



# International Journal of Health care and Biological Sciences

An International Online Open access Referred Journal  
Content available at [www.saajournals.org](http://www.saajournals.org)

Online ISSN: 2582-7499



## EVALUATION AND COMPARISON OF ANTIBIOTIC RESISTANCE OF PATHOGENIC AND COMMENSAL PHYLOGENETIC GROUP B2 ESCHERICHIA COLI

LAMYAA SABEEH ABDULLAH

Ministry of Education, General Directorate of Al-Qadisiyah Education, Diwaniyah, Iraq

DOI: <https://doi.org/10.46795/ijhcb.v7i3.956>

| ARTICLE HISTORY                                                                                                                                                                                                                                                                               | ABSTRACT                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   |
|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <p>Received on: 26-06-2026<br/>Revised on: 17-07-2026<br/>Accepted on: 08-09-2026</p> <p><b>Keywords:</b> <i>Escherichia coli</i>, <i>Phylogenetic groups</i>, <i>UTI</i>, <i>Commensal</i>, <i>antibiotic resistance</i>.</p> <p><b>*CORRESPONDING AUTHOR</b><br/>Lamyaa Sabeeh Abdullah</p> | <p><b>Background:</b> In this research, we evaluated the antibiotic resistance of phylogenetic group B2 of commensal and uropathogenic <i>Escherichia coli</i> (UPEC), since these isolates are considered the most virulent and the main cause of urinary tract infections, which may be transmitted from the digestive system to the urinary tract through fecal contamination.</p> <p><b>Methods:</b> Urine and stool samples were taken from 150 patients with urinary tract infection (UTI). Isolates were grown on selective media and named by biochemical tests. Each confirmed isolate was then phylotyped by multiplex PCR. The reaction amplified <i>chuA</i>, <i>yjaA</i> and the <i>TspE4.C2</i> fragment, following Clermont et al. (2000). Resistance was read by disc diffusion.</p> <p><b>Results:</b> Group B2 was carried by 59 (46%) UPEC and 30 (22%) commensal isolates. For most drugs the commensal B2 isolates were the more resistant of the two, but the gap was small. Tetracycline was the only drug to reach nominal significance (<math>P = 0.015</math>, Fisher's exact test), and it did not hold after correction for multiple comparisons. Every commensal B2 isolate was multi-drug resistant: 50% resisted 3–5 drugs and 13% resisted all ten, against 42% and 5% of UPEC B2 isolates.</p> <p><b>Conclusion:</b> Commensal and pathogenic B2 <i>E. coli</i> exhibited broadly similar antibiotic resistance profiles. No statistically significant differences survived correction for multiple testing, although a trend toward higher resistance among commensal isolates was observed. Both commensal and pathogenic isolates were most sensitive to ciprofloxacin and ceftriaxone, while they were highly resistant to the penicillin family.</p> |

This article is licensed under a Creative Commons Attribution-Non-commercial 4.0 International License.  
Copyright © 2026 Author(s) retains the copyright of this article.



### INTRODUCTION

Serotyping separates *E. coli* strains by three surface structures: the somatic O antigen, the flagellar H antigen and the capsular K antigen. The counts reported so far are 174, 56 and 80, respectively [1]. Most strains live in the gut without causing disease. A minority are pathogens, and these are sorted by the illness they produce into the enteropathogenic (EPEC), enterotoxigenic (ETEC), enteroinvasive (EIEC) and enterohemorrhagic (EHEC) groups [2,3]. A UTI that is treated poorly can leave lasting damage. Reported outcomes include renal scarring, raised blood pressure and chronic kidney disease, which is why these infections matter for public health [4].

