Sci. 2017;20(5):217-32.
69. Ali F, Ismael R. Dissemination of *Klebsiella pneumoniae* and *Klebsiella oxytoca* harboring blaTEM genes isolated from different clinical samples in Erbil City. Diyala J Med. 2017;12(2):40-51.
70. Saadi AT, Garjees NA, Rasool AH. Antibiogram profile of septic meningitis among children in Duhok, Iraq. Saudi Med J. 2017;38(5):517-20.
71. Hassan A, Hussein M, Jafer F. Investigation of gene resistance on bacteria *Pseudomonas aeruginosa* and *Klebsiella pneumoniae* isolates from patient with burn and wound infection to some antibiotics. Int J ChemTech Res. 2017;10(3):530-8.
72. Ali F, AL Marjan M, Authman S. Type II toxin antitoxin system in imipenem resistant *Klebsiella pneumoniae* in Baghdad, Iraq. Indian J Public Health Res Dev. 2018;9(12):1313-8.
73. Hammoudi A, Hussein A. Antibiotic resistance of *Klebsiella pneumoniae* isolates from in patients with burn infections. Wasit J Sci Med. 2018;11(1):133-45.
74. Shlash A, Tuwaij N. Molecular dissemination of Ambler class A and C β-lactamase genes among ceftriaxone resistant *Klebsiella pneumoniae* infection in Najaf City, Iraq. Biochem Cell Arch. 2018;18(2):2511-22.
75. Ali F. Distribution of CTX-M gene among *Escherichia coli* strains isolated from different clinical samples in Erbil City. Iraqi J Biotechnol. 2018;17(1):87-90.

76. Abbas M, Mahdi A. Study the risk factors, bacterial profile and antibiotic resistance pattern in urinary tract infections pediatric Iraqi patients. Int Res J Pharm. 2018;9(10):137-42.
77. Aljanaby AAJ, Aljanaby IAJ. Prevalence of aerobic pathogenic bacteria isolated from patients with burn infection and their antimicrobial susceptibility patterns in Al-Najaf City, Iraq—a three-year cross-sectional study. F1000Res. 2018;7:1157.
78. Ghaima KK, Khalaf ZS, Abdulhassan AA, Salman NY. Prevalence and antibiotic resistance of bacteria isolated from urinary tract infections of pregnant women in Baghdad hospitals. Biomed Pharmacol J. 2018;11(4).
79. Mohammed HH, Saadi AT, Yaseen NA. Detection of carbapenem antibiotic resistance in *Klebsiella pneumoniae* in Duhok City/Kurdistan Region/Iraq. Duhok Med J. 2020;14(1):28-43.
80. Al-Hasnawy HH, Judi MR, Hamza HJ. The dissemination of multidrug resistance (MDR) and extensively drug resistant (XDR) among uropathogenic *E. coli* (UPEC) isolates from urinary tract infection patients in Babylon Province, Iraq. Baghdad Sci J. 2019;16:986-922.
81. Al-Ouqaili MT. Molecular detection and sequencing of SHV gene encoding for extended-spectrum β-lactamases produced by multidrug resistance some of the gram-negative bacteria. Int J Green Pharm. 2019;12(4):S910-S918.
82. Ahmed H, Ganjo A. Detection of carbapenemase-producing *Klebsiella pneumoniae* and *Escherichia coli* recovered from clinical specimens in Erbil City Kurdistan Region of Iraq. Al-Mustansiriyah J Sci. 2019;30(2):1-9.
83. Pishtiwan AH, Khadija KM. Prevalence of blaTEM, blaSHV, and blaCTX-M genes among ESBL-producing *Klebsiella pneumoniae* and *Escherichia coli* isolated from thalassemia patients in Erbil, Iraq. Mediterr J Hematol Infect Dis. 2019;11(1):e2019041.
84. Ali F, Authman S, AL Marjani M. Imipenem resistance and biofilm formation in *Klebsiella pneumoniae* from some hospitals in Baghdad City. Pharm Sci Res. 2019;11(1):233-8.
85. Mustafa M, Abdullah R. Detection of 16S rRNA methylases and co-resistance with β-lactams among *Klebsiella pneumoniae* isolates from Iraqi patients. Baghdad Sci J. 2019;16(3):580-7.
86. Ahmed M, Meani S. Occurrence of *Klebsiella pneumoniae* carbapenemase KPC gene in *Klebsiella pneumoniae* isolated from patients in Anbar city of Iraq. KPC gene in K Pneumoniae. 2019;22:1-9.
