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

Mostafa Boroumand 1, Mohsen Naghmachi 2,* and Mohammad Amin Ghatee 2

1Research Committee, Yasuj University of Medical Sciences, Yasuj, Iran
2Cellular and Molecular Research Center, Yasuj University of Medical Sciences, Yasuj, Iran

*Corresponding author: Cellular and Molecular Research Center, Yasuj University of Medical Sciences, Yasuj, Iran. Email: naghmachi2013@gmail.com

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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*, *sulf*, *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 = 0.03), 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 B1 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-
3. Methods

The multiplex PCR method in Yasuj (Southwest Iran). Isolated from patients with urinary tract infections using EMB agar (EMB) and incubated at 37°C for 24 h. Then, samples were cultured on MacConkey and eosin methylnitrite within the last two weeks. After collection, urine samples were sent 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 isolates from patients with UTIs who were referred to medical labs for urine culture was isolated from patients with UTIs referring to medical labs for urine culture. The growth of isolates from patients with UTIs who were referred to medical labs for urine culture...
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                             | GGTTCACTCGAACACGGCAACGCA | 58 | 577 | (15)        |
| tetA R                             | CTTCTTCGAACAGATCAGGTAAGA | 61 | 614 |             |
| tet B F                            | CCTCAGCTTTCGAGCCGCTG   | 58 | 614 |             |
| tet B R                            | GCCCCTGTTGATGATCTCTT   | 61 | 614 |             |
| **Trimethoprim**                   |          |                           |           |             |
| dfrA F                             | GAGTTGCCAAGGCTGACACGC | 63 | 367 |             |
| dfrA R                             | GAGGGCGAAGTCTTGGGTAAAAC | 63 | 367 |             |
| **Beta-lactams**                   |          |                           |           |             |
| CITM F                             | TGCCGCAACTGACAGCAAAA | 63 | 462 |             |
| CITM R                             | TTCTTCTGACGTCGCCGTCGC | 63 | 462 |             |
| **Quinolones**                     |          |                           |           |             |
| qnr F                              | GGTATGGAATTATGGAATAAAG | 59 | 670 |             |
| qnr R                              | CTAACCGGCAGCATTTAAGA   | 59 | 670 |             |
| **Sulfonamide**                    |          |                           |           |             |
| sul1 F                             | CGCGCTGGGCTACTGCAAGCG | 63 | 433 | (26)        |
| sul1 R                             | GCGGATGGGCGAGTACGCCGGC | 63 | 433 |             |
| sul2 F                             | GCCTCAAGGCGACATGGCATT | 63 | 293 |             |
| sul2 R                             | GGTTTGGATACCGCCACCGGT | 63 | 293 |             |

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 (268 bp) and TspE4C2 (52 bp), well numbers 8 and 9 with trpAgpC (209 bp), and well numbers 10 and 11 with ArpAgpE (301 bp) genes.

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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, sulI, 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 cefotaxime (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 sulII resistance, and resistance to cefotaxime (P = 0.003), ceftriaxone (P = 0.011), cotrimoxazole (P = 0.003), and cefotaxime (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 resistance 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

| 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)   |
| sul1                       | 32 (68)     | 10 (83.3)  | 12 (70.5)  | 7 (70)     | 15 (57.6)  | 10 (76.9)       | 0 (0)    |
| 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 (15.4)   | 2 (15.4)        | 0 (0)    |
| CITM                       | 16 (34)     | 3 (25)     | 5 (29.4)   | 3 (30)     | 9 (34.6)   | 1 (23)          | 0 (0)    |
| qnr,sul1                   | 11 (23.4)   | 5 (41.7)   | 3 (17.6)   | 2 (20)     | 2 (7.69)   | 1 (25)          | 0 (0)    |
| sul1,sul2                  | 17 (36.17)  | 9 (75)     | 6 (35.2)   | 0          | 3 (15.7)   | 4 (30.7)        | 0 (0)    |
| 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 (15.7)   | 4 (30.7)        | 0 (0)    |
| qnr,sul1,dfrA              | 1 (2.1)     | 0 (0)      | 1 (5.8)    | 0          | 0 (0)      | 0 (0)           | 0 (0)    |

Values are expressed as No. (%).

Table 3. Distribution of Phylogenetic Groups Based on Gender

| 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 (16.6)   | 0 (0)    | 3 (30)     | 2 (15.4)        | 7 (26.9)       | 0.83    |
| Female| 36 (76.6)   | 12 (70.6)  | 10 (83.3)  | 4 (33.3) | 7 (70)     | 11 (84.6)       | 19 (73.1)      |         |

