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Detection of Phylogenetic Groups and Drug Resistance Genes of *Escherichia coli* Causing Urinary Tract Infection in Southwest Iran

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Abstract

Background: Many bacteria can cause urinary tract infections (UTIs), among which *Escherichia coli* is the most common causative agent. *Escherichia coli* strains are divided into eight phylogenetic groups based on the new Quadroplex-PCR method, which are different in terms of patterns of resistance to antibiotics, virulence, and environmental characteristics.
Objectives: This study aimed to determine the phylogenetic groups and the prevalence of drug resistance genes in *E. coli* strains causing UTIs.
Methods: In this descriptive cross-sectional study, 129 *E. coli* isolates obtained from the culture of patients with UTIs were evaluated for phylogenetic groups using the new method of Clermont et al. The identification of phylogenetic groups and antibiotic resistance genes was performed using the multiplex polymerase chain reaction (PCR) method.
Results: In this study, concerning the distribution of phylogenetic groups among *E. coli* isolates, the phylogenetic group B2 (36.4%) was the most common phylogenetic group, followed by phylogroups C (13.2%), clade I (10.1%), D (9.3%), and A (3.1%) while groups B1 and F were not observed in any of the isolates, and 20.2% had an unknown state. Also, out of 129 *E. coli* isolates, the total frequency of *tetA*, *tetB*, *sul1*, *sul2*, *CITM*, *DfrA*, and *qnr* resistance genes was 59.7%, 66.7, 69, 62, 30.2, 23.3, and 20.2%, respectively. In this study, there was a significant relationship between antibiotics (P = 0.026), cefotaxime (P = 0.003), and nalidixic acid (P = 0.044) and *E. coli* phylogenetic groups. No significant relationship was observed between *E. coli* phylogenetic groups and antibiotic resistance genes.
Conclusions: The results of this study showed that strains belonging to group B2 had the highest prevalence among other phylogroups, and also, the frequency of antibiotic resistance genes and drug-resistant isolates had a higher prevalence in this phylogroup. These results show that phylogroup B2 has a more effective role in causing urinary tract infections compared to other phylogroups, and this phylogroup can be considered a genetic reservoir of antibiotic resistance.

Keywords: Uropathogenic *Escherichia coli*, Drug Resistance, Bacteria, Phylogenetic Groups

1. Background

Many bacteria can cause urinary tract infections. Among them, *Escherichia coli* is the most common agent that can cause infections at different ages. Studies in different communities have shown that Gram-negative bacilli, especially *E. coli*, cause more than 80 - 90% of infections (1, 2). A group of researchers has emphasized that the type of *E. coli* phylogenetic group plays an important role in their pathogenicity (3-5). In 2000, Clermont et al., using triplex PCR and amplification of three genetic markers, TspE4.C2, chuA, and yjaA, classified extracellular *E. coli* strains into four groups: B2, B1, A, and D. These different isolates were separated from different sources (6). In 2013, Clermont et al. added a new *arpA* gene to the previous three genes to design a quadruple polymerase reaction that had greater resolution than the previous method. In this method, *E. coli* isolates were divided into eight phylogenetic groups B2, B1, A, D, F, E, C, and clade I (7). These phylogroups are distinct in terms of characteristics such as patterns of antibiotic resistance, virulence genes, use of sugars, and environmental characteristics (8, 9). Outpatient pathogenic strains are mainly in group B2 and to a lesser extent in group D, while commensal strains belong to groups 1B and A (4, 10, 11).

Today, one of the most important obstacles to the control and treatment of infectious diseases is the resistance of pathogenic bacteria to various antibiotics. Bacteria use different strategies to survive the harmful effects of antibiotics. Some microorganisms are inherently resistant, and others are resistant to other organisms through the mechanisms of resistance gene release. These antibiotic resistance genes are transmitted through plasmids, trans-

posons, and integrons (12-14).

