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Prevalence, Antimicrobial Resistance, and Phylogenetic Characterization of Esbl-Producing *Escherichia Coli* Isolated from Clinical Specimens in Can Tho, Vietnam

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ABSTRACT

Extended-spectrum *β*-lactamase (ESBL)-producing *Escherichia coli* poses a significant public health threat due to their capacity to inactivate broad-spectrum *β*-lactam antibiotics and accumulate multidrug resistance. This study aimed to investigate the prevalence, antimicrobial resistance profiles, ESBL-encoding genes, and phylogenetic groups of ESBL-producing *E. coli* isolated from clinical specimens at a major hospital in Can Tho, Vietnam. A total of 148 isolates were obtained from healthy individuals between 2024 and 2025, primarily from pus, sputum, urine, and blood samples, although the relatively small sample size may not fully represent the wider Can Tho population. Specimens were analyzed using selective culture, biochemical identification, and PCR assays for resistance genes (*blaCTX-M*, *blaTEM*, *blaSHV*) and phylogenetic groups (A, B1, B2, D). ESBL production was confirmed in 58.7% of isolates. Group D was predominant (42.0%), especially in extraintestinal specimens such as pus and sputum, highlighting its potential role in both asymptomatic carriage and clinical infection. High resistance rates were observed to ampicillin (99.3%), cefazolin (76.1%), and ciprofloxacin (71.7%), with amikacin showing the highest efficacy (97.1%). Gender-related variations in resistance were also noted, particularly in *β*-lactam response. Multi-drug, extensively-drug, and pan-drug resistance patterns significantly correlated with phylogenetic background. These findings underscore the urgent need for surveillance and stewardship strategies, particularly targeting high-risk clones such as group D to prevent community transmission.

Keywords: Antimicrobial resistance, *Escherichia coli*, Extended-spectrum *β*-lactamase, Phylogenetic groups, Can Tho, Vietnam.

Introduction

The worldwide emergence of *βEscherichia coli* strains capable of producing extended-spectrum *β*-lactamases (ESBLs) poses a serious challenge to public health, affecting both healthcare facilities and the broader community. These organisms hydrolyze third-generation cephalosporins and monobactams, compromising first-line antibiotics and increasing treatment failures, morbidity, mortality, and healthcare costs.[1, 2] Although initially associated with nosocomial infections, ESBL-producing *E. coli* are now frequently isolated from community settings, especially in developing countries.

In the Asia-Pacific region, surveillance programs have reported high prevalence rates. For instance, a multicenter study (2010–2013) across 13 countries found *E. coli* responsible for 46.1% of intra-abdominal infections, with an ESBL production rate of 38.2%, highest in India, China, Thailand, and Vietnam. In Latin America, 26.8% of *E. coli* from similar infections produced ESBL.[2] While in Mali, 64.5% of blood-culture isolates were ESBL-positive.[3] These trends reflect growing antimicrobial resistance (AMR) burdens globally.

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In Vietnam, both hospital and community data indicate rising ESBL prevalence. In Ba Vi, 46.2% of healthy individuals harbored ESBL-producing *E. coli*, and in Ho Chi Minh City, this rate reached 63.1%.[1, 4-6] Contributing factors include unregulated antibiotic use, animal contact, and poor sanitation—especially in peri-urban and rural areas..

In addition to *β*-lactam resistance, ESBL-producing *E. coli* often show resistance to fluoroquinolones, aminoglycosides, sulfonamides, and even colistin. The detection of the plasmid-mediated *mcr-1* gene, conferring colistin resistance, in both human and animal isolates in Vietnam, intensifies the concern over pan-drug-resistant strains.[7, 8] These genes are often harbored on mobile genetic elements, facilitating rapid horizontal transfer and dissemination across bacterial populations and host species.

Beyond resistance, the coexistence of virulence traits poses added risk. Enteropathogenic *E. coli* (EPEC) and enteroaggregative *E. coli* (EAEC) have carried ESBL genes in previous studies.[8, 9] Notably, 66.7% of EAEC strains in healthy elderly individuals harbored both virulence and resistance genes, suggesting potential for asymptomatic transmission.

Phylogenetic classification into groups A, B1, B2, and D helps predict pathogenicity. Groups B2 and D are commonly associated with extraintestinal pathogenic *E. coli* (ExPEC), while A and B1 are often commensal. Resistance patterns vary by group: B2 and D tend to resist *β*-lactams, whereas A and B1 show more fluoroquinolone resistance.[8, 10] However, data on phylogroup-specific resistance in healthy Vietnamese carriers remain limited.