87. Mohamed KG, Aljanaby AAJ. Urinary tract infections in Al-Kufa City Iraq and phenotypic detection of antimicrobial sensitivity pattern of bacterial isolates. Int J Pharm Res. 2020;1.
88. Hasan TH. Extended spectrum beta lactamase *E. coli* isolated from UTI patients in Najaf Province, Iraq. Int J Pharm Res. 2020;12(4):673-7.
89. Naqid IA, Balatay AA, Hussein NR, et al. Antibiotic susceptibility pattern of *Escherichia coli* isolated from various clinical samples in Duhok City, Kurdistan Region of Iraq. Int J Infect. 2020;7(3):e103740.
90. Abdul-Jabar H, Hameed Abd A, Abdulamir A. Efficacy of combinations of piperacilline/tazobactam, ceftazidime, amikacin and bacteriophage against Enterobacteriaceae sepsis in neonates: *in vitro* study. Syst Rev Pharm. 2020;11(10):165-70.
91. Polse R, Qarani S, Assafi M, et al. Incidence and antibiotic sensitivity of *Klebsiella pneumoniae* isolated from urinary tract infection patients in Zakho Emergency Hospital/Iraq. J Educ Sci. 2020;29(3):257-68.
92. Hateet R. Isolation and identification of some bacteria contemn in burn wounds in Misan, Iraq. Arch Razi Inst. 2021;76(6):1665.
93. Sharqi HM, Hassan OM, Obaid AS. Investigation of the antibiotic-resistant ESKAPE pathogens in Ramadi hospitals, Iraq. Indian J Forensic Med Toxicol. 2021;15(4):3306-13.
94. Ahmad AM. Detection the antibiotyping patterns of the most that common bacterial pathogens susceptible to different antimicrobial agents that cause wound infections, in Duhok, Iraq. J Univ Anbar Pure Sci. 2021;15(1).
95. Hasan T, Alasedi K, Aljanaby A. A comparative study of prevalence antimicrobials resistance *Klebsiella pneumoniae* among different pathogenic bacteria isolated from patients with urinary tract infection in Al-Najaf City, Iraq. Lat Am J Pharm. 2021;40:174-8.
96. Al-Qaysi M, Ahmed M, Habeeb W, et al. Genetic variants of multidrug-resistant *Klebsiella pneumoniae* isolated from Al-Ramadi Teaching Hospital, Iraq. Open Microbiol J. 2021;12:1-12.
97. Salman H, Alhameedawi A, Alsallameh S, et al. Prevalence of multi-antibiotic resistant bacteria isolated from children with urinary tract infection from Baghdad, Iraq. Microbiol Biotechnol Lett. 2021;50(1):147-56.
98. Abd Al-Hamed Z, Abd Al-Mayahi F. Antibiogram of *Klebsiella pneumoniae* that isolated from clinical and environmental samples in Al-Diwaniyah hospitals. Al-Qadisiyah J Pure Sci. 2021;26(2):13.
99. Alquraishi FE, Al-Saadi ZN, Al-Azzawi JAT. Determination of multi-drug resistant *Klebsiella pneumoniae* isolated from UTI patients in Wasit province, Iraq. Int J Health Sci. 2022;6(S6):7937-49.
100. Al-Fatlawi B, Jasim A. Determining the prevalence of upper and lower urinary tract infections' pathogens and their antibiotic susceptibility profile for adult patients in Al-Diwaniya, Iraq. Iraqi J Pharm Sci. 2022;31:86-90.
101. Jalil MB, Naji Al Atbee MY. The prevalence of multiple drug resistance *Escherichia coli* and *Klebsiella pneumoniae* isolated from patients with urinary tract infections. J Clin Lab Anal. 2022;36(9):e24619.
102. Al-Eqaby J, Al-Charrakh A, Mustafa A. Resistance to third-generation cephalosporins among hospitalized patients with monomicrobial Enterobacteriaceae bacteremia (mEB) in Baghdad, Iraq. J Pharm Negat Results. 2022;13(8):1-9.
103. Raouf FEA, Benyagoub E, Alkhudhairy MK, et al. Extended-spectrum beta-lactamases among *Klebsiella pneumoniae* from Iraqi patients with community-acquired pneumonia. Rev Assoc Med Bras. 2022;68(6):833-7.
104. Hamza O, Omran R. Multidrug drug resistance of *Escherichia*

coli and *Klebsiella* isolated from Iraqi patients and microbiota. J Biosci Med. 2022;10:240-52.