Genes resistant to tetracycline (*tetA*, *tetB*), fluoroquinolones (*qnr*), sulfonamides (*sul*), ampicillin (*CITM*), and cotrimoxazole (*slu*, *dfrA*) have been observed in recent decades in *E. coli* isolates that perform different functions (15, 16). Tetracycline resistance can be created by acquiring the tet gene. Besides, *tetA* and *tetB* genes reduce the accumulation of antibiotics within the bacterium by encoding efflux pumps, and the isolation and detection of this dozen have been reported to be higher than other tetracycline resistance genes (17, 18). Antunes et al. showed that the most important genes that make cotrimoxazole resistance (sulfonamide group) are the *sul1*, *sul2*, and *sul3* genes. The *sul* genes are involved in the first stage of folic acid synthesis, and *dfrA* genes are involved in the second stage of folic acid synthesis and induce resistance to sulfonamides (19).

The quinolone resistance is due to a mutation in the DNA gyrase subunit, and the *qnr* resistance genes are plasmid-dependent quinolone resistance factors that inhibit the inhibitory effect of these antibiotics on DNA gyrase and topoisomerase IV cause rapid spread of resistance in *Enterobacteriaceae* bacteria due to their location on different integrons (20, 21). In recent years, differences in the prevalence of resistance to antibiotics and antibiotic resistance genes in phylogenetic groups have been of great importance. Various reports have shown that the prevalence of distribution of antibiotic susceptibility and resistance profiles, as well as drug resistance genes, varies in the phylogenetic groups of uropathogenic *E. coli* (4, 22-25).

2. Objectives

This study aimed to determine the phylogenetic groups and genes of antibiotic resistance, including *tetA*, *tetB*, *sul1*, *sul2*, *dfrA1*, *CITM*, and *qnr* genes in *E. coli* species isolated from patients with urinary tract infections using the multiplex PCR method in Yasuj (Southwest Iran).

3. Methods

This study was performed on 129 *E. coli* isolates collected from patients with UTIs who were referred to medical diagnostic laboratories and Imam Sajjad and Shahid Beheshti hospitals in Yasuj between July and October 2017. The population of the study included outpatients with UTIs referring to medical labs for urine culture; the growth of *E. coli* was considered a positive result. Exclusion criteria were having an indwelling urinary catheter, being pregnant, having genitourinary abnormalities, and antibiotic therapy within the last two weeks. After collection, urine samples were cultured on MacConkey and eosin methylene blue agar (EMB) and incubated at 37°C for 24 h. Then, bacteria were identified using routine biochemical tests such as methyl red (MR), Voges-Proskauer (VP), Triple Sugar Iron (TSI) agar, indole production, and Simmons' citrate agar (Merck Germany). Isolated *E. coli* strains were stored in Trypticase soy broth (TSB), with sterile glycerol at -20°C (1).

3.1. Antimicrobial Susceptibility Test

The susceptibility of *E. coli* isolates to the antibiotics cefotaxime (30 μg), ampicillin (10 μg), cotrimoxazole (25 μg), ceftriaxone (30 μg), nalidixic acid (30 μg), aztreonam (30 μg), ciprofloxacin (30 μg), tetracycline (30 μg), and ceftizoxime (30 μg) (BD-BBL Company, American) was assessed using the Kirby-Bauer disk diffusion method as per the CLSI and clinical standards. To control the quality of the disks, *E. coli* ATCC 25922 was used (1).

3.2. DNA Extraction and Phylogenetic Grouping and Antibiotic Resistance Genes

After the final diagnosis, DNA extraction from bacteria was performed by the boiling method. Briefly, several loops of bacteria (24 h) were boiled in a microtube containing sterile distilled water for 10 min at 100°C and then centrifuged. The supernatant was kept as template DNA for PCR (1). The Quadruplex-PCR method was performed using primers described by Clermont et al., to identify uropathogenic *E. coli* phylogenetic groups (7). Table 1 shows the primers used to detect drug resistance genes *tetA*, *tetB*, *sul1*, *sul2*, *qnr*, *CITM*, and *dfrA* in uropathogenic strains of *E. coli*. In this test, antibiotic-resistant isolates were amplified, and band formation of *tetA*, *tetB*, *slu1*, *slu2*, *CITM*, *dfrA*, and *qnr* genes against molecular markers confirmed the presence of these genes in the resistant isolates as positive controls from the vial. The PCR without DNA samples in which the same volume of distilled water was added, instead of DNA, was used as a negative control.

