Original Article

Identification of Phylogenetic Groups and Antibiotic Resistance in *Escherichia coli* Isolated from Urinary Tract Infections in Basrah, Iraq

Hayfaa Al-Musawi, Amani Al-Abdullah[1], Labeed Al-Saad[2]

Department of Biology, College of Science, University of Basrah, Basrah, Iraq, [1]Department of Pathological Analyses, College of Science, University of Basrah, Basrah, Iraq, [2]Department of Intelligent Medical Systems and Information, College of Science and Information Technology, University of Basrah, Basrah, Iraq

Abstract

Background: *Escherichia coli* is the most common causal agent for urinary tract infections (UTIs), but several other bacteria can also cause UTIs. Phylogenetic classification is essential for understanding *E. coli* groups as well as the relationships between different strains. **Objectives:** The aim of the study was to identify phylogenetic groups and antibiotic resistance in uropathogenic *E. coli*. **Materials and Methods:** A study was done on patients with UTIs during the period from March 1, 2021, to the end of June 2021, and 57 *E. coli* isolates were included in the study. Analyzed for phylogenetic groups using the quadruplex-PCR technique. VITEK2 was used to assess the antibiotic resistance. The Chi square was used to estimate the relationship between variables, and $P < 0.05$ was regarded as significant. **Results:** The current study shows phylogenetic group D was the most common group (29.82%). Phylogroups A and B2 were the next with (24.56%) and (12.28%). Phylogroup F, the unknown group, and Clade I were the least common, with 8.8%, 5.26%, and 3.5%, respectively. Phylogroups C or E were not observed in this study. The study found a significant relationship between certain antibiotics and *E. coli* phylogenetic groups. Specifically, Piperacillin ($P = 0.011$), Ticarcillin and Cefepime ($P = 0.003$), Aztreonam ($P = 0.004$), and Ceftazidime ($P = 0.006$) were all significantly associated with certain phylogenetic groups of *E. coli*. This suggests that the resistance patterns of *E. coli* may be linked to their phylogenetic groups. **Conclusions:** Further research is needed to explore this relationship and its potential implications for the treatment of *E. coli* infections.

Keywords: Antimicrobial resistance, phylogenetic groups, urinary tract infection, uropathogenic *Escherichia coli*

Introduction

Urinary tract infections (UTIs) are common bacterial illnesses that can be caused by many different types of bacteria. Gram-negative bacteria, like *E. coli*, are the most common cause of UTIs. Accounting for 80%–90% of cases, as reported in studies conducted in various populations. Other Gram-negative bacteria that can cause UTIs include *Klebsiella pneumoniae*, *Proteus mirabilis*, and *Pseudomonas aeruginosa*. Gram-positive bacteria, such as *Enterococcus faecalis* and *Staphylococcus saprophyticus*, can also cause UTIs, but they are less common than Gram-negative bacteria. The elevated incidence of *E. coli* in UTIs is thought to stem from its capacity to colonize the gastrointestinal tract and migrate to the urinary tract, in addition to its potential to stick to and infiltrate urinary tract epithelial cells.[1] Clinical classifications of UTIs include complicated and simple. Acute kidney injury (AKI) is common among patients hospitalized AKI is common among patients hospitalized Patients who have renal failure, anatomical abnormalities of the urinary tract, or who use medical devices like catheters and are at

Access this article online	
Quick Response Code:	**Website:** https://journals.lww.com/mjby
	DOI: 10.4103/MJBL.MJBL_1101_23

Address for correspondence: Dr. Hayfaa Al-Musawi,
Department of Biology, College of Science,
University of Basrah, Basrah, Iraq.
E-mail: hifaamansor@gmail.com

Submission: 28-Jul-2023 **Accepted:** 07-Dec-2023 **Published:** 29-Mar-2025

How to cite this article: Al-Musawi H, Al- Abdullah A, Al-Saad L. Identification of phylogentic groups and antibiotic resistance in *Escherichia coli* isolate from urinary tract infections in Basrah, Iraq. Med J Babylon 2025;22:168-74.

