ISSN: 2588-4115; Infection Epidemiology and Microbiology. 2026;12(2):101-108 doi 10.52547/iem.12.2.101

Antibiotic Resistance and Integron Distribution in *Escherichia coli* Isolated from Urine Samples

ARTICLE INFO

Article Type
Original Article

Authors
Haider Qassim Raheem, *PhD*[1]
Ehsan F. Hussein, *PhD*[2]
Zahra Mohamed Jasim, *MSc*[3,]
Mustafa Raheem Tuama, *MSc*[1]

[1] DNA Research Center, University of Babylon, Al-Karamah Street - 51001, Babylon state, Iraq
[2] College of Science for Women, University of Babylon, Al-Karamah Street - 51001, Babylon State, Iraq
[3] Department of Environmental Health, Collage of Environmental Sciences, Al-Qasim Green University, Babylon, Iraq

**** Correspondence***
DNA Research Center, University of Babylon.
E-mail: haider.qassim@uobabylon.edu.iq

How to cite this article
Raheem H.Q., Hussein E.F., Jasim Z.M., Tuama M.R. Antibiotic Resistance and Integron Distribution in Escherichia coli Isolated from Urine Samples. Infection Epidemiology and Microbiology. 2026;12(2): 101-108.

Article History
Received: May 16, 2026
Accepted: July 14, 2026
Published: July 27, 2026

ABSTRACT

Background: Antimicrobial resistance among uropathogenic *Escherichia coli* represents a growing global health concern and is largely driven by the dissemination of resistance genes through mobile genetic elements such as integrons. This study aimed to investigate antimicrobial resistance patterns and the prevalence of class 1 and class 2 integrons among *E. coli* isolates recovered from patients with urinary tract infections (UTIs).

Materials & Methods: A total of 100 non-duplicate *E. coli* isolates were obtained from urine samples collected from hospitalized patients at Al-Hilla Teaching Hospital, Babylon, Iraq from January to July 2024. Antimicrobial susceptibility testing was performed using standard methods, and the presence of *intI1* and *intI2* genes was determined by polymerase chain reaction (PCR).

Findings: High resistance rates were observed to ampicillin (75%), nalidixic acid (69%), and co-trimoxazole (52%), whereas gentamicin (67%) and amikacin (69%) showed the highest susceptibility rates. Multidrug resistance was detected in 65% of the isolates. PCR analysis revealed that 75% of the isolates carried the *intI1* gene, while *intI2* was detected in 5% of the isolates. The presence of *intI1* was significantly associated with resistance to ampicillin, ciprofloxacin, and co-trimoxazole ($p < .05$).

Conclusion: The high prevalence of integron-associated multidrug resistance among *E. coli* isolates underscores the need for continuous surveillance and strengthened antimicrobial stewardship programs to optimize the management of UTIs.

Keywords: Integrons, *Escherichia coli*, Drug resistance, Urinary tract infections