The PCR temperature program for the detection of phylogenetic groups and antibiotic resistance genes was as follows: A cycle of 95°C for 5 min, 30 cycles including 94°C denaturation for one minute, the binding temperature of the primers for phylogenetic groups of 59°C for 20 s (Figure 1), the binding temperature of the primers to the template DNA for the studied resistance genes (Table 1), amplification at 72°C for 90 s, and final extension at 72°C for 5 min (Figures 2 and 3). Electrophoresis of PCR products was performed on a 2% agarose gel with DNA safe stain dye solution in the presence of a 100 bp marker (Pishgam, Iran) and 90-volt constant voltage for 60 min. The gel was examined with a UV Transilluminator (Major Science, Taiwan).

Table 1. Sequence of Primers of Antibiotic Resistance Genes Used for Polymerase Chain Reaction

Antimicrobial agent/Resistance gene	Sequence	Annealing Temperature (°C)	Size (bp)	References
Tetracycline				
tetA F	GGTTCACTCGAACGACGTCA	58	577	
tetA R	CTGTCCGACAAGTTGCATGA			
tet B F	CCTCAGCTTCTCAACGCGTG	58	634	
tet B R	GCACCTTGCTGATGACTCTT			
Trimethoprim				
dfra F	GGAGTGCCAAAGGTGAACAGC	63	367	(15)
dfra R	GAGGCGAAGTCTTGGGTAAAAAC			
Beta-lactams				
CITM F	TGGCCAGAACTGACAGGCAAA	63	462	
CITM R	TTTCTCCTGAACGTGGCTGGC			
Quinolones				
qnr F	GGGTATGGATATTATTGATAAAG	59	670	
qnr R	CTAATCCGGCAGCACTATTTA			
Sulfonamide				
sul1 F	CGGCGTGGGCTACCTGAACG	63	433	
sul1 R	GCCGATCGCGTGAAGTTCCG			(26)
sul2 F	GCGCTCAAGGCAGATGGCATT	63	293	
sul2 R	GGGTTTGATACCGGCACCCGT			