The novelty of this study lies in its comprehensive characterization of ESBL-producing *E. coli* from non-hospitalized individuals using phylogenetic analysis, resistance profiling, and genotypic screening—an approach scarcely reported in Vietnam. In particular, the association of resistance genes with phylogroups offers insight into potential reservoirs and transmission pathways in the community.

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This study investigated the prevalence, antibiotic resistance patterns, ESBL gene types, and phylogenetic groups of *E. coli* strains collected from healthy individuals in Can Tho, Vietnam. The results may help guide more effective antibiotic use policies and public health strategies in Vietnam and other low- and middle-income countries.

Materials And Methods

Study Design, Setting, and Sampling

This descriptive, cross-sectional study was conducted from July 2024 to July 2025 at Can Tho General Hospital, a major tertiary care facility in Can Tho City, Vietnam. Ethical approval was obtained from the Institutional Ethics Committee in Biomedical Research of Can Tho University of Medicine and Pharmacy (Approval No. 23.003/PCT-HDDD, dated June 15, 2023).

A total of 148 clinical specimens were collected from apparently healthy individuals visiting the hospital for routine check-ups or minor complaints. Inclusion criteria required participants to be afebrile and asymptomatic, without recent (within 3 months) antibiotic usage. All participants provided informed consent. The collected samples included pus, sputum, urine, and blood. Isolates were analyzed to determine both phenotypic characteristics and genetic profiles related to antimicrobial resistance.

Bacterial Isolation and Identification

All specimens were inoculated on MacConkey agar (Merck, Germany) supplemented with cefotaxime (1 µg/mL; Sigma-Aldrich, USA) to facilitate the selective growth of ESBL-producing *E. coli*. The plates were incubated at 37°C for 24 ± 3 hours. Colonies exhibiting typical *E. coli* morphology (pink to red lactose-fermenting colonies) were subjected to biochemical identification using standard tests: Triple Sugar Iron (TSI), Lysine Indole Motility (LIM), and Cellobiose-Lactose-Indole-*β*-d-Glucuronidase (CLIG) media.[11] For ambiguous results, identification was confirmed using API 20E test kits (BioMérieux, USA).

Antimicrobial Susceptibility Testing

Antimicrobial susceptibility testing was performed using the Kirby-Bauer disk diffusion method on Mueller-Hinton agar (OXOID, UK), following Clinical and Laboratory Standards Institute (CLSI) guidelines (M100-S25). A total of 148 *E. coli* isolates were evaluated for antimicrobial susceptibility using seven representative antibiotics from major therapeutic classes: ampicillin (AMP), ceftazidime (CAZ), ceftriaxone (CRO), cefazolin (CFZ), amikacin (AMK), gentamicin (GEN), and ciprofloxacin (CIP). *β*Antibiotic discs were obtained from BD BBL™ Sensi-Disc™ (Becton Dickinson, USA). Susceptibility results were categorized as Susceptible (S), Intermediate (I), or Resistant (R) according to CLSI interpretive standards. All tests were conducted in duplicate to validate consistency and reliability.

DNA Extraction and Preparation

Genomic DNA was extracted from confirmed *E. coli* colonies using a modified heat-shock protocol as previously described.[4, 12] Bacterial suspensions were centrifuged at 13,000 rpm for 5 minutes, washed twice with sterile phosphate-buffered saline (PBS, pH 7.4; Thermo Fisher, USA), and resuspended in 50 µL of TE buffer (10 mM Tris-HCl, 1 mM EDTA, pH 8.0). The suspension was heated at 95°C for 10 minutes to lyse the cells. DNA yield and purity were assessed by NanoDrop 2000c spectrophotometer (Thermo Fisher Scientific, USA), and aliquots were stored at −20°C for PCR amplification.

Molecular Characterization of Isolates

Multiplex PCR was performed to detect three major ESBL-encoding genes: *blaCTX-M*, *blaTEM*, and *blaSHV*, using published primer sets.[13] Each PCR reaction had a total volume of 25 µL, including 12.5 µL of DreamTaq Green PCR Master Mix (Thermo Fisher, USA), 0.5 µM of each primer, 2 µL of template DNA, and nuclease-free water to make up the final volume. The PCR process started with an initial denaturation at 95°C for 5 minutes, followed by 35 cycles of denaturation at 95°C for 30 seconds, annealing at 60°C for 30 seconds, and extension at 72°C for 1 minute. Finally, the reaction ended with a final extension at 72°C for 7 minutes.