CITATION LINKS

[1] Kulkarni R, et al. Roles of putative... [2] Chieng CC, Kong Q, Liou NS, Khasriya R, Horsley H. The clinical... [3] Raheem HG, Hussein EF, Rasheed AH. Class 1,2 integron... [4] Sadeghi A, Halaji M, Fayyazi A, Havaei SA. Characterization of... [5] Futoma-Kołoch B, et al. Current insights... [6] Ali N, Ali I, Din AU, Akhtar K, He B, Wen R. Integrons in... [7] Bhat BA, et al. Integrons in... [8] Ghaffari Lashkenari E, Mir MS, Mohammadi M, Javadi K, Halaji M. Investigating the... [9] Hejazi Dehaghani S, Raheem HQ, Latifpour M, Hallaji M. Antibiotic... [10] Clinical and Laboratory Standards... [11] Roe MT, Vega E, Pillai SD. Antimicrobial... [12] Sunde M. Prevalence and characterization of... [13] Kebede D, Shiferaw Y, Kebede E, Demsiss W. Antimicrobial... [14] AbdulHameed H, AbdulJabbar A. Isolation, identification... [15] Abdelwahab GE, et al. Antibiotics resistance in... [16] Alizade H. Escherichia coli in Iran... [17] Fatima S, Akbar A, Irfan M, Shafee M, Ali A, Ishaq Z, et al. Virulence factors and... [18] Ashika S, Sujatha K, Lakshmipriya N, Venkatesh K. Antimicrobial... [19] Alshaikh SA, El-Banna T, Sonbol F, Farghali MH. Correlation... [20] Tesfa K, Belete D, Mulu M, Gelaw A. Antimicrobial susceptibility... [21] Farahat EM, et al. Characterization of... [22] Mostaghimi Talieh M, et al. Phylogenetic analysis... [23] Yalda M, Haniyeh H, Mohammad T, Ali Ehsan S. Prevalence and... [24] Halaji M, Feizi A, Mirzaei A, Sedigh Ebrahim-Saraie H, Fayyazi A, Ashraf A, et al. The global... [25] Gümüş D, et al. High prevalence of class I and... [26] Samane M, et al. Characterization of... [27] Gonbadi MA, Dini Y, Mohammadi M, Pournajaf A, Halaji M. Integrons and... [28] Jiang H, Ran M, Wang X, Chen Q, Wang J, Ruan Z, et al. Prevalence and... [29] Li W, Ma J, Sun X, Liu M, Wang H. Antimicrobial resistance and... [30] Hama Soor Taib A. Distribution and... [31] Shariat A, Fathpoor F. Prevalence of class I... [32] Karimi Dehkordi M, Halaji M, Nouri S. Prevalence of...

Introduction

Urinary tract infections (UTIs) are among the most common bacterial infections, affecting individuals of all ages worldwide and representing a substantial burden on healthcare systems. *Escherichia coli* is the predominant etiological agent of UTIs, accounting for approximately 80–90% of community-acquired infections and a significant proportion of healthcare-associated cases [1, 2]. Owing to its prevalence and adaptability, *E. coli* has become a major focus of clinical and microbiological research, particularly in the field of antimicrobial resistance [3, 4].

The increasing incidence of antimicrobial-resistant *E. coli* has become a serious public health concern, limiting therapeutic options and contributing to treatment failure and recurrent infections. The dissemination of antimicrobial resistance among *E. coli* populations is largely driven by mobile genetic elements, including plasmids, transposons, and integrons, which facilitate horizontal gene transfer. Among these elements, integrons play a pivotal role in the acquisition and expression of antimicrobial resistance genes [5].

Integrons are genetic elements capable of capturing, integrating, and expressing gene cassettes through a site-specific recombination mechanism. Structurally, integrons are characterized by the presence of an integrase gene (*intI*), a recombination site (*attI*), and a promoter that regulates the expression of inserted gene cassettes. Based on differences in the integrase gene sequence, integrons are classified into several classes, of which class 1, class 2, and class 3 integrons are the most studied integrons in clinical settings. Although integrons themselves are not mobile, their association with transposons and plasmids enables their dissemination among bacterial populations [3, 6, 7].

Class 1 integrons are the most prevalent integron class among clinical isolates, which are strongly associated with multidrug-resistant phenotypes because of their ability to capture a wide variety of resistance gene cassettes. Class 2 integrons, often linked to the Tn7 transposon family, are less diverse but also contribute to the spread of antimicrobial resistance, whereas class 3 integrons have been infrequently reported. In uropathogenic *E. coli*, class 1 and class 2 integrons play a key role in mediating resistance to multiple antibiotic classes under antimicrobial selective pressure [7, 8].

Although integrons are increasingly recognized as key drivers of antimicrobial resistance, the prevalence of class 1 and class 2 integrons and their association with antimicrobial resistance patterns vary depending on geographic region, antimicrobial use patterns, and healthcare settings. Therefore, continuous surveillance is essential to understand local resistance mechanisms and guide effective treatment strategies.

Objectives: This study aimed to assess the prevalence of class 1 and class 2 integrons and their association with antimicrobial resistance profiles in *E. coli* isolates from patients with UTIs.