risk for developing complicated UTIs, which require prolonged treatment. On the other hand, simple UTIs typically affect healthy individuals with no underlying medical conditions and can be treated with a short course of antibiotics.[2] Clermont *et al*.[3] used triplex PCR to find the genes TspE4.C2, chuA, and yjaA. This helped them split extracellular *E. coli* bacteria into the B2, B1, A, and D groups. This was done with a quick and easy evolutionary grouping method based on the genes TspE4.C2, chuA, and yjaA. Clermont *et al*.[4] added a gene target to the system to increase its effectiveness. The three arpA genes that were already present were supplemented by a fourth gene. Compared to the earlier approach by Clermont *et al*.,[3] this produced a quadruple polymerase reaction with a better level of resolution. Using molecular techniques like quadruplex PCR, *E. coli* strains can be divided into eight phylogroups: A, B1, B2, C, D, E, F, and one cryptic clade I.[4] Commensal strains of *E. coli* are primarily found in phylogroups A and B1, while pathogenic strains are more commonly found in phylogroups B2 and D, although some pathogenic strains can also be found in other phylogroups. Understanding the phylogenetic classification of *E. coli* isolates is important for studying the epidemiology and pathogenesis of *E. coli* infections, as different phylogroups may have distinct virulence factors and clinical presentations.[5] Today pathogenic bacteria's resistance to various drugs is one of the major barriers to the prevention and treatment of infectious diseases. Various strategies are used by bacteria to survive UTIs, which can only be treated with antibiotics, but the spread of MDR bacterial strains globally has raised serious public health problems, especially for people who suffer repeated UTIs.[6] The cost of treatment and hospitalization is increased by using a wide range of antibiotics, like cephalosporins, fluoroquinolones, and aminoglycosides.[7] To produce current epidemiological data, it is crucial to screen for susceptibility at each site.[8,9] It seems that the prevalence and distribution of phylogenetic groups and antibiotic susceptibility patterns among uropathogenic *E. coli* (UPEC) isolated from patients in Basrah, Iraq, have not been well studied. Therefore, this study was carried out with the purpose of determining the occurrence and dispersion of phylogenetics along with their antibiotic resistance profiles, among uropathogenic *E. coli* (UPEC) strains that were obtained from patients in diverse regions of Basrah, Iraq.

Materials and Methods

In this study, 57 *E. coli* isolates total were analyzed for phylogenetic groups using the new method developed by Clermont *et al*. The quadruplex polymerase chain reaction (PCR) technique was used to identify phylogenetic groups. VITEK2 Compact automated system methods (bioMérieux, Marcy-l'Étoile, France) were used to assess the susceptibility to identify antibiotic resistance.

Inclusion criteria

In this study, patients of any age with symptoms of UTIs were included.

Exclusion criteria

This study excluded patients who had recently experienced UTIs symptoms, had a recent history of antibiotic use, or had received antibiotic medication at least 2 weeks prior to sampling.

Bacterial isolates

The study was conducted in Iraq from March 2020 until July 2021. The researchers collected urine samples from 200 patients affected by UTIs from different hospitals in Basrah city. Each patient's consent was obtained before specimen collection, and the study was approved by an institutional committee. The urine samples were collected using a clean-catch midstream technique and were placed in sterile screw-capped universal containers. The collected samples were then inoculated onto MacConkey agar and Hi-chromo *E. coli* agar and were incubated at 37°C for 24 h. *Escherichia coli* isolates were identified based on the positive cultures for UTI (103-105 cfu/mL) and were confirmed using Gram staining and standard biochemical tests including 1) methyl red (MR), 2) Voges-Proskauer (VP), TripleSugar Iron (TSI), 3) agar, indole, production, and 4) Simmons' citrate agar (Merck, Darmstadt, Germany). In Brain–heart infusion broth medium supplemented with 15% glycerol, isolated *E. coli* bacteria were kept at −20°C.

DNA extraction

To extract genomic DNA from each isolate, DNA was taken from a single pure colony of strain. The pure bacterial colonies were then put into 5 mL of the brain–heart infusion broth and incubated at 37°C for 24 h. After the final, DNA was taken from bacteria by using a DNA purification kit and following the instructions from the company that made it (Geneaid, New Taipei City, Taiwan). A 1% agarose gel was used to visualize the extracted DNA which then kept at −20°C until it was needed again. After that, PCR methods were used to check all of the *E. coli* isolates' DNA against the study's target genes. Each PCR reaction ended up being either 25 μL or 50 μL in size. Before it was used, the PCR mix was made up of a Go Tag Green Master Mix (2x) solution that was warmed at room temperature and mixed with a vortex. Before using them, primer solutions were also mixed with a blender, and the DNA in the supernatant was saved for PCR.

Quadroplex-PCR phylogenetic analysis

In this study, the Quadruplex-PCR approach was utilized to identify one of the eight major phylogenetic

Table 1: Primer's sequence of *E. coli* phylogroups

Gene	Name of the primer	Primer sequence (5′– 3′)	Size of the product (bp)
chuA	AchuA.1	%5′-ATGGTACCGGACGAACCAAC-3′	288
	AchuA.2	&5′-TGCCGCCACTACCAAAGACA-3′	
yjaA	AyjaA.1b	^5′-CAAACGTGAAGTGTCAGGAG-3′	211
	AyjaA.2b	*5′-AATGCGTTCCTCAACCTGTG-3′	
TspE4.C2	ATspE4C2.1b	/5′-CACTATTCGTAAGGTCATCC-3′	152
	TspE4C2.2bA	*5′-AGTTTATCGCTGCGGGTCGC-3	
arpA	AceK.fA	*5′-AACGCTATTCGCCAGCTTGC-3'	400
	ArpA1.rA	*5′-TCTCCCCATACCGTACGCTA-3′	