For phylogenetic classification, a multiplex PCR assay targeting three marker genes—*chuA* (279 bp), *yjaA* (211 bp), and *TspE4C2* (152 bp)—was performed. The primers used were as follows: for *chuA*, the forward primer was 5'-GACGAACCAACGGTCAGGAT-3' and the reverse primer was 5'-TGCCGCCAGTACCAAAGACA-3'; for *yjaA*, the forward primer was 5'-TGAAGTGTCAGGAGACGCTG-3' and the reverse primer was 5'-ATGGAGAATGCGTTCCTCAAC-3'; and for *TspE4C2*, the forward primer was 5'-GAGTAATGTCGGGGCATTCA-3' and the reverse primer was 5'-CGCGCCAACAAAGTATTACG-3'.

Ethical Considerations

This study was approved by the Ethics Committee in Biomedical Research of Can Tho University of Medicine and Pharmacy (Approval No. 23.003/PCT-HĐĐĐ, June 15, 2023). Participation was voluntary. Written informed consent was obtained from all participants or their legal guardians. Participants received detailed information about the study and were provided with appropriate counseling regarding hygiene practices and antimicrobial use.

Data analysis

Statistical analysis was performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA; 2019 release). Descriptive methods were applied to summarize the distribution of variables. Relationships between categorical data—such as resistance profiles and phylogenetic groupings—were evaluated using Chi-square tests or Fisher's exact tests when necessary. A p-value less than 0.05 was considered statistically significant.

Results And Discussion

Distribution of Phylogenetic Groups by Specimen Type

Table 1 presents the distribution of 148 *E. coli* isolates across four clinical specimen types—blood (n = 2), pus (n = 67), urine (n = 23), and sputum (n = 46)—classified into four phylogenetic groups (A1, B1, B2, D). Overall, group D was the most frequently identified (42.0%), followed by group A1 (28.3%), B2 (15.9%), and B1 (13.8%).

In blood samples (n = 2), isolates belonged to groups B2 and D (each 50.0%), with no representation of groups A1 or B1. Among pus isolates, group D predominated (53.7%), followed by A1 (25.4%), B2 (13.4%), and B1 (7.5%). For urine samples, groups A1 and D were equally prevalent (39.1%), with lower proportions of B1 (13.0%) and B2 (8.7%). In sputum, groups A1 and D remained common (28.3% and 26.1%), while B2 and B1 accounted for 21.7% and 23.9% of isolates.

Statistical analysis showed a significant association between phylogenetic group and specimen type for group D ($p = 0.020$), particularly in pus ($p = 0.031$) and sputum ($p = 0.013$). No significant differences were observed for groups A1, B1, or B2 ($p > 0.05$). Fisher's Exact Test was used where applicable.

Importantly, the small number of blood specimens (n = 2) limits the generalizability of findings for this group. Future studies should aim to include larger and more balanced sample sizes across all specimen types to strengthen statistical power and improve the representativeness of observed phylogenetic patterns.

These findings suggest that phylogenetic group D is particularly enriched in extraintestinal specimens (pus and sputum), supporting its potential role in invasive infections. Group B2 also appeared in blood and sputum, aligning with its known association with extraintestinal pathogenic *E. coli* (ExPEC). Meanwhile, group B1, typically associated with commensal strains, showed no significant enrichment in any specimen type.

Antibiotic Resistance Profiles of E. coli Isolates to β-lactams and Other Common Agents

Table 2 summarizes the antimicrobial susceptibility profiles of 148 *E. coli* isolates tested against seven commonly used antibiotics, with a

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focus on *β*-lactam agents. The data reveal alarmingly high resistance rates across most *β*-lactams.

Ampicillin exhibited the highest resistance, with 99.3% of isolates classified as resistant and only 0.7% as susceptible ($p < 0.001$). Resistance to cefazolin and ceftazidime was also high, observed in 76.1% and 68.8% of isolates. For ceftriaxone, 57.2% of isolates were resistant and 42.0% remained susceptible, while intermediate resistance was rare (0.7%).

In contrast, amikacin demonstrated excellent activity, with 97.1% of isolates susceptible and only 2.2% resistant. Gentamicin showed moderate activity, with 65.2% of isolates susceptible and 34.8% resistant. Resistance to ciprofloxacin was notably high (71.7%), suggesting a decline in fluoroquinolone effectiveness in the community setting.

All tested antibiotics showed statistically significant differences in resistance profiles ($p < 0.001$), except for the intermediate category, which was non-significant ($p = 1.000$).