Materials and Methods

Specimen collection and antimicrobial susceptibility testing: This study was conducted over a seven-month period from January to July 2024. During this period, a total of 100 *E. coli* isolates were collected from patients diagnosed with UTIs at Al-Hilla Teaching Hospital, Babylon, Iraq. The diagnosis of UTIs was based on clinical evaluation and laboratory findings consistent with UTIs. Inclusion criteria included all non-duplicate *E. coli* isolates recovered from urine cultures of clinically-diagnosed UTI patients with significant bacteriuria ($\geq 10^5$ CFU/mL). Exclusion criteria comprised repeated isolates from the same patient, samples with polymicrobial growth, and isolates from pa-

tients under 18 years of age. All bacterial isolates were identified using conventional biochemical methods and further confirmed using the VITEK 2 Compact automated identification system [9]. Antimicrobial susceptibility testing was performed using the VITEK 2 Compact system, and susceptibility profiles of the isolates were interpreted in accordance with the guidelines established by the Clinical and Laboratory Standards Institute (CLSI) [10]. The antibiotics evaluated in this study included ampicillin, gentamicin, ceftazidime, amikacin, cefotaxime, co-trimoxazole, nalidixic acid, ciprofloxacin, and meropenem (MAST, UK). Multidrug-resistant (MDR) isolates were defined as isolates exhibiting resistance to at least one antimicrobial agent in three or more distinct antimicrobial classes [8].

DNA extraction and polymerase chain reaction (PCR): Genomic DNA was extracted from *E. coli* isolates using a commercial DNA extraction kit (Bioneer, Republic of Korea) in accordance with the manufacturer's instructions. The purity and concentration of the extracted DNA were assessed using a NanoDrop spectrophotometer (Thermo Scientific, Wilmington, DE, USA). The extracted DNA was used as a template in polymerase chain reaction (PCR) assays to detect genes encoding integrase enzymes associated with class 1 and class 2 integrons (Table 1).

PCR amplification was performed in a total reaction volume of 20 µL, containing 1 µL of each forward and reverse primer, 1 µL of template DNA, 10 µL of PCR master mix (Ampliqon, Denmark), and 7 µL of nuclease-free distilled water. The primer sequences used in this study are listed in Table 1 [11, 12]. Amplification of the class 1 integron gene (*intI1*) was carried out under the following thermal cycling conditions: an initial denaturation at 95 °C for 5 min, followed by 30 cycles of denaturation at 94 °C for 50 s, annealing at 54 °C for 50 s, and extension at 72 °C for 1 min, with a final extension step at 72 °C for 5 min. Amplification conditions for the class 2 integron gene (*intI2*) were identical, except that the annealing temperature was set at 52 °C. PCR products were analyzed by electrophoresis on 1.5% agarose gels for 1 hr at 80 V. The gels were stained with ethidium bromide, and the amplified DNA fragments were visualized under ultraviolet illumination using a gel documentation system. To ensure the reliability of the PCR assays, *E. coli* strains previously confirmed to harbor integron genes were used as positive controls. Negative controls consisted of reaction mixtures containing all PCR components except template DNA, which were included to monitor potential contamination.

Statistical analysis: Data were analyzed using SPSS software, Version 22 (SPSS Inc., Chicago, IL, USA). Associations between the presence of class 1 integrons and antimicrobial resistance were assessed using Chi-square and Fisher›s exact tests, where appropriate. A *p*-value of less than 0.05 was considered statistically significant.

Findings

A total of 100 confirmed *E. coli* isolates were recovered from urine samples collected from hospitalized patients at Al-Hilla Teaching Hospital, Babylon, Iraq. Of these isolates,

Table 1) Oligonucleotide primers used to detect intI1 and intI2 integrase genes by polymerase chain reaction (PCR)

Genes	Sequence	Amplicon Size	Ref.
intI1	F= 3'-GGGTCAAGGATCTGGATTTC -5' R= 3'-ACATGGGTGTAAATCATCGTC-5'	484	[11]
IntI2	F= 3'-GCAAATGAAGTGCAACGC-5' R= 3'-ACACGCTTGCTAACGATG-5'	466	[12]

Table 2) Antibiotic susceptibility pattern of class 1 integron-positive and integron-negative uropathogenic Escherichia coli strains