Table 2: Amplification conditions

Step	Temperature	Time	No. of cycles
Initial denaturation	94	4 min	30
Denaturation	94	5 s	30
Annealing	59	20 s	30
Extension	72	35 s	30
Final extension	72	5 min	30

groups of *E. coli* using primers that were previously conducted by Clermont *et al*.[4] Table 1 shows the primer sequences that were used in this work. The reaction mixture involved24 µL of GoTaq Promega Green Master Mix (Promega USA), 4 µL of DNA template, 8 µL from four primers (1 µL each of forward primer and 1 µL reverse primer). The reaction mixture was completed to 50 µl usingnuclease-free water (NFW) [Table 2]. The PCR product was visualized using 2% agarose gel electrophoresis. The PCR products were kept at −20°C. The phylogenetic analysis of pathogenic *E. coli* was not performed in this study isolates were visualized in Figure 1.

Antimicrobial susceptibility test

In this study, the effectiveness of antibiotics against *E. coli* isolates obtained from UTI patients was determined in this investigation using the AST laboratory procedure. The VITEK2 Compact automated system techniques (bioMérieux, France) were employed for this purpose, as previously described in Bitew *et al*.[10]

Statistical analysis

Chi-square analysis was used to do statistical analysis and identify variations in the distributions of the investigated determinants (SPSS software, version 2.1, IBM, NC, USA). At $P < 0.05$, the significant level was established.

Ethical approval

The study was conducted in accordance with the ethical principles that have their origin in the Declaration of Helsinki. It was carried out with patients' verbal and analytical approval before the sample was taken. The study protocol and the subject information and consent form were reviewed and approved by a local ethics committee according to document number 427 (including the number and the date April 19, 2021) to get this approval.

Results

The *E. coli* was the most common gram-negative bacteria isolated from the 200 uropathogen samples collected. Specifically, 57 *E. coli* isolates were obtained from 120 positive growths, which accounted for 68.4% of the total growths obtained. These findings suggest that *E. coli* is a major contributor to UTIs in the population studied, particularly among individuals in the reproductive age range [Figure 2]. A total of 57 isolates were identified as conventional *E. coli* using the biochemical method. All of these isolates were included for further investigation, including phylogenetic analysis. The technique described by Clermont *et al*.,[4] was used to determine the phylogenetic groups of the collected *E. coli* isolates. With 17 strains (29.82%), phylogenetic group D was the most prevalent, followed by phylogroups A. 14 (24.56%) were present. About 9 (15.78%) in B2 and 7 (12.28%) in B1 Whereas Clade I have two strains (3.5%), the unknown group has 3 (5.26%), and phylogroup F has 5 (8.8%). Groups C and E were not found in any of the isolates, however. The appendix of the study displays the antibiotic susceptibility pattern for Gram-negative bacteria and presents the results of an antimicrobial susceptibility test conducted on *E. coli* Gram-negative isolates obtained from UTI patients (n = 57) against 13 antimicrobial agents. The findings of the investigation indicated that the isolates exhibited resistance to penicillin antibiotics, including Piperacillin and ticarcillin, with resistance rates of 72% (41 isolates) and 64.90% (37 isolates), respectively. Cephems, such as Cefepime and ceftazidime, demonstrated resistance rates of 72% (41 isolates) and 61.40% (35 isolates), respectively. The resistance rate to Monocyclin, represented by azatreonam, was found to be 68.40% (39 isolates). Regarding quinolone