About 150 million people are affected each year worldwide [5]. Direct costs in the United States alone reach roughly 3.5 billion dollars annually [6]. Phylogenetic variation in *E. coli* tracks several factors at once. Region, host habits, how often antibiotics are taken, the resistance already present, doubling time and virulence have all been linked to it [7]. Genome size splits along the same lines. Groups A and B1 carry less DNA than groups B2 and D [8]. A second scheme sorts the species by pathotype. Extra-intestinal pathotypes drive UTI, neonatal meningitis and bloodstream infection. Intestinal pathotypes drive diarrheal disease in adults and children [9,10]. Commensal isolates fall mostly in groups A and B1. Intestinal pathogens sit in groups B1, A and D, while

extra-intestinal pathogens sit mainly in groups B2 and D [11]. Grouping is done with a small set of marker sequences. These were chosen by screening gene libraries from strains of known group and keeping the fragments that separated them [12]. Of the three markers used here, *chuA* carries out heme transport in *E. coli* O157:H7, *yjaA* codes for a protein of unknown role, and *TspE4.C2* is a putative lipase esterase fragment [11–13].

Commensal *E. coli* present in the intestine is the main source of urinary tract infections in females, as it can easily move from the digestive system to the urinary system due to the short urethra [14]. Previous studies have shown that genotype B2 is the most important cause of urinary tract infections, and that it may be present as commensal bacteria in the digestive system. *E. coli* resistance to antibiotics is one of the most prominent obstacles that lead to the exacerbation of infections, especially urinary tract infections [15]. On the other hand, previous studies have shown that commensal bacteria, especially phylogenetic group B2, are resistant to most antibiotics. Therefore, we believe that the transmission of these bacteria to the urinary tract provides the patient with isolates that are difficult to treat [14,16,17]. The current study aims to isolate and diagnose these bacteria from the intestines and urinary tract and evaluate their response to antibiotics.

## MATERIALS AND METHODS

### Study Design and Sample Collection

This was a cross-sectional study. Paired urine and stool samples were taken from 150 patients with a urinary tract infection. All were first episodes with no prior treatment, and none of the patients had a gastrointestinal complaint. Sampling ran at Al-Diwaniyah General Teaching Hospital from January to March 2024. The hospital and every patient gave informed consent beforehand. Any sample that could not go straight to the microbiology laboratory was held in Cary-Blair transport medium.

### Isolation and Identification of *Escherichia coli*

Every stool specimen was first screened by microscopy for bacterial cells, parasites and other material. Chosen stool and urine specimens were streaked onto MacConkey agar. Plates were held overnight at 37°C in an aerobic bacteriological incubator. Rose-pink colonies were picked on shape and color, then passed to Eosin Methylene Blue agar for a further 24 h at 37°C. A green metallic sheen on this medium marked a presumptive *E. coli*. Biochemical tests settled the identification. Confirmed isolates were then grown twice: in nutrient broth, to supply DNA for the molecular work, and on Mueller-Hinton agar, for susceptibility testing [14].

### Antibiotic Susceptibility Test

Susceptibility of the confirmed isolates was read by the modified disc-diffusion technique. The ten discs listed in Table 1 were applied, and zones were interpreted against Clinical and Laboratory Standards Institute (CLSI) breakpoints. These ten agents are the panel

CLSI lists for routine testing and reporting of Enterobacteriaceae [14,15].

### DNA Extraction and Primer Preparation

Genomic DNA was recovered with the Genomic DNA Mini Kit (Geneaid, USA) as the manufacturer directs. A 0.5% agarose gel stained with 5 µL ethidium bromide was run to check that DNA was present. The extracts were then kept at –20°C until amplification. The primers of Table 2, taken from the triplex phylotyping assay of Clermont et al. [18], arrived lyophilized. Each was taken up in deionized distilled water to give a 100 pmol/µL stock. Working solutions of 10 pmol/µL were made from this stock in deionized water, with volumes set by CIV1 = C2V2.

Table 01: Antibiotic discs and their manufacturers

| Antibiotics   | Symbo<br>l | Concentratio<br>n (µg/disc) | Manufacture<br>r  |
|---------------|------------|-----------------------------|-------------------|
| Ciprofloxacin | CIP        | 10                          | Ankara,<br>Turkey |
| Amoxicillin   | AMX        | 25                          | Ankara,<br>Turkey |
| Ampicillin    | AMP        | 10                          | Ankara,<br>Turkey |
| Tetracycline  | TET        | 30                          | Ankara,<br>Turkey |
| Sulfonamide   | S          | 300                         | Ankara,<br>Turkey |
| Cefotaxime    | CTX        | 30                          | Ankara,<br>Turkey |
| Ceftriaxone   | CRO        | 30                          | Ankara,<br>Turkey |
| Imipenem      | IPM        | 10                          | Ankara,<br>Turkey |
| Meropenem     | MEM        | 10                          | Ankara,<br>Turkey |
| Cefepime      | FEP        | 30                          | Ankara,<br>Turkey |