Class	Antibiotics	Total (n=100)			IntI1-positive (n = 75)			IntI1 -negative (n = 25)			*P* Value*
		S No (%)	I No (%)	R No (%)	S No (%)	I No (%)	R No (%)	S No (%)	I No (%)	R No (%)	
Aminopenicillin class	Ampicillin	20 (20)	5 (5)	75 (75)	10 (13.3)	2 (2.7)	63 (84)	10 (4)	3 (12)	12 (48)	< .001
Aminoglycosides	Gentamicin	67 (67)	13 (13)	20 (20)	52(69.4)	10 (13.3)	13 (17.3)	15(60)	3(12)	7(28)	.38
	Amikacin	69 (69)	9 (9)	22 (22)	57 (76)	3 (4)	15 (20)	12 (48)	6 (24)	7 (28)	.57
Extended-spectrum cephalosporins	Ceftazidime	46 (46)	5 (5)	49 (49)	34 (45.3)	5 (6.7)	36 (48)	12 (48)	0	13 (52)	.9
	Cefotaxime	45 (45)	3 (3)	52 (52)	32 (42.7)	3 (4)	40 (53.3)	13 (52)	0	12 (48)	.81
Folate pathway inhibitors	Co-trimoxazole	41 (41)	7 (7)	52 (52)	27 (36)	3 (4)	45 (60)	14 (56)	4 (16)	7 (28)	.01
Quinolones and fluoroquinolones	Nalidixic acid	27 (27)	4 (4)	69 (69)	20 (26.7)	3 (4)	52 (69.3)	7 (28)	1 (4)	17 (68)	1.00
	Ciprofloxacin	43 (43)	5 (5)	52 (52)	27 (36)	0 (0)	48 (64)	16 (64)	5 (20)	4 (16)	< .001
Carbapenem	Meropenem	100 (100)	0 (0)	0 (0)	75 (75)	0 (0)	0 (0)	25 (25)	0 (0)	0 (0)	-

* Class 1 integron carriage and antimicrobial resistance were compared using Chi-square and Fisher's exact tests, where appropriate. S: sensitive, I: intermediate, R: resistant

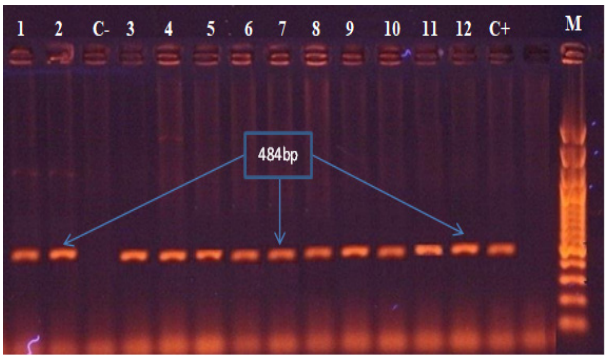

Figure 1) Agarose gel electrophoresis (1.5%) of PCR-amplified *intI1* gene (484 bp) from *E. coli* clinical isolates. Lanes 1–12: clinical isolates; Lane C+: positive control; Lane C–: negative control; Lane M: DNA molecular weight marker. The expected amplicon size of 484 bp is indicated.

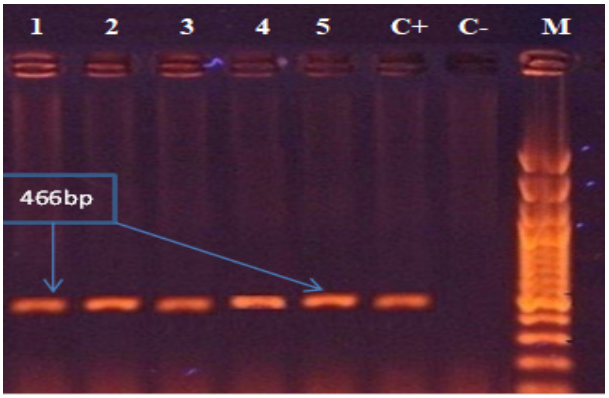

Figure 2) Agarose gel electrophoresis (1.5%) of PCR-amplified *intI2* gene (466 bp) from *E. coli* clinical isolates. Lanes 1–5: clinical isolates; Lane C+: positive control; Lane C–: negative control; Lane M: DNA molecular weight marker.

66 (66%) were obtained from male patients and 34 (34%) from female patients.

