J Current Biomed Report
ISSN: 2717-1906
2026, Volume 7, Number 1
Research Paper

Distribution of aminoglycoside-modifying enzyme genes in *Escherichia coli* isolated from urinary tract infections

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Article info:
Received: 05 Dec 2025
Accepted: 13 Mar 2026

Keywords:
Escherichia coli
Urinary tract infection
Aminoglycosides
Antimicrobial resistance

ABSTRACT

Urinary tract infections (UTIs) are among the most common bacterial infections worldwide. *Escherichia coli* is responsible for more than 80% of community- and hospital-acquired UTIs. Aminoglycosides are widely used as first-line therapy for severe Gram-negative infections, but antimicrobial resistance particularly through aminoglycoside-modifying enzymes (AMEs) threatens treatment success. The aim of this study was to investigate the relationship between aminoglycoside resistance and the prevalence of genes encoding modifying enzymes in *Escherichia coli* isolates obtained from urinary tract infections. This cross-sectional study was performed at Babol University of Medical Science. A total of 178 *E. coli* isolates were collected from urine samples of UTI patients. Antimicrobial susceptibility testing was performed according to CLSI guidelines, and the presence of AME-encoding resistance genes (*aac(6')-Ib-cr, aadA1, aphA1, aph(6), Aph(6'-VI*) was examined using PCR. Overall, 71% of isolates were from females and 29% from males; 57% were obtained from children and 43% from adults. Phenotypic results showed high susceptibility to gentamicin (78%) and amikacin (75%), while resistance was 20.8% and 18%, respectively. The most frequent AME gene was *aac(6')-Ib-cr* (37%), followed by *aphA1* (15%) and *aadA1* (14%). Statistical analysis showed a significant association only between *aphA1* and gentamicin resistance. Although aminoglycoside susceptibility remains high, AME genes are already disseminating among uropathogenic *E. coli*, indicating the risk of future resistance expansion. Phenotypic and genetic data provide valuable guidance for treatment and resistance monitoring.

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1. Introduction

Urinary tract infections (UTIs) are one of the most prevalent bacterial infections globally and represent a major cause of morbidity in both community and hospital settings. *Escherichia coli* accounts for more than 80% of UTIs and remains the most significant uropathogen. Strains of *E. coli* capable of causing UTIs are termed uropathogenic *Escherichia coli* (UPEC). UPEC are strains of *E. coli* that deviate from their typical role as components of the gut microbiota, proliferate, and persist within the urinary tract. They possess a distinct array of virulence factors and strategies that enable them to colonize the urinary system and cause disease. These *E. coli* strains are consistently associated with UTIs and represent one of the most common pathotypes among extraintestinal pathogenic *E. coli* (ExPEC) [1].

Antimicrobial therapy usually is the first strategy in treating a UTI but the increased rate of antimicrobial resistance (AMR) among UPEC has become a major concern, especially the global spread of resistance to aminoglycosides, which are prescribed as broad-spectrum antibiotics for the treatment of Gram-negative infections, including UTI caused by *E. coli* [2,3]. Aminoglycosides have been explored as potential therapeutic agents not only for bacterial infections but also for fungal infections, parasitic diseases and certain genetic disorders. Nevertheless, their principal clinical application today remains the treatment of Gram-negative bacterial infections, including those caused by *Acinetobacter baumannii*, *E. coli*, *Klebsiella pneumoniae*, and *Pseudomonas aeruginosa* [4].

E. coli has acquired multiple determinants that confer resistance to aminoglycosides. The mechanisms underlying aminoglycoside resistance include enzymatic inactivation of the drug, active efflux, reduced outer-membrane permeability, and amino-acid substitutions in ribosomal proteins. Among these, enzymatic inactivation by a large family of aminoglycoside-modifying enzymes (AMEs) is the most common mechanism [3]. This enzyme family comprises three major subclasses: aminoglycoside acetyltransferases (AAC), aminoglycoside nucleotidyltransferases (ANT), and aminoglycoside phosphotransferases (APH), which represent the predominant types reported [4]. Although more than 85 AMEs have been identified, only a subset is responsible for clinically significant aminoglycoside resistance. The most frequently detected AME-encoding genes in *E. coli* include *aac(3)-II*, followed by *aac(6')-I*, *ant(3')-I, aph(3')-II*, and *ant(2")-I* [3].