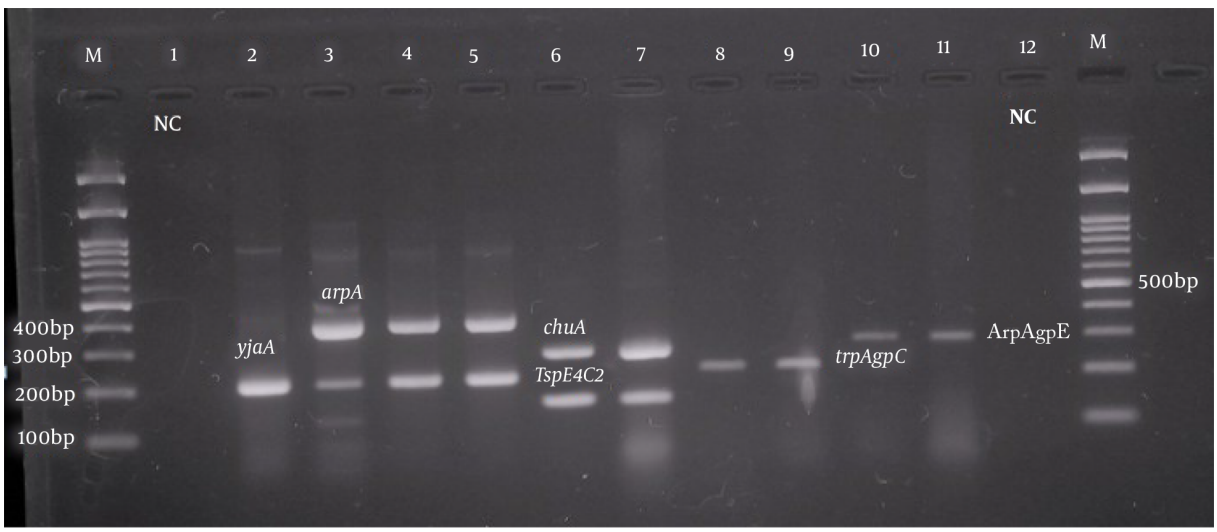

Figure 1. Phylogenetic analysis of pathogenic *Escherichia coli* isolates. Left to right: 100 bp marker, well number one as a negative control, well numbers 2, 3, 4, and 5 with *yjaA* (211 bp) and *arpA* (400 bp), well numbers 6 and 7 with *chuA* (288 bp) and TspE4.C2 (152 bp), well numbers 8 and 9 with *trpAgp* C (219 bp), and well numbers 10 and 11 with *ArpAgpE* (301 bp) genes.

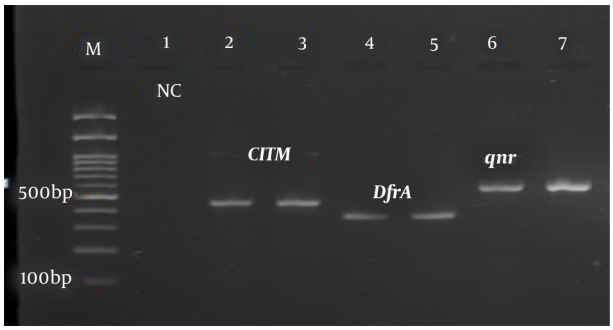

Figure 2. Multiplex PCR test results to determine *CITM*, *dfrA*, and *qnr* genes. Left to right: 100 bp marker, well number one as a negative control, wells two and three with *CITM* gene (462 bp), well number four with *dfrA* gene (367 bp), and well numbers six and seven with *qnr* gene (670 bp).

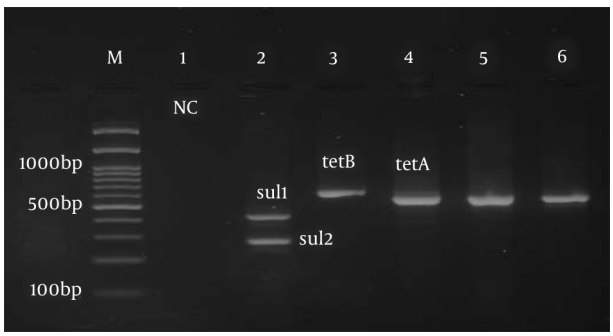

Figure 3. Multiplex PCR test results to determine *tetA*, *tetB*, *sul1*, and *sul2* genes. Left to right: 100 bp marker, well number one as a negative control, well number two with *sul1* (433 bp) and *sul2* (293 bp) genes, and well numbers three and four with *tetB* (634bp) and *tetA* (577bp) genes.

3.3. Statistical Analysis

Statistical analysis was performed using the chi-square test and Fisher's exact test with SPSS software (version 18.0). The significance level was set at $P < 0.05$.

4. Results

The prevalence of urinary tract infections was higher in females of all age groups. In this study, 99 (76.7%) of the studied samples were related to women and 30 (23.3%) to men. The phylogenetic groups of the collected *E. coli* isolates were determined using the method mentioned by Clermont et al. (7). The PCR results showed that 36.4% of the strains belonged to group B2, 20.2% to the unknown group, 13.2% to group C, 10.1% to group Clade I, 9.3% to group D, 7.8% to group E, 3.1% to group A, and phylogenetic groups B1 and F were not observed among the strains studied in this study (0%). Also, the distributions of *tetA*, *tetB*, *sul1*, *sul2*, *dfrA1*, *CITM*, and *qnr* antibiotic resistance genes were 59.7, 66.7, 69, 62, 23.3, and 30.2%, respectively. The prevalence of antibiotic resistance genes among phylogenetic groups is shown in Table 2. In the statistical analysis based on Fisher's exact test, no significant relationship was observed between gender and phylogenetic groups (Table 3). In our study, the prevalence of antibiotic resistance and antibiotic-resistant genes was higher in the isolates of phylogenetic group B2 than in other phylogenetic groups (Table 4).