Table 02: Triplex PCR primers for *E. coli* phylogenetic groups analysis [18]

| Gene                 |   | Oligonucleotide primer<br>sequence (5'–3') | Amplic<br>on (bp) |
|----------------------|---|--------------------------------------------|-------------------|
| <i>chuA</i>          | F | GACGAACCAACGGTCA<br>GGAT                   | 279               |
| <i>chuA</i>          | R | TGCCGCCAGTACCAAA<br>GACA                   | 279               |
| <i>yjaA</i>          | F | TGAAGTGTCTCAGGAGAC<br>GCTG                 | 211               |
| <i>yjaA</i>          | R | ATGGAGAATGCGTTCC<br>TCAAC                  | 211               |
| <i>TspE4.<br/>C2</i> | F | GAGTAATGTCTGGGGCA<br>TTCA                  | 152               |
| <i>TspE4.<br/>C2</i> | R | CGCGCCAACAAAGTAT<br>TACG                   | 152               |

Table 03: Interpretation key for phylogenetic group assignment based on triplex PCR [18]

| Phylogenetic Group | chuA | yjaA | TspE4.C2 |
|--------------------|------|------|----------|
| A                  | –    | –    | –        |
| B1                 | –    | –    | +        |
| B2                 | +    | +    | ±        |
| D                  | +    | –    | ±        |

### PCR Reaction Mixtures for Genotypic Analysis

Phylogenetic group was assigned by multiplex PCR. Reactions were set up as the supplier specifies (Geneaid, USA). Every primer set was run in a 0.2 mL AccuPower PCR PreMix tube, following the instructions of Bioneer Corporation. Each tube was vortexed until the lyophilized pellet had gone into solution and the contents were even, then loaded into a Professional TR/O thermocycler. The program opened with 3 min at 95°C. Then 30 cycles followed, each of 30 sec at 95°C, 10 sec at 59°C and 1 min at 72°C. A closing step of 5 min at 72°C completed the run. Amplicons were resolved on a 1.5% agarose gel [15].

### Statistical Analysis

Data were handled in SPSS version 17 with Microsoft Excel 2010. Proportions in the commensal and UPEC groups were compared by Fisher's exact test. Table 7 holds ten such comparisons made at once, so a Bonferroni threshold of  $P < 0.005$  ( $0.05 \div 10$ ) was applied there. Every test was two-tailed. A P value below 0.05 is reported as nominally significant.

## RESULTS

The present study included 150 UTI patients whose ages ranged from 19 to 77 years with a mean age of 39.55 years (Table 3). In this study, UTI patients did not have any gastrointestinal infection, and most of them (90%) harbored *E. coli* in their intestinal tract. Moreover, 85% of them had UTI resulting from uropathogenic *Escherichia coli* (UPEC).

Table 03: Demographic properties and microbial diagnosis of participants

| Properties               | Values           |
|--------------------------|------------------|
| Age range                | 19–77 years      |
| Age mean $\pm$ SD        | 39.55 $\pm$ 11.6 |
| Males (%)                | 63 (42%)         |
| Females (%)              | 87 (58%)         |
| Total number             | 150              |
| Microbial diagnosis      | N (%)            |
| UPEC                     | 128 (85%)        |
| Commensal <i>E. coli</i> | 135 (90%)        |

Figure 01 shows the banding patterns from which the groups were read. Group B2 accounted for 59 UPEC isolates (46%) and 30 commensal isolates (22%). Between the two collections the frequencies of groups

A, B2 and D differed sharply ( $P < 0.001$ , Fisher's exact test); group B1 did not. Table 4 lists the counts.