105. Hasan DL, Khalid HM, Mero WM. Phenotypic and molecular study of extended-spectrum β-lactamases producing Enterobacteriaceae from urinary tract infection in Zakho city, Kurdistan Region/Iraq. Acad J Nawroz Univ. 2022;11(3):305-13.
106. Khulaif MJ, Al-Charrakh AH. Detection of class 1 integron and antibiotic resistance of β-lactamase-producing Escherichia coli isolated from four hospitals in Babylon, Iraq. Med J Babylon. 2023;20(2):375-82.
107. Al-Khfaji Z, Sagban S, Al-Musawi A. Prevalence of drug-resistant strains of Escherichia coli and Klebsiella pneumoniae isolated from women with urinary tract infections in Karbala City, Iraq. Egypt J Bot. 2023;63(1):295-303.
108. Mohammed A, Al-Rawi D, Buniya H. Evaluation of antibiotic resistance of Klebsiella pneumoniae isolated from patients in hospitals in Iraq. Acta Microbiol Bulg. 2023;39(4):1-7.
109. Hussein A, Abdulsattar B, Al-Saryi N, Edreesb W. Detection of some β-lactamase genes in Klebsiella pneumoniae isolated from some Baghdad hospitals, Iraq. Al-Mustansiriyah J Sci. 2024;35(2):1-9.
110. Qaddoori BH. Etiological spectrum and antimicrobial resistance among bacterial pathogen association with urinary tract infection in Wasit Governorate/Iraq. Cent Asian J Med Nat Sci. 2023;4(2):552-62.
111. Mohamed I, Al-Taai H. Phylogenetic analysis of Klebsiella pneumoniae isolated from nosocomial and community infection in Diyala, Iraq. Iraqi J Sci. 2023;64(6):2726-40.
112. Mohammed M, Mahmood S. The prevalence of Pks genotoxin among Klebsiella pneumoniae isolated from different clinical samples in Baghdad, Iraq. Iraqi J Sci. 2024;65(7):3716-24.
113. Kamel H, Ali G. Determine the prevalence of pathogens and their antibiotics sensitivity for patients suffering from urinary tract infections in Babylon, Iraq. SAR J Pathol Microbiol. 2024;5(3):59-66.
114. Hussein R, AL-Kubaisy S, Al-Ouqaili M. 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. 2024;17(11):102544.
115. Younus N. Phenotypic and genotypic characterization of multidrug-resistant Escherichia coli and Klebsiella pneumoniae isolated from women with urinary tract infections in Mosul City. Iraqi J Sci. 2024;65(1):24-35.
116. Raheema R, Hadi S, Chabuck Z. Study of some virulence genes from uropathogenic Klebsiella pneumoniae and Pseudomonas aeruginosa isolated in Wasit Province, Iraq. Med J Babylon. 2024;21(1):207-13.
117. Idbeis H, AL-Aidy S. Analysis of bacterial uropathogens and antimicrobial resistance profiles among diabetic patients in Al-Wasit City, Iraq. J Sci Med Life. 2024;2(4):1-9.
118. Hussain E. Detection of virulence iroN gene in Klebsiella pneumoniae in urinary tract infections patients from Iraq. J Basrah Res. 2024;50(1):279.
119. Mohammed S, Mahdi N, Akbar H. Sensitivity of Klebsiella pneumoniae bacteria isolated from the urine of patients with urinary tract infections in Kirkuk City/Iraq. J Prev Diagn Manag Hum Dis. 2024;4(4):1-9.
120. Mohanna Z, AL-Yasseen A. Distribution of carbapenemase genes among carbapenem-resistant Klebsiella pneumoniae isolates from the patients in Najaf, Iraq. Biomed Biotechnol Res J. 2024;8(3):297-304.
121. Hameed A. Peningkatan mengkhawatirkan bakteri MDR pada pasien uremia di Irak. Academia Open. 2024;9(2). DOI: 10.21070/acopen.9.2024.9412.
122. Issa FA. Antibiotic resistance patterns of common uropathogens isolated from females at Zakho City, Kurdistan Region, Iraq. Sci J Univ Zakho. 2024;12(4):490-6. https://doi.org/10.25271/sjuoz.2024.12.4.1395.
123. Al-Jasser AM, Al-Khalil SA, Al-Dosary H. Emergence of carbapenem resistance in E. coli and K. pneumoniae isolates in hospitals in Iraq: a surveillance study. BMC Infect Dis. 2022;22(1):292-8.
124. Al-Sulaiman M, Al-Mashhadani M, Al-Hasan R. Surveillance and control of antibiotic resistance in Iraq: challenges and perspectives. J Glob Antimicrob Resist. 2019;17:170-7.