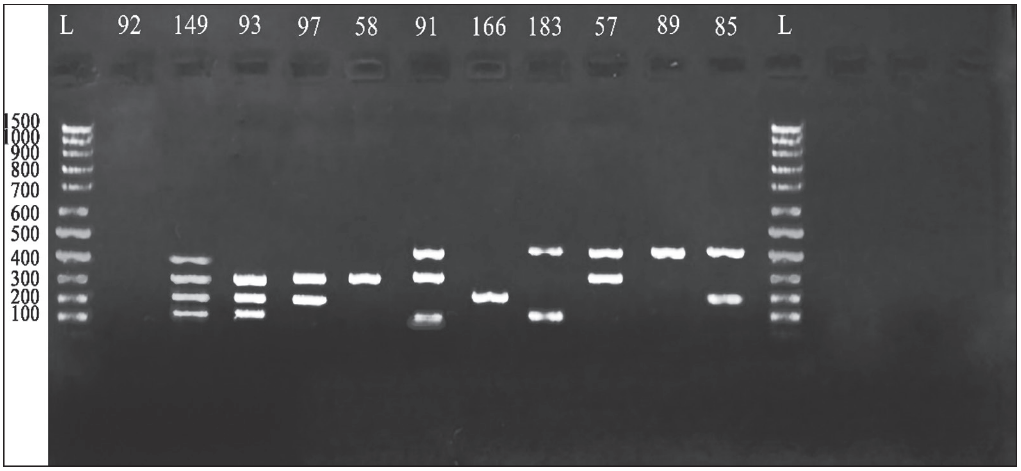

Figure 1: Quadruplex-PCR profiles of the New Clermont Phylogenetic Group Method. Lane 1, unknown (− − − −); lane 2, unknown (+ + + +); lane 3, group B2 (− + + +); lane 4, group B2 (− + + −); lane 5, group F (− + − −); lane 6, group D (+ + − +); lane 7, clad 1 (− −+ −); lane 8, group B1 (+ − − +); lane 9, group D (++− −); lane 10, group A (+ − − −); lane 11, group A (+ − + −). L: DNA Ladder (100 bp)

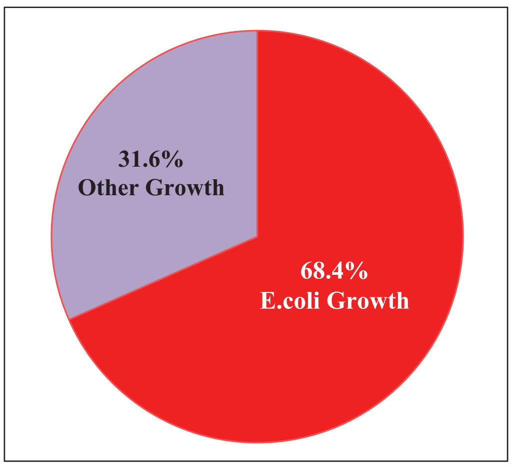

Figure 2: Growth percentage *E. coli* from all positive growth samples collected

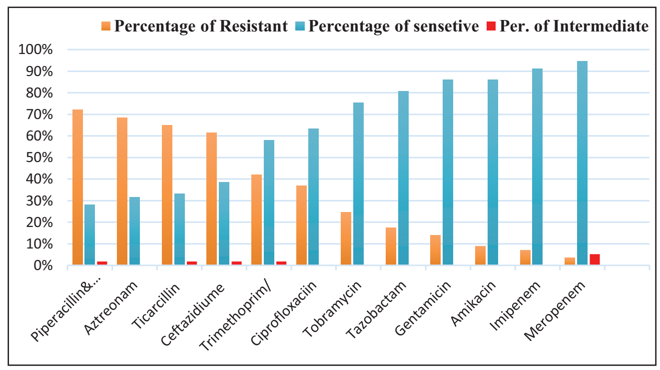

Figure 3: Results of antimicrobial susceptibility test

antibiotics, which include Ciprofloxacin, the resistance among the 21 isolates was found to be 36.80%. The isolates exhibited a resistance rate of 42% (24 isolates) to Miscellaneous antibiotics, including trimethoprim and sulfamethoxazole. Aminoglycosides, such as Tobramycin, Gentamicin, and Amikacin, displayed resistance rates of 24.60% (14 isolates), 14% (8 isolates), and 8.8% (5 isolates), respectively. The resistance rate to β-lactam combinations, including Piperacillin-tazobactam, was 17.5% (10 isolates). It is important to note that antibiotic resistance is a growing concern worldwide, and it is crucial to use antibiotics judiciously to avoid the development and spread of resistant bacteria. Four of the isolates were resistant to carbapenems, which include imipenem and meropenem, at a rate of 7%, and two of the isolates were resistant to carbapenems at a rate of 3.5%. as can be seen in Figure 3. In our work, more isolates from phylogenetic group B2 had antibiotic-resistant genes and were resistant to antibiotics than isolates from other phylogenetic groups [Table 3]. Phylogenetic group B2 isolates had a larger prevalence of antibiotic resistance and antibiotic-resistant genes than isolates from other phylogenetic groups. The phylogenetic groups of *E. coli* were found to have a significant relationship with Piperacillin (P = 0.011), Ticarcillin and Cefepime (P = 0.003), Aztreonam (P = 0.004), and Ceftazidime (P = 0.006). In this study as well.

Discussion

Several studies have shown how antibiotic resistance varies by phylogenetic group. In this study, we tried to find out for the first time in Basrah, South Iraq, how common phylogenetic group distribution is and how antibiotic resistance varies by uropathogenic *E. coli* phylogenetic group. According to several investigations, commensal *E. coli* isolates typically belong to groups A and B1, while extraintestinal pathogenic strains primarily (but not exclusively) belong to groups B2 and D. These findings were published in Yazdanpour *et al.*[1,11] The phylogenetic group D contained the most strains, totaling 17, which accounted for 29.82% of the total. Phylogroups A and