We found a significant relationship between the *E. coli* phylogenetic groups of ceftizoxime ($P = 0.026$), cefotaxime ($P = 0.003$), and nalidixic acid ($P = 0.048$) antibiotics. Also, in this study, no significant relationship was observed between *E. coli* phylogenetic groups and antibiotic resistance genes. In the present study, we found a significant relationship between the presence of genes encoding antibiotic resistance, including *qnr* gene and resistance to nalidixic acid ($P = 0.016$) and ciprofloxacin ($P = 0.034$), the gene encoding sul1 resistance, and resistance to cefotaxime ($P = 0.003$), ceftriaxone ($P = 0.011$), cotrimoxazole ($P = 0.003$), and ceftizoxime ($P = 0.011$), the presence of *tetA* resistance gene and resistance to tetracycline ($P = 0.006$), ampicillin ($P = 0.001$), aztreonam ($P = 0.005$), and ciprofloxacin ($P = 0.001$) antibiotics, the presence of *tetB* encoding gene and tetracycline resistance ($P = 0.037$), as well as the presence of *dfrA1* coding gene and ceftriaxone resistance ($P = 0.041$) (Table 5).

5. Discussion

Various studies have shown that the patterns of antibiotic resistance and susceptibility, the number of virulence genes, as well as genes encoding antibiotic resistance in *E. coli* in different geographical areas are associated with specific genetic groups (4, 27, 28). Therefore, in this study, we tried to investigate the prevalence of phylogenetic groups, antibiotic resistance genes, and the distribution of these resistance genes and antibiotic resistance patterns in uropathogenic *E. coli* phylogenetic groups based on the new method of Clermont et al. for the first time in Yasuj (Southwest Iran). The results of several studies indicate that extraintestinal pathogenic strains mainly belong to groups B2 and D (to a lesser extent) and, also, the commensal isolates of *E. coli* belong to groups A and B1 (2, 10, 22). In the present study, the most common phylogenetic groups belonged to group B2 with a prevalence of 36.4%. It was followed by unknown phylogenetic (20.2%), C (13.2%), Clade I (10.1%), D (9.3%), E (7.8%), and phylogenetic group A with a prevalence of 3.1%. As expected, in the present study, the highest frequency belonged to the B2 phylogroups.

The results of our study are consistent with other studies in Iran (2, 4, 29) and other parts of the world, includ-

Table 2. Distribution of Antibiotic Resistance Genes in Relation to Phylogenetic Groups in *Escherichia coli*[a]

Antibiotic resistance genes	B2 (n = 47)	D (n = 12)	C (n = 17)	E (n = 10)	U (n = 26)	Clade I (n = 13)	A (n = 4)
tetA	24 (51.7)	7 (58.3)	11 (64.7)	7 (70)	16 (61.5)	10 (76.9)	2 (50)
tetB	32 (68)	10 (83.3)	12 (70.5)	7 (70)	15 (57.6)	10 (76.9)	0 (0)
sul1	35 (74.4)	10 (83.3)	11 (64.7)	3 (30)	18 (69.23)	8 (61.5)	4 (100)
sul2	25 (53)	11 (91.6)	12 (70.5)	5 (50)	14 (53.8)	10 (76.9)	3 (75)
dfrA	12 (25.5)	2 (16.6)	5 (29.4)	2 (20)	2 (7.69)	6 (46)	1 (25)
CITM	16 (34)	3 (25)	5 (29.4)	3 (30)	9 (34.6)	3 (23)	0 (0)
qnr	11 (23.4)	2 (16.6)	3 (17.6)	1 (10)	7 (26.9)	2 (15.3)	0 (0)
qnr,sul1	7 (14.8)	1 (8.3)	1 (5.8)	0 (0)	4 (15.38)	2 (15.3)	0 (0)
sul1, sul2	17 (36.17)	9 (75)	9 (52.9)	1 (10)	8 (30.7)	5 (38.4)	3 (75)
tetA, tetB	18 (38.2)	5 (41.6)	11 (64.7)	5 (50)	12 (46)	9 (69.2)	1 (25)
sul1, sul2, tetA, tetB	7 (14.8)	4 (33.3)	6 (35.2)	0	3 (11.5)	4 (30.7)	0 (0)
qnr,sul1, dfrA	1 (2.1)	0 (0)	1 (5.8)	0 (0)	0 (0)	0 (0)	0 (0)