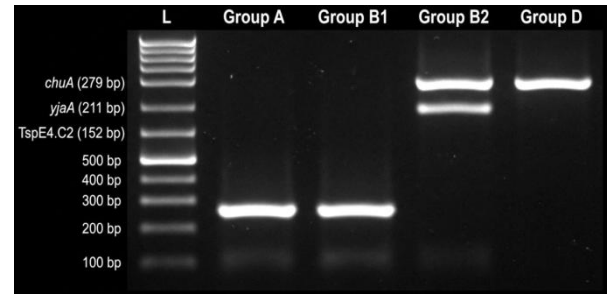


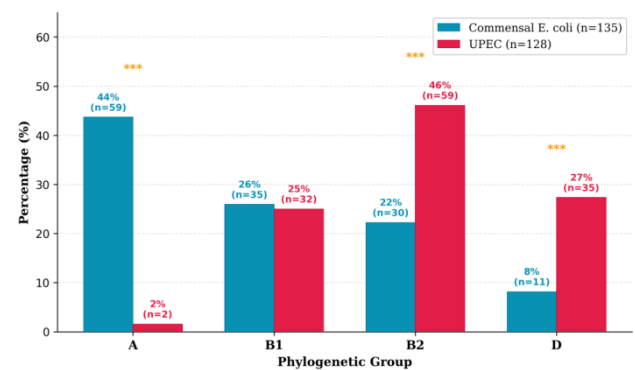
Figure 01: Triplex PCR banding patterns used to assign the four phylogenetic groups (A, B1, B2, D) from chuA, yjaA and TspE4.C2.

Table 04: Distribution of *E. coli* according to phylogenetic groups (Fisher's exact test)

| Phylogenetic groups | Commensal <i>E. coli</i> N (%) | UPEC N (%) | P value  |
|---------------------|--------------------------------|------------|----------|
| A                   | 59 (44%)                       | 2 (2%)     | < 0.001* |
| B1                  | 35 (26%)                       | 32 (25%)   | 0.888    |
| B2                  | 30 (22%)                       | 59 (46%)   | < 0.001* |
| D                   | 11 (8%)                        | 35 (27%)   | < 0.001* |
| Total               | 135                            | 128        |          |

\*  $P < 0.05$  (Fisher's exact test)

Figure 3: Distribution of *E. coli* Phylogenetic Groups in Commensal and UPEC Isolates



\*\*\*  $P < 0.001$ , Fisher's exact test

Figure 03: Distribution of *E. coli* phylogenetic groups (A, B1, B2, D) in commensal and UPEC isolates.

After conducting antibiotic susceptibility testing, we found high resistance to amoxicillin, ampicillin, tetracycline, and cefotaxime among commensal bacteria (100%, 100%, 100%, and 93%, respectively) and pathogenic bacteria (97%, 100%, 83%, and 85%, respectively). On the other hand, ciprofloxacin and ceftriaxone were the most effective antibiotics against commensal bacteria, with resistance rates of 17% and 7%, respectively. This pattern was also found in pathogenic bacteria, where the resistance rates were 10% and 8%, as shown in Tables 05 and 06. The carbapenems imipenem and meropenem were relatively effective against the studied isolates,

especially the pathogenic phylogenetic group B2 (17% and 14% resistance, respectively), while the resistance rates among commensal phylogenetic group B2 appeared higher, at 30% and 20%, respectively.

Table 05: Antibiotic resistance of phylogenetic group B2 commensal E. coli (n = 30)

| Antibiotics   | Resistance N (%) | Sensitivity N (%) |
|---------------|------------------|-------------------|
| Ciprofloxacin | 5 (17)           | 25 (83)           |
| Amoxicillin   | 30 (100)         | 0 (0)             |
| Ampicillin    | 30 (100)         | 0 (0)             |
| Tetracycline  | 30 (100)         | 0 (0)             |
| Sulfonamide   | 21 (70)          | 9 (30)            |
| Cefotaxime    | 28 (93)          | 2 (7)             |
| Ceftriaxone   | 2 (7)            | 28 (93)           |
| Imipenem      | 9 (30)           | 21 (70)           |
| Meropenem     | 6 (20)           | 24 (80)           |
| Cefepime      | 19 (63)          | 11 (37)           |