Table 3: Antibiotic resistance profile of *E. coli* among different phylogroups

Antibiotic groups	Antibiotic	Group A, N = 14	Group B1, N = 7	Group B2, N = 9	Group D, N = 17	Group F, N = 5	Clad 1, N = 2	NT, N = 3	Total 57	P value*
Penicillin	Piperacillin	8 57.1 d	5 (71.4) a	7 (77.8) a	13 (76.47) a	5 (100) a	1 (50) a	2 (66.67) a	41 (72%)	0.011
	Ticarcillin	11 78.6a	4 (57.1) b	5 (55.6) c	11 (64.7) c	4 (80) b	1 (50) a	1 (33.33) b	37 (64.9%)	0.003
β-Lactam Carbapenems	Imipenem	1 (7.1) g	(0) f	0 h	1 (5.88) h	1 (20)d	1 (50) a	0c	4 (7%)	1
	Meropenem	0 f	(0) f	0 h	1 (5.88) h	1 (20)d	0b	0c	2 (3.5%)	1
β-Lactam combinations	Piperacillin/ Tazobactam	1 (7.1) g	2 (28.6) d	3 (33.33) d	2 (11.76) g	1 (20)d	1 (50) a	0c	10 (17.5%)	0.849
Minocycline	Aztreonam	10 (71.4) b	4 (57.1) b	6 (66.66) b	12 (70.59) b	5 (100) a	1 (50) a	1 (33.33) b	39 (68.4%)	0.004
Fluroquinolones	Cefepime	10 (71.4) b	3 (42.9) c	7 (77.8) a	13 (76.47) a	5 (100) a	1 (50) a	2 (66.67) a	41 (72%)	0.003
	Ceftazidime	9 (64.3) c	3 (42.9) c	6 (66.7) b	11 (64.71) c	4 (80)b	1 (50) a	1 (33.33) b	35 (61.40%)	0.006
Ciprofloxacin	Ciprofloxacin	5 (35.7) e	1 (14.3) e	4 (44.4) e	7 (41.18) e	3 (60) c	1 (50) a	0c	21 (36.8%)	0.164
Aminoglycosides	Tobramycin	5 (35.7) e	1 (14.3) e	1 (11.11) g	5 (29.41) e	1 (20)d	1 (50) a	0c	14 (24.60%)	0.103
	Gentamicin	0 f	1 (14.3) e	2 (22.2) f	4 (23.53) f	0e	1 (50) a	0c	8 (14%)	0.392
	Amikacin	1 (7.1) g	1 (14.3) e	0 h	1 (5.88) h	1 (20)d	1 (50) a	0%	5 (8.8%)	1
Sulfonamides: Miscellaneous	Trimethoprim/ sulfamethoxazole	5 (35.7) e	2 (28.6) d	7 (77.8) a	5 (29.41) e	3 (60)c	1 (50) a	1 (33.3%)	24 (42%)	0.16

B followed. There were 14 of them, which is 24.56%. B2 has 9 (15.78%) of the total, while B1 only has 7 (12.28%). While phylogroup F accounts for 5 (8.8%) of the strains, an unknown group contributed 3 (5.26%), while clade I contributed 2 (3.5%). but neither group C nor group E was found in any of the isolated organisms. Phylogroup D contained the greatest number of individuals overall. The results of the current study were very different from those of a prior study that Iraq Wasite had reported. The earlier study showed that group B2 had the highest frequency of UPEC isolates (23.6%). According to Al-Guranie and Al-Mayahie's research from 2020, group E was not found in any of the isolates.[12] The findings obtained are strikingly comparable to those found by Bozcal *et al*.[13] in the Izmir province of Turkey. phylogenetic classification of ExPEC, D. research indicated phylogroup D was the prevalent strain.[13] Reported that the phylogenetic classification of ExPEC, D and our study was the same as one done in Mexico on UPEC strains,[14] which found that phylogroup D was more common than other genetic phylogroups.[15] After phylogroup D, phylogroup A was the most common group in our study, which is in line with what Derakhshandeh *et al*.[16] found. We found that group A was better than group B2 in this study.[16] Some studies (Munkhdelger *et al*.[17]) said that 24.56% of the isolates were in phylogenetic group A, which is linked to commensal bacteria. Even though it was thought that most of the isolates in this evolutionary group lived in the gut, the urinary system was where they were able to disseminate illness. This may be a result of critical genes that directly contribute to disease or particular hypothesized components that facilitate bacterial host invasion. Which helps the bacteria better adapt to their surroundings.[17] Additionally, Romanus and Eze[18] provided evidence suggesting that the gut may be the primary site where strains responsible for UTIs reside.[18]

Although the phylogenetic group B2 was not the most prevalent in our investigation, it has been reported as the most frequent group in various regions, including Iraq,[19] Iran.[20]