[a] Values are expressed as No. (%).

Table 3. Distribution of Phylogenetic Groups Based on Gender [a]

Sex	B2 (n = 47)	C (n = 17)	D (n = 12)	A (n = 4)	E (n = 10)	Clade I (n = 13)	Unknown (n = 26)	P-Value
Male	11 (23.4)	5 (29.4)	2 (16.7)	0 (0)	3 (30.0)	2 (15.4)	7 (26.9)	0.83
Female	36 (76.6)	12 (70.6)	10 (83.3)	4 (3.1)	7 (70.0)	11 (84.6)	19 (73.1)	

[a] Values are expressed as No. (%) unless otherwise indicated.

Table 4. Frequency of Antibiotic Resistance Among Phylogenetic Groups of Uropathogenic *Escherichia coli*[a]

Phylogenetic groupe	Ampicillin	Tetracycline	Nalidixic Acid	Co-Trimoxazole	Ciprofloxacin	Cefotaxime	Ceftriaxone	Aztreonam	Ceftezoxim
B2 (n = 47)	36 (76.5)	28 (59.5)	32 (68)	33 (70)	25 (53)	22 (46.8)	22 (46.8)	28 (59.5)	30 (63.8)
C (n = 17)	12 (70.5)	10 (58.8)	9 (52.9)	10 (58.8)	7 (41)	7 (41)	8 (47)	9 (52.9)	8 (47)
D (n = 12)	11 (91.6)	10 (83.3)	4 (33.3)	8 (66.6)	8 (66.6)	7 (58.3)	7 (58.3)	9 (75)	4 (33.3)
E (n = 10)	8 (80)	4 (40)	5 (50)	3 (30)	3 (30)	4 (40)	4 (40)	5 (50)	2 (20)
Clade I (n = 13)	11 (84.6)	10 (76.9)	5 (38.4)	9 (69.2)	6 (46)	7 (53.8)	8 (61.5)	9 (69.2)	10 (76.9)
Unknowen (n = 26)	24 (92.3)	20 (76.9)	10 (38.4)	17 (65)	14 (53.8)	15 (57.6)	15 (57.6)	22 (84.6)	20 (76.9)
A (n = 4)	2 (50)	3 (75)	2 (50)	2 (50)	2 (50)	1 (25)	1 (25)	3 (75)	2 (50)
Total	104 (80.6)	85 (65.8)	67 (51.95)	82 (63.5)	65 (50.3)	63 (48.8)	65 (50.3)	85 (65.8)	76 (58.9)
P-value	0.340	0.406	0.048 [b]	0.664	0.491	0.003 [b]	0.615	0.120	0.026 [b]

[a] Values are expressed as No. (%) unless otherwise indicated.
[b] P-value < 0.05 is significant.

ing Pakistan (30), Mexico (31), and South Korea (11). However, in a study conducted in Kerman (Iran), phylogroup A and in another study in Mexico on UPEC strains, phylogroups D had a higher frequency than other genetic phylogroups. This may be due to reasons such as different distributions of *E. coli* strains in different geographical areas (32, 33). In our study, after phylogroup B2, the most common phylogroup belonged to the unknown group, which is consistent with the studies by Iranpour et al. and Najafi et al. in Iran, with the report of a 27% unknown phylogroup (4, 34). However, in a study conducted by Ghosh et al. in India, 77% of the isolates remained unclassified, which contradicted the results of the present study (35). Clermont et al. in 2013 reported that a new quadruplex PCR method does not classify only one percent of *E. coli* strains into the eight phylogenetic groups. However, about 20.2% of the total *E. coli* isolates in our study remained unclassified.