Antimicrobial susceptibility testing revealed high resistance rates among the isolates, with the highest resistance observed to ampicillin (75%), nalidixic acid (69%), and co-trimoxazole (52%). In contrast, the highest susceptibility rates were observed to meropenem (100%), amikacin (69%), and gentamicin (67%). PCR analysis of *intI* genes demonstrated that 75% (75 of 100) of the isolates carried *intI1*, whereas 5% (5 of 100) harbored *intI2*. Concurrent carriage of both *intI1* and *intI2* was identified in 2% (2 of 100) of the isolates (Figure 1 and 2). The presence of *intI1* was significantly associat-

ed with resistance to ampicillin (p< .001), ciprofloxacin (p< .001), and co-trimoxazole (p= .01). No significant association was observed between *intI1* carriage and resistance to ceftazidime (p= .90), cefotaxime (p= .81), amikacin (p= .57), or nalidixic acid (p= 1.00). Carriage of *intI2* was not significantly associated with resistance to any of the antimicrobials tested. Antimicrobial resistance profiles stratified by *intI1* status are presented in Table 2.

Discussion

The increasing resistance of bacterial pathogens in both community and hospital settings poses a major global health challenge. Increasing antimicrobial resistance among uropathogenic bacteria has complicated the treatment of UTIs, largely driven by horizontal gene transfer mediated by mobile genetic elements such as integrons. The spread of MDR isolates highlights the clinical importance of integron-associated resistance genes in limiting effective therapeutic options [5].

Antimicrobial susceptibility testing showed high resistance among *E. coli* isolates to ampicillin, nalidixic acid, and co-trimoxazole, whereas carbapenems and aminoglycosides, particularly gentamicin and amikacin, retained higher activity. These results indicate that commonly-used first-line agents may be unsuitable for empirical treatment of UTIs in the studied setting.

Comparable resistance patterns have been reported in previous studies. Kebede et al. (2025) documented high resistance to ampicillin (53.8%) among Gram-negative isolates, while amikacin (83.3%) and gentamicin (81.3%) remained highly effective [13]. Similarly, AbdulHameed and AbdulJabbar (2022) reported very high resistance rates to trimethoprim/sulfamethoxazole (89.5%) and ampicillin (94%), with minimal resistance to amikacin and nitrofurantoin (2.9% each) [14]. Consistent with this study findings, studies conducted in Iran and the United Arab Emirates have documented low susceptibility of *E. coli* isolates to penicillin-class antibiotics, with sensitivity rates below 10% and ranging from 11 to 42%, respectively [15, 16]. Other studies have also reported widespread resistance among uropathogenic *E. coli* (UPEC) isolates to β-lactams, fluoroquinolones, and co-trimoxazole, while maintaining higher susceptibility to amikacin and carbapenems [17-19].

The high level of antimicrobial resistance observed may reflect inappropriate antibiotic use and inadequate infection control practices, highlighting the need for continuous local surveillance and strengthened antimicrobial stewardship to improve UTI management.

In the present study, a high prevalence of MDR *E. coli* was observed, with %65 of the isolates classified as MDR. This finding highlights the growing challenge of managing UTIs caused by resistant pathogens and is consistent with reports from several regions. The MDR rate obtained in this study is closely comparable to those reported in Ethiopia (64.7%) by Tesfa et al. (2025) and Egypt (62.5%) by Farahat et al. (2025), although the isolates in the latter study remained largely susceptible to ciprofloxacin and fully susceptible to meropenem [20, 21]. However, considerably higher MDR rates have been reported in other regions, such as Iran; for example, Mostaghimi et al. (2024) reported an MDR prevalence of 88.5% among UPEC isolates [22], while other studies in the country have reported lower but still substantial rates, such as 54.2% among 633 UPEC isolates [23]. Variation in the prevalence of MDR across studies and regions is likely driven by differences in antibiotic use practices, regulatory control, prescribing behavior, infection control measures, as well as inappropriate antibiotic use without laboratory guidance. These findings highlight the

need for continuous regional surveillance and strengthened antimicrobial stewardship to limit the further spread of MDR *E. coli* in UTI-associated infections.

The present study investigated the contribution of integrons to antimicrobial resistance in *E. coli* isolates and demonstrated a high prevalence of *intI1*. Specifically, 75% of *E. coli* isolates carried the *intI1* gene, whereas *intI2* was detected in only 5%, and concurrent carriage of both classes was observed in 2%. These findings support the known predominance of *intI1* genes among clinical *E. coli* isolates and reinforce their role as important genetic platforms in the dissemination of antimicrobial resistance determinants [24].