The most important thing about the new Clermont quadruplex-PCR method for *E. coli* phylogroup is that it adds four new phylogroups (C, E, F, and clade I) to the four big phylogroups (A, B1, B2, and D) that were already known. The results of this study are very close to what Al-Guranie and Al-Mayahie.[12] But different with results Iranpour *et al*. Iran showed that phylogroup E makes up 9.3% of UPEC isolates, but phylogroup F makes up 8.8%.[9] This is in agreement with what Iraqi wasite showed (15.7%) (Mohsin *et al*. but it is different from what Boroumand *et al*. said about group F making up 0% of UPEC isolates.[20]

When compared to other evolutionary groups, F and Clade had the lowest frequency in our study. Cryptic clades are usually linked to *E. coli* in the periphery. This means that the results may be due to not following good hygiene practices. Clermont *et al*.,[4] say that Most likely, unassigned strains are the result of a lot of gene mixing between two different groups or of flexible genomes caused by gene loss and gain.[4] On the other hand, none of the *E. coli* isolates studied in this study belonged to phylogenetic groups C or E. In this study, 3.5% of the *E. coli* isolates from people with UTIs could not be put into a category. Findings are hard to explain, but maybe these are not part of the job. Clermont *et al*.,[3] found that strains are either very rare phylogroups or the result of a mix of two different phylogroups.[3] Phylogenetic groups were spread out differently in this study than in other studies. This could be because of differences in geographic regions, host health status, health factors, antibiotic use patterns, genetic variation, and the part of the body where bacteria were isolated.[21]

Bacteria have become more resistant to antibiotics as a result of the discovery and development of new antibiotics. It is important to quickly and accurately identify resistant bacteria to choose the best treatments and stop the spread of resistance.[22]To report the findings of our study, we observed that the prevalence of resistance to Cephems, which includes Cefepime and Ceftazidime, was approximately 72% (41 isolates). ADditionally, we found that the resistance rate for Cefepime and Ceftazidime was approximately 61.40% (35 isolates). This is consistent with previous studies that have reported cephalosporin resistance rates between 50 and 70%.[14,23-26] The high rates of antimicrobial resistance are likely due to the widespread use of these agents. *Escherichia coli* isolated from UTI infections has shown to be resistant to the cephalosporin and penicillin groups. It is worth noting that this resistance appears to be more prevalent in developing nations in comparison to European nations.[27] In this study, 42% of bacteria are resistant to trimethoprim-sulfamethoxazole, the higher rates in Kuwait (48%), Mexico (56.1%), and New Delhi (84%) agreed with it.[28] In this study, 36.8% of isolates were resistant to fluoroquinolones. This was almost the same as what was found in Iran (34% resistance) and Kuwait (31% resistance).[24,26]

In every study, more than half of the people who had quinolone and lactam resistance In Bangladesh, only Moue *et al*. discovered 13.9% resistance to lactams, which is roughly the same as what we discovered (17.5%).[29]

To improve therapy outcomes, empirical treatment approaches against ESBL enzymes must be modified.[14,25,26] After analyzing the data, we found that the prevalence of aminoglycoside resistance varied widely across the studies, ranging from 2% to 85.24%. In our study, we observed that the resistance rates for the aminoglycoside antibiotics Tobramycin, Gentamicin, and Amikacin were 24.6%, 14%, and 8.8%, respectively. These results are consistent with the findings of Sabir *et al*. (12.7%)[30] and Pourzare *et al*. (12.5%).[14] Our findings are in line with the previous studies, including Ranjbar *et al.*,[24] Derakhshan *et al.*,[25,26] Pourzare *et al*.[14,25] Neamati *et al*. (2015),[26] and Taheri *et al.*,[31] Carbapenems like imipenem and meropenem (7% and 4.5%) had the least resistant bacteria. Percent of bacteria are resistant to carbapenems, which is the same as what our study found.[14,25,26,32]

However, it was seen that both imipenem and meropenem were very sensitive to UPEC. Meropenem has been shown to work well against gram-negative bacteria and this finding was the same as what an Iraqi study found.[33]

Table 3 shows that our study found a strong link between phylogenetic groups and drug resistance (P 0.05). Some studies (Molina-Lopez *et al*.[34] have also found a link between phylogenetic groups and drug resistance. The findings of this investigation suggest that uropathogenic *E. coli* isolates have become increasingly prevalent in recent years. Notably, this study is the first to report on the prevalence of phylogenetic groups and their correlation with antibiotic resistance patterns in strains that cause UTIs in Basrah, located in southern Iraq. The results of our study showed that group D strains were the most common and that this phylogroup also had more strains that were resistant to antibiotics. Based on these results, phylogroup D can be thought of as a genetic source of antibiotic resistance because it is better at causing UTIs in Basrah City than other phylogroups. Using a "One Health" approach, the link between phylogenetic group and antibiotic resistance in ExPEC that was reported helps us learn more about these bacteria. This makes it more likely that we can control them and lower the risk of AMR.