Table 06: Antibiotic resistance of phylogenetic group B2 UPEC (n = 59)

| Antibiotics   | Resistance N (%) | Sensitivity N (%) |
|---------------|------------------|-------------------|
| Ciprofloxacin | 6 (10)           | 53 (90)           |
| Amoxicillin   | 57 (97)          | 2 (3)             |
| Ampicillin    | 59 (100)         | 0 (0)             |
| Tetracycline  | 49 (83)          | 10 (17)           |
| Sulfonamide   | 31 (53)          | 28 (47)           |
| Cefotaxime    | 50 (85)          | 9 (15)            |
| Ceftriaxone   | 5 (8)            | 54 (92)           |
| Imipenem      | 10 (17)          | 49 (83)           |
| Meropenem     | 8 (14)           | 51 (86)           |
| Cefepime      | 43 (73)          | 16 (27)           |

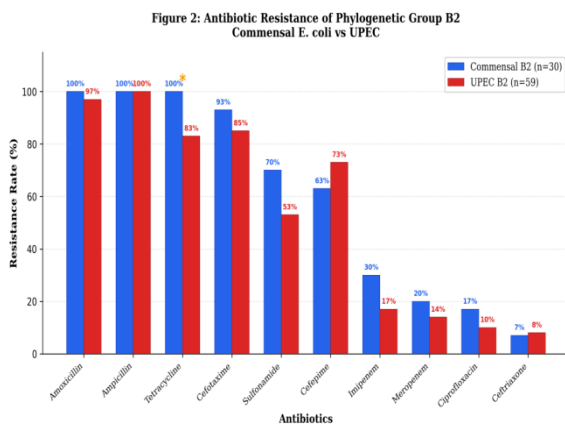


Figure 02: Frequency of antibiotic resistance of phylogenetic group B2 of commensal E. coli and UPEC.

When comparing antibiotic resistance between commensal and pathogenic phylogenetic group B2 (Table 7), commensal B2 isolates showed numerically higher resistance rates to most antibiotics compared to pathogenic phylogenetic group B2. However, of the ten comparisons performed using Fisher's exact test, only

tetracycline reached nominal significance (P = 0.015). After Bonferroni correction for ten simultaneous comparisons (adjusted threshold P < 0.005), no individual antibiotic showed a statistically significant difference between the two groups.

Table 07: Comparison of antibiotic resistance between phylogenetic group B2 of commensal E. coli and UPEC (Fisher's exact test)

| Antibiotics   | B2 Commensal N (%) | B2 UPEC N (%) | P value | OR (95% CI)         |
|---------------|--------------------|---------------|---------|---------------------|
| Ciprofloxacin | 5 (17)             | 6 (10)        | 0.498   | 1.77 (0.49 – 6.34)  |
| Amoxicillin   | 30 (100)           | 57 (97)       | 0.548   | —                   |
| Ampicillin    | 30 (100)           | 59 (100)      | 1.000   | —                   |
| Tetracycline  | 30 (100)           | 49 (83)       | 0.015 * | —                   |
| Sulfonamide   | 21 (70)            | 31 (53)       | 0.172   | 2.11 (0.83 – 5.36)  |
| Cefotaxime    | 28 (93)            | 50 (85)       | 0.322   | 2.52 (0.51 – 12.49) |
| Ceftriaxone   | 2 (7)              | 5 (8)         | 1.000   | 0.77 (0.14 – 4.23)  |
| Imipenem      | 9 (30)             | 10 (17)       | 0.178   | 2.10 (0.75 – 5.91)  |
| Meropenem     | 6 (20)             | 8 (14)        | 0.540   | 1.59 (0.50 – 5.11)  |
| Cefepime      | 19 (63)            | 43 (73)       | 0.465   | 0.64 (0.25 – 1.64)  |

\* Nominally significant at P < 0.05; does not survive Bonferroni correction (threshold P < 0.005)