The prevalence of integrons examined in the present study was within the wide range described globally. A meta-analysis published in 2019 reported an overall worldwide prevalence of 47% for *intI1* in *E. coli*, with rates varying substantially across regions [24]. Similar distributions have been documented in more recent studies, including those conducted in Turkey and Iran [25-27], in which *intI1* was consistently more prevalent than class 2 integrons, while dual carriage remained relatively uncommon.

The predominance of *intI1* over *intI2* in this study could be explained by the clinical origin of the isolates, as hospitalized patients with UTIs are exposed to higher antimicrobial selective pressure, favoring the maintenance and spread of class 1 integrons [27-29]. This finding is consistent with the findings of Jiang et al. (2025), who reported that class 1 integrons were significantly more prevalent in clinical and hospital-derived *E. coli* isolates than in isolates from community or animal sources [28]. Furthermore, class 1 integrons exhibit higher adaptability and fitness under antibiotic selective pressure due to their more efficient expression and regulation compared to class 2 integrons.

In this study, *intI1* carriage was significantly associated with resistance to ampicillin, ciprofloxacin, and co-trimoxazole but not with cephalosporins, aminoglycosides, or nalidixic acid. In contrast, *intI2* carriage showed no significant association with resistance to any of the antimicrobials tested. Similar correlations between *intI1* and multidrug resistance, particularly to β-lactams and trimethoprim-sulfamethoxazole, have been reported in previous studies conducted in Iran and Iraq [30, 31]. However, variability in *intI1* prevalence and resistance associations across studies may reflect differences in study populations, infection sources, antimicrobial use patterns, and circulating resistance mechanisms, including the involvement of other mobile genetic elements [32]. Overall, the findings of this study align with existing evidence indicating that *intI1* is the most prevalent integron type among clinical *E. coli* isolates, which is often linked to resistance against commonly used antibiotics. These observations emphasize the importance of continued surveillance of integron-mediated resistance as part of broader efforts to monitor and control antimicrobial resistance in UTI-associated *E. coli*.

Conclusions

This study revealed high antimicrobial resistance and multidrug resistance among *E. coli* isolates causing UTIs in Iraq. Resistance to ampicillin, nalidixic acid, and co-trimoxazole was the highest, while gentamicin and amikacin remained more effective. Class 1 integrons were predominant and significantly associated with resistance, underscoring the need for continued surveillance and antimicrobial stewardship.

This study has several limitations. First, only *intI1* and *intI2* were examined, and various cassette regions were not characterized, limiting our ability to link specific gene cassettes with resistance phenotypes. Addition-

ally, the single-center design and modest sample size (n=100) may limit the generalizability of the findings; thus, results should be interpreted with caution.

Acknowledgments
Not applicable.

Ethical permissions: The study protocol was approved by the local ethics committee (Approval No. 338, dated September 1, 2023, Iran) and performed in accordance with the Declaration of Helsinki. Bacterial isolates were obtained from routine urine cultures of patients referred to the clinical microbiology laboratory. Written informed consent was obtained from all adult participants, and no samples from minors were included. Patient data remained anonymized to ensure privacy.

Authors' contributions: HQR, EFH, and ZMJ conceived and designed the experiments. ZMJ, MRT, and EFH performed the experiments. EFH, ZMJ, and MRT analyzed the data. HQR, EFH, and ZMJ contributed to the writing of the manuscript. HQR, and ZMJ contributed to manuscript revision and English editing. All authors read and approved the final manuscript version of the manuscript.

Conflicts of interests: The authors report no conflicts of interest in this work.

Funding/Supports: The authors received partial financial support from the Ministry of Higher Education and Scientific Research / University of Babylon, Iraq. Additional funding was provided by the corresponding author's personal resources. The funders had no role in the study design; data collection, analysis, and interpretation; or manuscript preparation.

Consent to participate: Consent was get from the Health Ministry of iraq.

Declaration of Artificial Intelligence (AI) Use: The authors declare no use of Artificial Intelligence.