Conclusion

The results of this study demonstrated that group D strains were the most prevalent among the other phylogroups and that this phylogroup also had a greater prevalence of antibiotic resistance. According to these findings, phylogroup D can be thought of as a genetic reservoir for antibiotic resistance because it is more effective than other phylogroups at producing UTIs in Basrah city. Following a One Health Approach, the described association between phylogenetic group and antibiotic resistance in ExPEC increases our understanding of these bacteria and, as a result, increases our chances of controlling them and lowering the risk for AMR.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Yazdanpour Z, Tadjrobehkar O, Shahkhah M. Significant association between genes encoding virulence factors with antibiotic resistance and phylogenetic groups in community acquired uropathogenic Escherichia coli isolates. BMC Microbiol 2020;20:1-9.
2. Abdul Waheed YA, Al-Shireefy HM. Incidence of acute kidney injury in hospitalized COVID-19 patients. Med J Babylon 2022;19:589-94.
3. Clermont O, Bonacorsi S, Bingen E. Rapid and simple determination of the Escherichia coli phylogenetic group. Appl Environ Microbiol 2000;66:4555-8.
4. Clermont O, Christenson JK, Denamur E, Gordon DM. The Clermont Escherichia coli phylo-typing method revisited: Improvement of specificity and detection of new phylo-groups. Environ Microbiol Rep 2013;5:58-65.