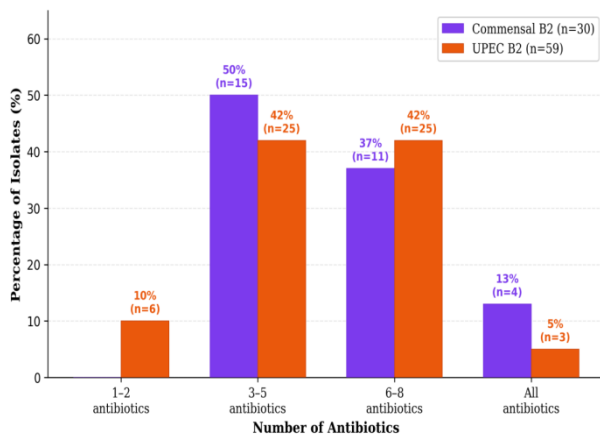
OR = Odds Ratio; CI = Confidence Interval; — = not estimable due to zero cells

When evaluating multiple antibiotic resistance (MAR), we found that 10% of UPEC phylogenetic group B2 were resistant to 1 to 2 antibiotics, while all commensal phylogenetic group B2 were resistant to at least 3 antibiotics. We found 50% of commensal B2 resistant to 3–5 antibiotics, and 13% resistant to all studied antibiotics, compared to 42% and 5% of UPEC

B2, respectively. However, none of these differences reached statistical significance (Table 8).

Table 08: Multiple antibiotic resistance patterns of studied isolates (Fisher's exact test)

| Number of antibiotics | B2 Commensal N (%) | B2 UPEC N (%) | P value |
|-----------------------|--------------------|---------------|---------|
| 1–2                   | 0 (0%)             | 6 (10%)       | 0.093   |
| 3–5                   | 15 (50%)           | 25 (42%)      | 0.509   |
| 6–8                   | 11 (37%)           | 25 (42%)      | 0.653   |
| All antibiotics       | 4 (13%)            | 3 (5%)        | 0.220   |
| Total                 | 30                 | 59            |         |



No significant differences ( $P > 0.05$ , Fisher's exact test)

Figure 04: Multiple antibiotic resistance (MAR) patterns of phylogenetic group B2 commensal E. coli and UPEC.

## DISCUSSION

The results of the current study showed that phylogenetic group B2 constitutes 22% of the commensal E. coli in the large intestine, while it constitutes 46% of the E. coli that causes urinary tract infections, which may have been transmitted to the urinary tract due to fecal contamination from the digestive system. The present study is one of the first to specifically compare group B2 in commensal and pathogenic isolates, as this group is the main cause of urinary tract infections according to previous studies [15,16]. We also found that these isolates have high resistance to antibiotics, especially the commensal group B2, which in turn, when transmitted through contamination to other body systems, are pathogenic and difficult to treat [14,19,20].

Every commensal B2 isolate in this work was multi-drug resistant. Three routes could bring that about. Resistance genes may sit together on one mobile element and be selected as a set. One mechanism may cover more than one drug class. Or the host may simply have met several agents in turn. A narrow phenotype of one or two drugs is easy to explain this way. Losing amoxicillin or tetracycline often means losing the rest of that class as well. A phenotype spanning 9 or 10 agents across unrelated classes is not

so easy. Patterns that wide point instead to plasmids that carry resistance genes [15,21–23].

Published work on group B2 does not agree. Anantham [24] reported chloramphenicol and tetracycline gene cassettes at high frequency in group D and at low frequency in group B2. Skurnik et al. [25] had earlier placed B2, whether commensal or pathogenic, among the least resistant groups, with multi-drug resistance concentrated elsewhere. Our B2 isolates behave unlike either report. Closer to home, Khadhim and Kazaal [14] tied multiple resistance patterns to Int12-positive isolates of groups A and B2, which may go some way to explaining the rates seen here.

In our hands the commensal B2 isolates were the more resistant of the two sets. The margin was narrow. No drug held its difference once multiple comparisons were accounted for. Tetracycline alone was nominally significant ( $P = 0.015$ , Fisher's exact test), and it fell below the Bonferroni threshold. Two features of the gut may account for the trend. Repeated antibiotic courses reach the colon and leave behind whatever survives them. The colon also holds a dense mixed flora, and plasmids carrying resistance genes pass between its members by several routes [26,27]. Resistant organisms taken in with food or water add to the same pool. What matters here is that the two B2 populations resist in much the same pattern. That similarity fits the idea that commensal B2 organisms move from the gut to the urinary tract and cause infection there [28].