Given the clinical importance of these resistance mechanisms, identifying the genetic basis of aminoglycoside resistance and recognizing emerging determinants is essential for improving therapeutic strategies. Furtheremore, aminoglycoside resistance mediated by AME-encoding genes is increasing among *E. coli* isolates involved in UTIs, limiting treatment options and increasing healthcare costs. Determining the correlation between phenotypic resistance and AME genes enables better antibiotic selection and infection-control strategies. Therefore, in the present study conducted for the first time in northern Iran we aimed to investigate the relationship between aminoglycoside resistance and the prevalence of genes encoding aminoglycoside-modifying enzymes in *E. coli* isolates recovered from UTIs in children.

2. Materials and Methods

2.1 Study design and bacterial isolation

This cross-sectional study examined 178 *E. coli* isolates collected from urine samples in hospitals affiliated to Babol University of Medical Sciences, North of Iran. In order to determine the definitive identity and confirm the diagnosis of *E. coli* strain, all isolates were identified by standard biochemical tests (specific colony form, Gram staining, biochemical tests such as MR-VP, citrate, urease, nitrate reduction, motility at 37 °C, indole production, and gas production) and their confirmation was established in a previous study conducted by our team. After the phenotypic confirmation of the isolates, in order to finally confirm the genus and species of *E. coli*, the polymerase chain reaction (PCR) method was used to amplify the uid (beta-glucuronidase) gene [5]. In order to preserve the bacterial strains for a long time, stock samples were prepared using BHI liquid culture medium containing 20% sterile glycerol and finally all samples were stored in a freezer at -20°C for further studies. Notably, a subset of UPEC isolates was selected from a collection of *E. coli* strains obtained between 2021 and 2024 [6].

2.2 Antimicrobial susceptibility testing (AST)

The Antimicrobial susceptibility test of the strains was performed according to the recommendation of the Institute of Clinical and Laboratory Standards (CLSI) using disc diffusion method. This method was performed using the special culture medium Muller-Hinton Agar manufactured by Merck, Germany and the pattern of bacterial sensitivity to gentamicin and amikacin antibiotic discs was determined. The following antibiotic disks were used: gentamicin (GM, 10 µg) and amikacin (AN, 30 µg). *E. coli* ATCC 25922 was used as the quality control strain [7].

2.3 Genomic DNA extraction and molecular detection of AME genes

Genomic DNA was extracted from freshly cultured colonies as previously described. The presence of AME genes including *aac(6')-Ib-cr, aadA1, aphA1, aph(6), Aph(6'-VI* were determined using PCR assays (Table 1). The thermal cycling conditions were: initial denaturation at 94°C for 5 minutes, then 30 cycles (94°C

for 45 seconds, annealing temperature suitable for each gene for 30 seconds, extension at 72°C for 30 seconds) and final extension at 72°C for 5 minutes [8]. The PCR amplicons were separated on a 1.5% agarose gel and visualized under UV illumination.

2.4 Statistical analysis

A chi-square test and Fisher's exact test were used to analyze group differences in frequency distributions in SPSS (version 27.0; IBM Corp., Armonk, NY, USA). Comparisons of score distributions were performed with the Mann–Whitney U test. Statistical significance was defined as a p value of <0.05.

3. Results

3.1 Study population

In this study, out of 178 confirmed UPEC strains, 57/3 % (102/178) were isolated from children and 42/7% (76/178) from adults. Additionally, 71/3% (127/178) of the isolates were obtained from female patients, while 28/9% (51/178) were from male patients.

3.2 Phenotypic antimicrobial resistance of UPEC isolates

The antimicrobial susceptibility patterns of 178 UPEC isolates were determined against the tested antibiotics. The results indicated that a high proportion of isolates were susceptible to gentamicin (78.7%) and amikacin (75.3%). Conversely, resistance to these antibiotics was observed in 20.8% of isolates for gentamicin and 18% for amikacin. The detailed antibiotic resistance rates of the UPEC isolates are summarized in Table 2.