References

1. Kulkarni R, Dhakal BK, Slechta ES, Kurtz Z, Mulvey MA, Thanassi DG. Roles of putative type II secretion and type IV pilus systems in the virulence of uropathogenic Escherichia coli. PLoS One. 2009;4(3):e4752.
2. Chieng CC, Kong Q, Liou NS, Khasriya R, Horsley H. The clinical implications of bacterial pathogenesis and mucosal immunity in chronic urinary tract infection. Mucosal Immunol. 2023;16(1):61-71.
3. Raheem HG, Hussein EF, Rasheed AH. Class 1,2 integron genes distribution in Pseudomonas aeruginosa isolated from clinical specimens. Res J Pharm Technol. 2022;15(7):3165-8.
4. Sadeghi A, Halaji M, Fayyazi A, Havaei SA. Characterization of plasmid-mediated quinolone resistance and serogroup distributions of uropathogenic Escherichia coli among Iranian kidney transplant patients. BioMed Res Int. 2020;2020(1):2850183.
5. Futoma-Kołoch B, Sarowska J, Abd El-Salam M, Miñana-Galbis D, Drabová B, Guz-Regner K, et al. Current insights into antibiotic resistance in uropathogenic Escherichia coli and interventions using selected bioactive phytochemicals. Antibiotics. 2025;14(12):1242.
6. Ali N, Ali I, Din AU, Akhtar K, He B, Wen R. Integrons in the age of antibiotic resistance: Evolution, mechanisms, and environmental implications: A review. Microorganisms. 2024;12(12):2579.
7. Bhat BA, Mir RA, Qadri H, Dhiman R, Almilaibary A, Alkhanani M, et al. Integrons in the development of antimicrobial resistance: Critical review and perspectives. Front Microbiol. 2023;14:1231938.
8. Ghaffari Lashkenari E, Mir MS, Mohammadi M, Javadi K, Halaji M. Investigating the relationship between mutations in gyrA and parC genes and resistance to fluoroquinolones in uropathogenic Escherichia coli isolates. Microb Drug Resist. 2025;31(10):317-22.
9. Hejazi Dehaghani S, Raheem HQ, Latifpour M, Hallaji M. Antibiotic resistance and virulence patterns of O25 and O16 serogroups in uropathogenic Escherichia coli. BMC Res Notes. 2025;18(1):157.
10. Clinical and Laboratory Standards Institute. M100-S24: Performance standards for antimicrobial susceptibility testing: Twenty-fourth informational supplement. Wayne, P: Clinical and Laboratory Standards Institute; 2024.
11. Roe MT, Vega E, Pillai SD. Antimicrobial resistance markers of class 1 and class 2 integron-bearing Escherichia coli from irrigation water and sediments. Emerg Infect Dis. 2003;9(7):822-6.