## CONCLUSION

The results of our research showed that phylogenetic group B2 constitutes 22% of the commensal E. coli and 46% of the UPEC, with this difference being highly significant ( $P < 0.001$ ). While all commensal B2 isolates were multi-drug resistant and showed numerically higher resistance rates than UPEC B2 isolates, no statistically significant differences in individual antibiotic resistance survived correction for multiple comparisons. Only tetracycline showed a nominally significant difference ( $P = 0.015$ ). Both commensal and pathogenic B2 isolates were most sensitive to ciprofloxacin and ceftriaxone, while they were highly resistant to the penicillin family. These findings suggest that commensal B2 E. coli in the intestine may serve as a reservoir of antibiotic-resistant strains that can cause urinary tract infections.

## REFERENCES

1. Fratamico PM, DebRoy C, Liu Y, Needleman DS, Baranzoni GM, Feng P. Advances in Molecular Serotyping and Subtyping of Escherichia coli. Front Microbiol. 2016;7:644.
2. Ramos S, Silva V, Dapkevicius MLE, Caniça M, Tejedor-Junco MT, Igrejas G, Poeta P. Escherichia coli as Commensal and Pathogenic Bacteria Among Food-Producing Animals: Health Implications of

- Extended Spectrum  $\beta$ -lactamase (ESBL) Production. *Animals* (Basel). 2020;10(12):2239.
3. Mbelle NM, Feldman C, Osei Sekyere J, Maningi NE, Modipane L, Essack SY. The Resistome, Mobilome, Virulome and Phylogenomics of Multidrug-Resistant *Escherichia coli* Clinical Isolates from Pretoria, South Africa. *Sci Rep*. 2019;9(1):16457.
4. Foxman B. Urinary tract infection syndromes: occurrence, recurrence, bacteriology, risk factors, and disease burden. *Infect Dis Clin North Am*. 2014;28(1):1–13.
5. Flores-Mireles AL, Walker JN, Caparon M, Hultgren SJ. Urinary tract infections: epidemiology, mechanisms of infection and treatment options. *Nat Rev Microbiol*. 2015;13(5):269–284.
6. Foxman B. Epidemiology of urinary tract infections: incidence, morbidity, and economic costs. *Am J Med*. 2002;113 Suppl 1A:5S–13S.
7. Duriez P, Clermont O, Bonacorsi S, Bingen E, Chaventré A, Elion J, Picard B, Denamur E. Commensal *Escherichia coli* isolates are phylogenetically distributed among geographically distinct human populations. *Microbiology* (Reading). 2001;147(Pt 6):1671–1676.
8. Bergthorsson U, Ochman H. Distribution of chromosome length variation in natural isolates of *Escherichia coli*. *Mol Biol Evol*. 1998;15(1):6–16.
9. Kaper JB, Nataro JP, Mobley HL. Pathogenic *Escherichia coli*. *Nat Rev Microbiol*. 2004;2(2):123–140.
10. Russo TA, Johnson JR. Proposal for a new inclusive designation for extraintestinal pathogenic isolates of *Escherichia coli*: ExPEC. *J Infect Dis*. 2000;181(5):1753–1754.
11. Suits MD, Lang J, Pal GP, Couture M, Jia Z. Structure and heme binding properties of *Escherichia coli* O157:H7 ChuX. *Protein Sci*. 2009;18(4):825–838.
12. Askari N, Ghanbarpour R. Molecular investigation of the colicinogenic *Escherichia coli* strains that are capable of inhibiting *E. coli* O157:H7 in vitro. *BMC Vet Res*. 2019;15(1):14.
13. Kuznetsova MV, Mihailovskaya VS, Remezovskaya NB, Starčić Erjavec M. Bacteriocin-Producing *Escherichia coli* Isolated from the Gastrointestinal Tract of Farm Animals: Prevalence, Molecular Characterization and Potential for Application. *Microorganisms*. 2022;10(8):1558.