Although the explanation for these results is indescribable, it can be said that the strains in the unknown group are the results of recombination of different phylogroups or are very rare phylogroups (7). On the other hand, in this study, phylogenetic groups B1 and F were not found in

Table 5. Distribution of Antimicrobial Resistance Genes in Antibiotic Resistance Patterns of *Escherichia coli* Strains Causing Urinary Tract Infection [a]

Antibiotic Resistance Genes	Ampicillin	Tetracycline	Nalidixic Acid	Co-Trimoxazole	Ciprofloxacin	Cefotaxim	Cefteriaxon	Azteronam	Ceftezoxim
tetA	69 (89.6)	55 (71.4)	37 (48.1)	50 (64.9)	34 (44.2)	35 (45.5)	33 (42.5)	52 (67.5)	37 (48.1)
P-value	0.001 [b]	0.006 [b]	0.902	0.290	0.013 [b]	0.402	0.597	0.005 [b]	0.566
tetB	73 (84.9)	57 (66.3)	47 (54.7)	62 (71.2)	32 (37.2)	43 (50)	43 (50)	51 (53.9)	40 (46.5)
P-value	0.072	0.037 [b]	0.170	0.001 [b]	0.964	0.159	0.089	0.426	0.820
sul1	73 (82)	52 (58.4)	48 (53.9)	61 (68.5)	38 (42.7)	46 (51.7)	46 (51.7)	52 (58.4)	47 (52.8)
P-value	0.201	0.561	0.203	0.003 [b]	0.069	0.003 [b]	0.011 [b]	0.625	0.011 [b]
sul2	62 (77.5)	54 (67.5)	39 (48.8)	56 (70)	27 (33.8)	33 (41.3)	31 (38.8)	44 (55)	37 (46.3)
P-value	0.111	0.106	0.160	0.018 [b]	0.309	0.440	0.394	0.852	0.820
qnr	22 (84.6)	14 (53.8)	18 (69.2)	17 (65.4)	14 (53.8)	15 (57.7)	16 (61.5)	18 (69.2)	16 (61.5)
P-value	0.638	0.696	0.016 [b]	0.557	0.034 [b]	0.299	0.111	0.247	0.100
CITM	31 (79.5)	25 (64.1)	14 (35.9)	21 (53.8)	13 (33.3)	13 (33.3)	12 (30.8)	18 (46.2)	15 (38.5)
P-value	0.146	0.201	0.084	0.840	0.259	0.146	0.157	0.300	0.288
dfrA	24 (80)	18 (60)	19 (63.3)	18 (60)	14 (46.7)	11 (36.7)	7 (23.3)	18 (60)	17 (56.7)
P-value	0.638	0.696	0.016 [b]	0.557	0.034 [b]	0.299	0.111	0.247	0.100

[a] Values are expressed as No. (%) unless otherwise indicated.
[b] P-value < 0.05 is significant.

any of the *E. coli* isolates studied, which is consistent with the results obtained by Ghosh et al. (35). These differences in the distribution of phylogenetic groups in the present study compared to other studies could be due to differences in geographical areas, host health status, nutritional factors, patterns of antibiotic use, genetic factors, as well as differences in the anatomical area of bacterial isolation (5, 36).

The discovery of antibiotics, the production and development of new antibiotics, and the widespread use of these antibiotics for the treatment of bacterial infectious diseases have led bacteria to become more resistant to various antibiotics. Due to the increasing prevalence of resistance to antibiotics, the rapid and timely detection of resistant strains seems necessary to select appropriate treatment options and prevent the spread of resistance (24). Mechanisms of bacterial resistance to antibiotics are different. Usually, the presence of genes encoding antibiotic resistance is the main cause of antibiotic resistance in bacterial strains, and the most common resistances are controlled by transmissible plasmids (14, 37).