5. Katongole P, Bulwadda Kisawuzi D, Kyobe Bbosa H, Patrick Kateete D, Florence Najjuka C. Phylogenetic groups and antimicrobial susceptibility patterns of uropathogenic *Escherichia coli* clinical isolates from patients at Mulago National Referral Hospital, Kampala, Uganda [version 1; peer review: 2 approved with reservations]. F1000Research 2019;8:1828.
6. 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 infections, a systematic review and meta-analysis. Microb Drug Resist 2020;26.
7. Fayyazi A, Halaji M, Sadeghi A, Havaei SA. High frequency of integrons and efflux pump in uropathogenic *Escherichia coli* isolated from Iranian kidney and non-kidney transplant patients. Gene Rep 2020;21:100873.
8. Ahmed SS, Shariq A, Alsalloom AA, Babikir IH, Alhomoud BN. Uropathogens and their antimicrobial resistance patterns: Relationship with urinary tract infections. Int J Health Sci (Qassim) 2019;13:48-55.
9. Daoud N, Hamdoun M, Hannachi H, Gharsallah C, Mallekh W, Bahri O. Antimicrobial susceptibility patterns of *Escherichia coli* among Tunisian outpatients with community-acquired urinary tract infection (2012-2018). Curr Urol 2020;14:200-5.
10. Bitew A, Molalign T, Chanie M. Species distribution and antibiotic susceptibility profile of bacterial uropathogens among patients complaining urinary tract infections. BMC Infect Dis 2017;17:1-8.
11. Kranz J, Schmidt S, Lebert C, Schneidewind L, Mandraka F, Kunze M, *et al*. The 2017 update of the German clinical guideline on epidemiology, diagnostics, therapy, prevention, and management of uncomplicated urinary tract infections in adult patients part II: Therapy and prevention. Urol Int 2018;100:271-8. doi:10.1159/000487645.
12. Al-Guranie DR, Al-Mayahie SM. Prevalence of *E. coli* ST131 among Uropathogenic *E. coli* Isolates from Iraqi Patients in Wasit Province, Iraq. Int J Microbiol 2020;2020:8840561. doi:10.1155/2020/8840561.
13. Bozcal E, Eldem V, Aydemir S, Skurnik M. The relationship between phylogenetic classification, virulence and antibiotic resistance of extraintestinal pathogenic *Escherichia coli* in İzmir province, Turkey. PeerJ 2018;6:e5470. doi:10.7717/peerj.5470.
14. Pourzare M, Derakhshan S, Roshani D. Distribution of uropathogenic virulence genes in *Escherichia coli* isolated from children with urinary tract infection in Sanandaj, Iran. Arch Pediatr Infect Dis 2017;In press: p. e41995.
15. Ramírez-Castillo FY, Moreno-Flores AC, Avelar-González FJ, Márquez-Díaz F, Harel J, Guerrero-Barrera AL. An evaluation of multidrug-resistant Escherichia coli isolates in urinary tract infections from Aguascalientes, Mexico: Cross-sectional study. Ann Clin Microbiol Antimicrob 2018;17:34. doi:10.1186/s12941-018-0286-5.
16. Derakhshandeh A, Firouzi R, Motamedifar M, Arabshahi S, Novinrooz A, Boroojeni AM, *et al*. Virulence Characteristics and Antibiotic Resistance Patterns among Various Phylogenetic Groups of Uropathogenic Escherichia coli Isolates. Jpn J Infect Dis 2015;68:428-31. doi:10.7883/yoken.JJID.2014.327.
17. Munkhdelger Y, Gunregjav N, Dorjpurev A, Juniichiro N, Sarantuya J. Detection of virulence genes, phylogenetic group and antibiotic resistance of uropathogenic Escherichia coli in Mongolia. J Infect Dev Countr 2017;11:51-7. doi:10.3855/jidc.7903.
18. Romanus II, Eze AT. Antibiotics susceptibility patterns and clonal relatedness of uropathogenic *Escherichia coli* in *Abakaliki, Ebony*i state. Canad J Pure Appl Sci 2011;5:1475-9.
19. Mohammed EJ, Hasan KC, Allami M. Phylogenetic groups, serogroups and virulence factors of uropathogenic *Escherichia coli* isolated from patients with urinary tract infection in Baghdad, Iraq. Iran J Microbiol 2022;14:445-57.
20. Boroumand M, Naghmachi M, Ghatee MA. Detection of phylogenetic groups and drug resistance genes of *Escherichia coli* causing urinary tract infection in southwest Iran. Jundishapur J Microbiol 2021;14:e112547.
21. Staji H, Rassouli M, Jourablou S. Comparative virulotyping and phylogenomics of *Escherichia coli* isolates from urine samples of men and women suffering urinary tract infections. Iran J Basic Med Sci 2019;22:211-4. doi:10.22038/ijbms.2018.28360.6880.
22. Farajzadah Sheikh A, Goodarzi H, Yadyad MJ, Aslani S, Amin M, Jomehzadeh N, *et al*. Virulence-associated genes and drug susceptibility patterns of uropathogenic *Escherichia coli* isolated from patients with urinary tract infection. Infect Drug Resist 2019;12:2039-47. doi:10.2147/IDR.S199764.
23. Shah C, Baral R, Bartaula B, Shrestha LB. Virulence factors of uropathogenic Escherichia coli (UPEC) and correlation with antimicrobial resistance. BMC Microbiol 2019;19:204. doi:10.1186/s12866-019-1587-3.
24. Raeispour M, Ranjbar R. Antibiotic resistance, virulence factors and genotyping of *Uropathogenic Escherichia coli* strains. Antimicrob Resist Infect Cont 2018;7:1-9.
25. Derakhshan S, Pourzare M, Roshani D. Distribution of virulence factors according to antibiotic susceptibility among *Escherichia coli* isolated from urinary tract infection. Ind J Nephrol 2018;28:191-7.
26. Neamati F, Firoozeh F, Saffari M, Zibaei M. Virulence genes and antimicrobial resistance pattern in uropathogenic Escherichia coli isolated from hospitalized patients in Kashan, Iran. Jundishapur J Microbiol 2015;8:e17514. doi:10.5812/jjm.17514.
27. Kot B. Antibiotic resistance among uropathogenic. Pol J Microbiol 2019;68:403-15.
28. Assafi MS, Ali FF, Polis RF, Sabaly NJ, Qarani SM, *et al*. An Epidemiological and multidrug resistance study for E coli isolated from urinary tract infection (Three Years of Study). Baghdad Sci J 2022;19:0007-0007.
29. Moue A, Aktaruzzaman SA, Ferdous N, Karim MR, Khalil M, Das AK. Prevalence of urinary tract infection in both outpatient department and in-patient department at a medical college setting of Bangladesh. Int J Biosci 2015;7:146-52.
30. Sabir S, Ahmad Anjum A, Ijaz T, Asad Ali M, Ur Rehman Khan M, Nawaz M. Isolation and antibiotic susceptibility of E. coli from urinary tract infections in a tertiary care hospital. Pak J Med Sci 2014;30:389-92.
31. Taheri M, Saleh M, Nemati AH, Ariana M, Shojaei E, Mardani M, *et al*. Antibiotic resistance pattern and phylogenetic groups of the uropathogenic *Escherichia coli* isolates recovered from the urinary catheters of the hospitalized patients. Iran J Microbiol 2016;4:76-82.
32. Yun KW, Kim HY, Park HK, Kim W, Lim IS. Virulence factors of uropathogenic Escherichia coli of urinary tract infections and asymptomatic bacteriuria in children. J Microbiol Immunol Infect 2014;47:455-61. doi:10.1016/j.jmii.2013.07.010.
33. Maka G, Shah S, Bano S, Tunio SA. Antibiotic susceptibility profiling of gram-negative bacteria causing upper respiratory tract infections in Hyderabad, Sindh. JLBSR [Internet] 2020;1:12-5. Available from: https://www.jlbsr.org/index.php/jlbsr/article/view/2.
34. Molina-López J, Aparicio-Ozores G, Ribas-Aparicio RM, Gavilanes-Parra S, Chávez-Berrocal ME, Hernández-Castro R, *et al*. Drug resistance, serotypes, and phylogenetic groups among uropathogenic *Escherichia coli* including O25-ST131 in Mexico City. J Infect Dev Countr 2011;5:840-9. doi:10.3855/jidc.1703.