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

Table 4. Frequency of Antibiotic Resistance Among Phylogenetic Groups of Uropathogenic Escherichia coli

| Phylogenetic group | Ampicillin | Tetracycline | Sulfamethoxazole | Ciprofloxacin | Co-trimoxazole | Cefotaxime | Ceftriaxone | Aztreonam | Ceftazidim |
|--------------------|------------|--------------|------------------|--------------|----------------|------------|------------|-----------|-----------|
| B2 (n = 47)        | 36 (76.5)  | 28 (59.5)    | 32 (68)          | 33 (70)      | 25 (53)        | 22 (46.8)  | 22 (46.8)  | 28 (63.8) | 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)  | 4 (30.7)  |
| 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) |
| Unknown (n = 26)   | 24 (92.3)  | 20 (76.9)    | 10 (38.4)        | 17 (65)      | 14 (53.8)      | 6 (57.8)   | 6 (57.8)   | 22 (84.6) | 20 (76.9) |
| A (n = 4)          | 2 (50)     | 3 (75)       | 2 (50)           | 1 (25)       | 1 (25)         | 3 (75)     | 2 (50)     |           |           |
| Total              | 104 (80.6) | 85 (65.8)    | 67 (51.95)       | 82 (63.8)    | 65 (50.3)      | 63 (48.8)  | 65 (50.3)  | 85 (65.8) | 76 (58.9) |

P-value: 0.340 0.406 0.048 0.664 0.491 0.003 0.615 0.120 0.026

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

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 paraaminobenzoic acids (PABAs), which leads to the production of folic acid. The *hsu* 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*, *sul2*, *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 phylgroup 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 phylgroup (4, 10, 22, 25). Our study showed a significant relationship between phylogenetic

### Table 5. Distribution of Antimicrobial Resistance Genes in Antibiotic Resistance Patterns of *Escherichia coli* Strains Causing Urinary Tract Infections

| Antibiotic Resistance Genes | Ampicillin | Tetracycline | Nalidixic Acid | Co-Trimoxazole | Ciprofloxacin | Ceftazidim | Ceftazidim + Ceftaxim | Azteronam | CEZTAM |
|-----------------------------|------------|--------------|----------------|----------------|---------------|-------------|----------------------|-----------|--------|
| *tetA*                      | 69 (98.6)  | 55 (78.4)    | 37 (53.3)      | 60 (84.8)      | 59 (84.8)     | 14 (41.2)   | 35 (45.5)            | 34 (44.2) | 52 (70.5) |
| P-value                     | 0.005< 0.005< | 0.005< | 0.005< | 0.005< | 0.005< | 0.005< | 0.005< | 0.005< | 0.005< |
| *tetB*                      | 73 (84.9)  | 57 (78.4)    | 47 (65.7)      | 42 (59.4)      | 32 (52.8)     | 10 (30.6)   | 30 (40.5)            | 30 (40.5) | 38 (53.3) |
| P-value                     | 0.077< 0.005< | 0.077< | 0.077< | 0.077< | 0.077< | 0.077< | 0.077< | 0.077< | 0.077< |
| *sul1*                      | 75 (88.5)  | 52 (71.4)    | 48 (68.5)      | 60 (84.8)      | 50 (75)       | 22 (63.6)   | 40 (52.8)            | 40 (52.8) | 47 (52.8) |
| P-value                     | 0.209< 0.005< | 0.209< | 0.209< | 0.209< | 0.209< | 0.209< | 0.209< | 0.209< | 0.209< |
| *sul2*                      | 62 (73.5)  | 54 (73.5)    | 39 (56.9)      | 54 (78.9)      | 27 (76.4)     | 33 (74)     | 40 (67.2)            | 38 (67.2) | 45 (67.2) |
| P-value                     | 0.16< 0.005< | 0.16< | 0.16< | 0.16< | 0.16< | 0.16< | 0.16< | 0.16< | 0.16< |
| *qnr*                       | 22 (26.8)  | 14 (19.7)    | 18 (25.3)      | 14 (19.7)      | 14 (19.7)     | 14 (19.7)   | 14 (19.7)            | 14 (19.7) | 18 (24.2) |
| P-value                     | 0.65< 0.01< | 0.65< | 0.65< | 0.65< | 0.65< | 0.65< | 0.65< | 0.65< | 0.65< |
| *CITM*                      | 50 (61.5)  | 25 (34.0)    | 51 (70.5)      | 41 (57.1)      | 27 (74.2)     | 33 (74)     | 40 (53.3)            | 40 (53.3) | 45 (53.3) |
| P-value                     | 0.046< 0.01< | 0.046< | 0.046< | 0.046< | 0.046< | 0.046< | 0.046< | 0.046< | 0.046< |
| *dfrA*                      | 24 (29.5)  | 18 (24.6)    | 19 (26.3)      | 18 (24.6)      | 14 (19.7)     | 10 (28.6)   | 16 (40.5)            | 16 (40.5) | 18 (24.6) |
| P-value                     | 0.619< 0.01< | 0.619< | 0.619< | 0.619< | 0.619< | 0.619< | 0.619< | 0.619< | 0.619< |

*Values are expressed as No. (%) unless otherwise indicated. P-value < 0.05 is significant.*
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, sul1, and sul2 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

Authors’ Contribution: Study concept and design, Mostafa Boroumand and Mohsen Naghmachi; Analysis and interpretation of data, Mostafa Boroumand and Mohsen Naghmachi; Drafting of the manuscript, Mostafa Boroumand, Mohsen Naghmachi, and Mohammad Amin Ghati; Acquisition of data, Mostafa Boroumand; Study supervision, Mohsen Naghmachi.

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Ethical Approval: This study was registered as a research project approved by the Student Research Committee of Yasuj University of Medical Sciences under the code of ethics IR.YUMS.REC.1397.161.

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