3.3 Presence of AME genes

The examination of 178 *E. coli* isolates revealed that the aac(6′)-Ib-cr gene was the most prevalent, detected in 37.1% of the isolates (66/178). This was followed by *aphA1*, identified in 15.2% (27 isolates), and *aadA1*, detected in 14% (25 isolates). In contrast, the *aph(6)* and *aph(6')-VI* genes exhibited the lowest frequencies, occurring in only 1.1% (2 isolates) and 0.6% (1 isolate), respectively.

Based on the data presented in Table 3, the most frequent co-occurrence of aminoglycoside-modifying enzyme genes was *aac(6')-Ib-cr* + *aph(6)*, identified in 11 isolates (6.2%), followed by *aac(6')-Ib-cr* + *aadA1* in 8 isolates (4.5%).

3.4 Analysis of AME Resistance Genes in Relation to Aminoglycoside Susceptibility Profiles

As demonstrated in Table 4, analysis of the resistance genes in the UPEC isolates, showed that *aac(6')-Ib-cr* exhibited the highest prevalence among both gentamicin- and amikacin-resistant and -susceptible strains, making it the most common determinant of resistance compared with the other genes studied.

Table 1. Primers included in the PCR assays used for specific identification of AME gene

Aminoglycoside-modifying enzyme genes	Primer sequence (5'-3')	Size products (bp)	Reference
aphA1-f	AAACGTCTTGCTCGAGGC	461	[19]
aphA1-r	CAAACCGTTATTCATTCGTGA		
Aph(6')-VI-f	AGCGAAAATGTTGAGTTGGCT	399	[20]
Aph(6')-VI r	TCCGTGATATCGCCATGAGA		
aph (6)-f	GAGCGCACCTTCGACTATGC	248	[21]
aph (6)-r	GCCATGGCGTTTACGGCCAG		
aadA1-f	TTATTTGCCGACTACCTTGGTG	792	[22]
aadA1-r	ATGAGGGAAGCGGTGATCG		
aac(6')-Ib-f	CTCGAATGCCTGGCGTGTTT	482	[23]
aac(6')-Ib-r	TTGCGATGCTCTATGGGCTA		

Table 2. Antibiotics resistance pattern in UPEC isolates

Antibiotic	R No. (%)	I No. (%)	S No. (%)	Total No. (%)
Gentamicin	37 (20.8)	1 (0.6)	140 (78.7)	178 (100)
Amikacin	32 (18.0)	12 (6.7)	134 (75.3)	178 (100)

R: Resistant; I: Intermediate; S: Susceptible

Table 3. Frequency of aminoglycoside-modifying enzyme gene combinations in studied isolates.

Aminoglycoside-modifying enzyme genes	No. (%)
aac(6')-Ib-cr+ aph(6)	11 (6.2)
aac(6')-Ib-cr+ aadA1	8 (4.5)
aph(6) + aada1	6 (3.4)
aac(6')-Ib-cr+ aph(6')-VI	2 (1.1)
aph(6) + aph(6')-VI	2 (1.1)
aph(6) + aphA1	1 (0.6)
aadA1+ aph(6')-VI	1 (0.6)

Table 4. Antibiogram results and AME gene distribution in UPEC isolates

Antibiotic susceptibility		*aac(6')-Ib-cr* No. (%)	*aadA1* No. (%)	*aphA1 No. (%)*	*aph(6')-VI No. (%)*	*aph(6) No. (%)*
Gentamicin (37 isolates)	R	15 (40.5%)	9 (24.3%)	4 (10.8%)	0	0
Gentamicin (140 isolates)	S	50 (35.7%)	16 (11.4%)	22 (15.7%)	1 (0.7%)	2 (1.4%)
Gentamicin (1 isolate)	I	1 (100%)	0	1 (100%)	0	0
Amikacin (32 isolates)	R	11 (34.4%)	3 (9.4%)	4 (12.5%)	0	0
Amikacin (134 isolates)	S	53 (39.5%)	21 (15.7%)	22 (16.4%)	1 (0.75%)	2 (1.5%)
Amikacin (12 isolates)	I	2 (16.7%)	1 (8.3%)	1 (8.3%)	0	0