14. Khadhim MK, Kazaal MA. Antibiotics Resistance and Integron Class I among Commensal *Escherichia coli*. *AL-Qadisiya Medical Journal*. 2017;13(24):1–10.
15. Khadhim MM, Kazaal MA. Association Between Antibiotic Resistance and Integron Class 2 Among Commensal *Escherichia coli* Genotypic Groups. *AL-Qadisiya Medical Journal*. 2018;14(25):1–15.
16. Bibbal D, Ruiz P, Sapountzis P, Mazuy-Cruchaudet C, Loukiadis E, Auvray F, Forano E, Bruguère H. Persistent Circulation of Enterohemorrhagic *Escherichia coli* (EHEC) O157:H7 in Cattle Farms. *Front Vet Sci*. 2022;9:852475.
17. Lange ME, Clarke ST, Boras VF, et al. Commensal *Escherichia coli* Strains of Bovine Origin Competitively Mitigated *Escherichia coli* O157:H7 in a Gnotobiotic Murine Intestinal Colonization Model. *Animals* (Basel). 2023;13(16):2577.
18. Clermont O, Bonacorsi S, Bingen E. Rapid and simple determination of the *Escherichia coli* phylogenetic group. *Appl Environ Microbiol*. 2000;66(10):4555–4558.
19. Kazaal MA, Hamad WA, Atiya WH, Saeed BJ, Abd-Alsatar AN. Impact of Antibiotic Resistance on Sustainable Development Goals. *AIP Conference Proceedings*. 2023;2776:020016.
20. Pais S, Costa M, Barata AR, Rodrigues L, Afonso IM, Almeida G. Evaluation of Antimicrobial Resistance of Different Phylogroups of *Escherichia coli* Isolates from Feces of Breeding and Laying Hens. *Antibiotics* (Basel). 2022;12(1):20.
21. Bakhtiari S, Mahmoudi H, Seftjani SK, Amirzargar MA, Ghiasvand S, Ghaffari ME, Adabi M. Antibiotic resistance pattern and phylogenetic groups of the uropathogenic *Escherichia coli* isolates from urinary tract infections in Hamedan, west of Iran. *Iran J Microbiol*. 2020;12(5):388–394.
22. Iranpour D, Hassanpour M, Ansari H, Tajbakhsh S, Khamisipour G, Najafi A. Phylogenetic groups of *Escherichia coli* strains from patients with urinary tract infection in Iran based on the new Clermont phylotyping method. *Biomed Res Int*. 2015;2015:846219.
23. Sohrabi R, Zeighami H. Determination of phylogenetic groups and antibiotic resistance in uropathogenic and commensal *Escherichia coli* isolated from patients in Zanjan City. *J Adv Med Biomed Res*. 2016;24:107–118.
24. Anantham S. Analysis of persistent and antibiotic resistant commensal *Escherichia coli* from healthy adults. PhD thesis. School of Molecular Bioscience, University of Sydney, Australia. 2014:1–279.
25. Skurnik D, Le Menac'h A, Zurakowski D, Mazel D, Courvalin P, Denamur E, Andremonet A, Ruimy R. Integron-associated antibiotic resistance and phylogenetic grouping of *Escherichia coli* isolates from healthy subjects free of recent antibiotic exposure. *Antimicrob Agents Chemother*. 2005;49(7):3062–3065.
26. Maguire AJ, Brown DF, Gray JJ, Desselberger U. Rapid screening technique for class I integrons in Enterobacteriaceae and nonfermenting gram-negative bacteria and its use in molecular epidemiology. *Antimicrob Agents Chemother*. 2001;45(4):1022–1029.
27. Whelan S, Lucey B, Finn K. Uropathogenic *Escherichia coli* (UPEC)-Associated Urinary Tract Infections: The Molecular Basis for Challenges to Effective Treatment. *Microorganisms*. 2023;11(9):2169.

28. Norouzian H, Katouli M, Shahrokhi N, Sabeti S, Pooya M, Bouzari S. The relationship between phylogenetic groups and antibiotic susceptibility patterns of *Escherichia coli* strains isolated from feces and urine of patients with acute or recurrent urinary tract infection. *Iran J Microbiol.* 2019;11(6):478–487.