Tetracycline-resistant strains are highly prevalent among antibiotic-resistant *E. coli*. Tetracycline is a bacteriostatic antibiotic that binds to ribosomes and prevents protein synthesis from lengthening. The presence of resistant genes in bacteria is associated with the acquisition of the *tet* gene (18, 38). Another drug that is widely used for treating urinary tract infections is trimethoprim-sulfamethoxazole. Its resistance mechanism in *E. coli* is due to uropathogenicity because of the structural similarity of sulfonamides to para aminobenzoic acids (PABAs), which leads to the production of folic acid. The *lsu* genes cause resistance to sulfonamides (39).

Quinolones and fluoroquinolones are the drugs of choice for treating urinary tract infections caused by *E. coli* due to their antibiotic resistance. The *qnr* gene causes resistance to fluoroquinolones (20, 21). Genetic markers of bacterial antibiotic resistance have often been reported in various studies. The prevalence and distribution of these genetic profiles vary depending on the country, source, and year of bacterial isolation, and antibiotic prescribing policy (15, 33, 40). In this study, we investigated the prevalence of some genes causing antibiotic resistance in 129 *E. coli* isolates by PCR. The prevalence rates of the resistance genes *tetA*, *tetB*, *sul1*, *sui2*, *qnr*, *CITM*, and *dfrA* were 59.7, 66.7, 69, 62, 20.2, 30.2, and 23.3%, respectively. The frequency of antibiotic resistance genes in the present study was close to the results of studies conducted in Iran (14, 15, 41) and other parts of the world, including Algeria (42) and Mexico (43). The high prevalence of antibiotic resistance genes may be due to the indiscriminate use of antibiotics as well as the horizontal transmission of strains containing these antibiotic resistance genes in patients with urinary tract infections.

In this study, the distribution of multidrug resistance in different *E. coli* phylogenetic groups showed that phylogroup B2 isolates were more resistant than the isolates of other phylogenetic groups. Past studies have shown that the prevalence of antibiotic resistance among *E. coli* strains is related to the B2 phylogenetic group less than to other phylogenetic groups. Although difficult to explain, different social and environmental conditions may play a role (32, 44, 45). However, our findings are consistent with some studies that have shown the most resistant isolates were in the B2 phylogroup (4, 10, 22, 25). Our study showed a significant relationship between phylogenetic

groups and antibiotic resistance ($P < 0.05$) (Table 4). Some studies have also reported an association between phylogenetic groups and antibiotic resistance (28, 46). This indicates that the balance between antibiotic resistance and virulence factors is disturbed, and one of the two variables is increased or decreased in favor of the other, and it can be said that the strains that carry pathogenic genes are compatible with drug resistance (11).

5.1. Conclusion

Based on this study, it can be concluded that in recent years, isolates of uropathogenic *E. coli* have emerged that, in addition to having multiple virulence genes, have become resistant to different types of antibiotics. This study was the first report on the prevalence of phylogenetic groups, and their relationships with antibiotic resistance patterns and antibiotic resistance genes among *E. coli* strains causing UTIs in Yasuj (Southwestern Iran). One of the limitations of our study is the lack of study of the prevalence of virulence genes of *E. coli* in general uropathogens and their distribution and relationship with these phylogenetic groups. Finally, the results of our study showed that strains belonging to group B2 were most common among other phylogroups and also antibiotic resistance genes and drug-resistant isolates in this phylogroup (phylogroup B2) had a higher prevalence among patients with urinary tract infections in Yasuj (Southwestern Iran). Also, in this study, among the antibiotic resistance genes, the *tetB*, *tetA*, *slu1*, and *slu2* genes had a higher prevalence. Understanding the prevalence of antimicrobial resistance genes and phylogenetic groups of uropathogen *E. coli* causing urinary tract infections can help physicians treat urinary tract infections by selecting appropriate antibiotics in this geographical area to prevent the emergence of antibiotic-resistant strains.

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Footnotes

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