R: Resistant; I: Intermediate; S: Susceptible

In contrast, the *Aph(6')-VI* and *aph(6)* genes demonstrated the lowest frequencies and were either not detected or identified only at very minimal levels across most groups. The statistical analysis showed that, except for the association between the presence of the *aphA1* gene and gentamicin resistance, the other observed associations were not statistically significant ($P > 0.05$)

4. Discussion

UTI is the most common infection in communities after respiratory infections. Studying and collecting data on the prevalence of microbial species and their antibiotic susceptibility is effective in guiding empirical treatment [9]. The emergence of antibiotic resistance among hospital-associated pathogens remains one of the most serious challenges in modern medicine [10]. Aminoglycosides remain essential agents for the treatment of serious bacterial infections. Nevertheless, the increasing prevalence of aminoglycoside resistance represents a significant concern in both community and hospital settings [11,12].

Investigating antibiotic resistance patterns is crucial for epidemiological surveillance, informing therapeutic strategies, and guiding the management and prevention of bacterial diseases. In the present study, a high percentage of UPEC isolates were susceptible to gentamicin (78.7%) and amikacin (73.3%), whereas only 20.8% and 18% were resistant, respectively. These findings underscore the substantial efficacy of both agents in our patient population and highlight their continued value as reliable therapeutic options. Comparisons with previous studies provide additional insight into resistance dynamics. Ghotaslou et al. reported resistance rates of 51% for gentamicin and 8% for amikacin [13]. While amikacin resistance in their study closely mirrors our findings, gentamicin resistance in our isolates was markedly lower. These differences may reflect regional variations in prescribing practices, selective pressure associated with antibiotic consumption, or differences in the circulating bacterial populations. Hemati et al. (2020) reported even lower resistance rates for gentamicin (10%) and amikacin (3.9%) [14]. Such discrepancies may arise from geographic variation, differences in sampling populations (outpatients versus hospitalized patients), or local antibiotic stewardship policies. Nevertheless, both Hemati's study and our findings emphasize the continued clinical relevance of gentamicin and amikacin while highlighting the importance of ongoing regional resistance surveillance. Ghamari et al. (2024), in Tehran, documented markedly high aminoglycoside resistance, with 64.6% of isolates exhibiting resistance. The highest resistance was observed to kanamycin (44.6%) and gentamicicin (38.5%), whereas amikacin remained the most effective aminoglycoside with a resistance rate of only 4.6% [15].

In our investigation, among 178 *E. coli* isolates, aac(6′)-1b-cr emerged as the most prevalent resistance gene (37.3%), followed by *aphA1* (15.2%) and *aadA1* (14%). Conversely, *Aph(6')-VI* and *aph(6)* were detected at very low frequencies (0.6% and 1.1%, respectively), indicating their limited contribution to aminoglycoside resistance in this setting. These findings underscore the key role of *aac(6')-1b-cr* in mediating aminoglycoside resistance among the isolates studied. Given the low prevalence of certain genes and the lack of statistically significant associations in most cases (except between *aphA1* and gentamicin resistance), it is plausible that additional resistance mechanisms contribute to gentamicin and amikacin resistance in this region and warrant further investigation. In Ghamari et al.'s study, *aac(3)-Iva* was the most prevalent gene (49.2%), followed by *aac(6)-Ib (40%), aac(3)-IIa (32.3%),* and *ant(2)-Ia* (30.8%), suggesting a dominant contribution of these genes to aminoglycoside resistance [15].

Soleimani et al., examining a*ac(3)-IIa* and plasmid profiles in UPEC strains, reported the highest resistance to tobramycin (24.6%) and confirmed the presence of the gene in 51 isolates. While resistance patterns vary across studies, both investigations emphasize the important contribution of resistance genes to the dissemination of aminoglycoside resistance [16]. Akrami et al. (2020) also reported *ant(2")-Ia* and *aph(3')-Ia* as the most prevalent aminoglycoside resistance genes, followed by *aac(3')-IIa*. Gene coexistence for example *ant(2")-Ia* with *aac(3')-IIa* or *aph(3')-Ia* was frequently observed among isolates nonsusceptible to tobramycin and gentamicin. These

findings highlight the role of gene combinations in shaping phenotypic resistance. Moreover, Akrami et al. emphasized that gentamicin remains effective against biofilm-forming and multidrug-resistant isolates, a conclusion consistent with our findings and underscoring gentamicin's clinical relevance [17].