12. Sunde M. Prevalence and characterization of class 1 and class 2 integrons in Escherichia coli isolated from meat and meat products of Norwegian origin. J Antimicrob Chemother. 2005;56(6):1019-24.
13. Kebede D, Shiferaw Y, Kebede E, Demsiss W. Antimicrobial susceptibility and risk factors of uropathogens in symptomatic urinary tract infection cases at Dessie Referral Hospital, Ethiopia. BMC Microbiol. 2025;25(1):126.
14. AbdulHameed H, AbdulJabbar A. Isolation, identification, and antibiotic resistance profile distribution of clinical E. coli in Iraqi patients. Eurasian Med Res Periodical. 2022;8:116-21.
15. Abdelwahab GE, Ishag HZ, Al Hammadi ZM, Al Yammahi SM, Mohd Yusof MF, Al Yassi MS, et al. Antibiotics resistance in Escherichia coli isolated from livestock in the Emirate of Abu Dhabi, UAE, 2014-2019. Int J Microbiol. 2022;2022(1):3411560.
16. Alizade H. Escherichia coli in Iran: An overview of antibiotic resistance: A review article. Iran J Public Health. 2018;47(1):1-12.
17. Fatima S, Akbar A, Irfan M, Shafee M, Ali A, Ishaq Z, et al. Virulence factors and antimicrobial resistance of uropathogenic Escherichia coli EQ101 UPEC isolated from UTI patient in Quetta, Balochistan, Pakistan. BioMed Res Int. 2023;2023(1):7278070.
18. Ashika S, Sujatha K, Lakshmipriya N, Venkatesh K. Antimicrobial resistance properties of uropathogenic Escherichia coli in adults with urinary tract infection and molecular characterisation of virulence factor genes in resistant strains. Int J Acad Med Pharm. 2023;5(5):1002-7.
19. Alshaikh SA, El-Banna T, Sonbol F, Farghali MH. Correlation between antimicrobial resistance, biofilm formation, and virulence determinants in uropathogenic Escherichia coli from Egyptian hospital. Ann Clin Microbiol Antimicrob. 2024;23(1):20.
20. Tesfa K, Belete D, Mulu M, Gelaw A. Antimicrobial susceptibility patterns and associated risk factors of bacterial isolate among urinary tract infection suspected patients at University of Gondar Comprehensive Specialized Hospital, northwest Ethiopia. BMC Infect Dis. 2025;25(1):1723.
21. Farahat EM, Hassuna NA, Hammad AM, Fattah MA, Bakry IA, Khairalla AS. Characterization of multidrug-resistant Escherichia coli isolated from clinical (UPEC) and environmental samples. Mol Biol Rep. 2025;52(1):535.
22. Mostaghimi Talieh M, Pournajaf A, Bijani A, Mohammadi M, Rajabnia M, Halaji M. Phylogenetic analysis, biofilm formation, antimicrobial resistance, and relationship between these characteristics in uropathogenic Escherichia coli. Mol Biol Rep. 2024;51(1):327.
23. Yalda M, Haniyeh H, Mohammad T, Ali Ehsan S. Prevalence and antimicrobial resistance pattern of uropathogenic Escherichia coli at a tertiary care hospital: A retrospective study. J Med Bacteriol. 2024;12(2):43-9.
24. Halaji M, Feizi A, Mirzaei A, Sedigh Ebrahim-Saraie H, Fayyazi A, Ashraf A, et al. The global prevalence of class 1 integron and associated antibiotic resistance in Escherichia coli from patients with urinary tract infection: A systematic review and meta-analysis. Microb Drug Resist. 2020;26(10):1208-18.
25. Gümüş D, Kalaycı Yüksek F, Camadan F, Oral M, Macunluoğlu A, Anğ Küçüker A. High prevalence of class I and class II integrons in uropathogenic E. coli strains (UPECS) and their relationship with antibiotic resistance, phylogeny, and virulence. J Ist Faculty Med. 2022;85(1):77-85.
26. Samane M, Zahra GH, Maryam FP, Peivand N, Fahimeh M, Zahra H, et al. Characterization of integrons, extended spectrum beta lactamases, and genetic diversity among uropathogenic Escherichia coli isolates from Kerman, southeast of Iran. Iran J Microbiol. 2023;15(5):616.
27. Gonbadi MA, Dini Y, Mohammadi M, Pournajaf A, Halaji M. Integrons and efflux pump-mediated antibiotic resistance in multidrug-resistant (MDR) uropathogenic Escherichia coli. BioMed Res Int. 2026;2026:e2158306.
28. Jiang H, Ran M, Wang X, Chen Q, Wang J, Ruan Z, et al. Prevalence and characterization of class I integrons in multidrug-resistant Escherichia coli isolates from humans and food-producing animals in Zhejiang province, China. BMC Microbiol. 2025;25(1):76.
29. Li W, Ma J, Sun X, Liu M, Wang H. Antimicrobial resistance and molecular characterization of gene cassettes from class 1 integrons in Escherichia coli strains. Microb Drug Resist. 2022;28(4):413-8.
30. Hama Soor Taib A. Distribution and molecular characterization of integron classes from Escherichia coli and Klebsiella pneumoniae isolates in Sulaymaniyah province of Iraq. Microbiol Spectr. 2025;13(10):e0175125.
31. Shariat A, Fathpoor F. Prevalence of class I, II, and III integrons in uropathogenic Escherichia coli strains isolated from patients with urinary tract infection in Shiraz, Iran. Jundishapur J Med Sci. 2022;21(4):500-13.
32. Karimi Dehkordi M, Halaji M, Nouri S. Prevalence of class 1 integron in Escherichia coli isolated from animal sources in Iran: A systematic review and meta-analysis. Trop Med Health. 2020;48:16.