Further international studies demonstrate significant geographic and genetic variability. Xi et al. (China, 2020) reported prevalences of *aac(6')-Ib* and *aac(3)-II* of 32.7% and 59.2% [18]; Masamori et al. (Iraq, 2019) identified *aac(6')-Ib* in 98.3% of isolates; and Azyu et al. (China, 2012) found *aac(3)-II* to be predominant. These differences may reflect local genetic diversity, antibiotic usage patterns, plasmid dissemination, and methodological variation.

Abo-State et al. (Egypt) identified *aac(3')-IIa (40%), aac(6')-Ib (30%), aph(3')-Ia (23.3%), ant(2")-Ia (20%), aph(3') (13.3%)*, and *aac(3')-Ib* (6.6%) as the most common genes. Amikacin was found to be the most effective agent against *E. coli*, consistent with our findings and demonstrating that, despite the presence of AME genes, susceptibility to certain aminoglycosides particularly amikacin and gentamicin remains well preserved [11].

Overall, despite regional and genetic variability, our findings highlight the high susceptibility of isolates to amikacin and gentamicin and the notable prevalence of *aac(6')-1b-cr*. From a clinical perspective, integrating phenotypic and genotypic insights is essential for informed therapeutic decision-making and resistance surveillance, enabling the monitoring of antibiotic effectiveness and local resistance gene trends. This, in turn, supports more effective treatment strategies and helps limit the spread of antimicrobial resistance.

This study has several limitations. First, only a limited panel of AME genes was screened. Second, non-enzymatic mechanisms including efflux pumps and reduced membrane permeability were not assessed.

In summary, this study indicates that most isolates were susceptible to amikacin and gentamicin, with only a small proportion exhibiting resistance, underscoring the clinical relevance of these agents as therapeutic options for urinary tract infections. Analysis of AME genes showed that the most prevalent resistance determinant in the study population was *aac(6')-Ib-cr* with a frequency of 37.1%, followed by *aphA1* and *aadA1*.

Other genes, including *aph(6)* and *aph(6')-VI*, were detected at low frequencies, suggesting their limited contribution to resistance. The data also imply that mechanisms beyond AME genes may play a role in resistance to amikacin and gentamicin, warranting further investigation. Overall, integrating phenotypic and genotypic resistance data provides valuable guidance for therapeutic decision-making, regional resistance surveillance strategies, and efforts to limit the spread of antimicrobial resistance. The results highlight the need for continuous monitoring of resistance patterns and close attention to the role of AME genes in UPEC isolates, while confirming the reliability of amikacin and gentamicin as effective treatment options.

Declaration of artificial intelligence (AI) in the writing process

The authors declare whether AI or AI-assisted technologies were used during the preparation of this manuscript. If used, AI tools were employed solely to improve language quality, grammar, readability, and organizational structure. The authors carefully reviewed and edited all AI-generated content and take full responsibility for the accuracy, integrity, and originality of the final manuscript. No AI tool was used to generate, analyze, or interpret scientific data or images, or to draw scientific conclusions. The use of AI-assisted technologies complies with current publication ethics recommendations and journal policies.

Authors' contributions

AL, MH and MM: designed the study and supervised data acquisition. ZA ans AL: data acquisition and results analysis. AL, MH and MM: performed the statistical analysis and drafted the initial manuscript. HS, MH and MM: critically revised the manuscript for important intellectual content. All authors read and approved the final version of the manuscript.

Conflict of interest

No potential conflict of interest was reported by the authors.

Ethical declarations

The study protocol was reviewed and approved by the institutional ethical board (Approval No. IR.MUBABOL.HRI.REC.1402.269). However, because we only used leftovers from clinical specimens, the local ethics committee waived the need for informed consent.

Financial support

This study, part of Amirreza Larijani 's MD thesis, was supported by Babol University of Medical Sciences in Babol, I.R. Iran (grant number: 724135282).

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