These findings highlight a troubling pattern of widespread resistance to first-line *β*-lactams and fluoroquinolones*β* among community-acquired *E. coli* strains. The continued effectiveness of amikacin is encouraging; however, it must be preserved through judicious use, especially given the increasing gentamicin resistance observed. These data underscore the urgent need for revised empirical treatment strategies that reflect local resistance trends and support the implementation of targeted antibiotic stewardship programs.

Table 1: Distribution of *E. coli* Phylogenetic Groups by Specimen Type

Phylogenetic Group	Total (n = 148)	Blood (n = 2)	Pus (n = 67)	Urine (n = 23)	Sputum (n = 46)	p-value
Group A1 (%)	28.3	0.0	25.4	39.1	28.3	0.553
Group B1 (%)	13.8	0.0	7.5	13.0	23.9	0.086
Group B2 (%)	15.9	50.0	13.4	8.7	21.7	0.208
Group D (%)	42.0	50.0	53.7	39.1	26.1	0.020*
p-value	—	0.568	0.031*	0.587	0.013*	—

*Statistically significant ($p < 0.05$)

Table 2: Antibiotic Susceptibility Profiles of *E. coli* Isolates (n = 148)

Antibiotic	Resistant (%)	Susceptible (%)	Intermediate (%)	p-value
Ampicillin (AMP)	99.3	0.7	0.0	<0.001
Ceftazidime (CAZ)	68.8	31.2	0.0	<0.001
Ceftriaxone (CRO)	57.2	42.0	0.7	<0.001
Cefazolin (CFZ)	76.1	23.2	0.7	<0.001
Amikacin (AMK)	2.2	97.1	0.7	<0.001
Gentamicin (GEN)	34.8	65.2	0.0	<0.001
Ciprofloxacin (CIP)	71.7	27.5	0.7	<0.001
p-value	<0.001	<0.001	1.000	—

Multidrug Resistance Patterns by E. coli Phylogenetic Group

The distribution of *E. coli* strains exhibiting multidrug resistance (MDR), extensive drug resistance (EDR), and pan drug resistance (PDR) across the four phylogenetic groups is summarized in Table 3. Analysis of the 148 isolates revealed notable differences in resistance profiles among the groups.

Table 3: Distribution of MDR, EDR, and PDR among *E. coli* Phylogenetic Groups

Phylogenetic Group	MDR (%)	EDR (%)	PDR (%)	p-value
Group A1 (n = 39)	15.4	74.4	5.1	0.019*
Group B1 (n = 19)	52.6	36.8	10.5	0.141*
Group B2 (n = 22)	18.2	72.7	4.5	0.294*
Group D (n = 58)	41.4	41.4	12.1	0.045*
p-value	0.005	0.001	0.610	—

Note: MDR: Multidrug-resistant; EDR: Extensively drug-resistant; PDR: Pan drug-resistant

In group A1 (n = 39), the majority of isolates exhibited EDR (74.4%), while a smaller proportion were classified as MDR (15.4%) and PDR (5.1%). Group B1 (n = 19) showed the highest rate of MDR (52.6%) among all groups, alongside EDR at 36.8% and PDR at 10.5%. Group B2 (n = 22) demonstrated a similar EDR rate to group A1 (72.7%), but lower MDR (18.2%) and PDR (4.5%). Notably, group D (n = 58) exhibited balanced proportions of MDR and EDR (both 41.4%), and the highest PDR rate observed (12.1%).

Statistical analysis revealed significant associations between phylogenetic group and both MDR ($p = 0.005$) and EDR ($p = 0.001$), while differences in PDR distribution did not reach significance ($p = 0.610$). Within-group comparisons were statistically significant in groups A1 ($p = 0.019$) and D ($p = 0.045$), suggesting that specific phylogroups are strongly associated with particular resistance phenotypes.

These findings underscore the emergence of extensive resistance in community-acquired *E. coli*, particularly among group A1 and B2 isolates, which displayed high EDR rates. Group B1, though traditionally considered less virulent, had the highest MDR rate, indicating its potential as a silent reservoir of resistance. The elevated

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PDR rate in group D, combined with its clinical association with extraintestinal infections, positions it as a critical target for surveillance. Overall, the significant correlation between resistance levels and phylogenetic background highlights the need for genotypically informed resistance monitoring.

Distribution of Resistance and Phylogenetic Marker Genes by E. coli Phylogenetic Group

Table 4 summarizes the distribution of ESBL-encoding resistance genes and phylogenetic marker genes among *E. coli* isolates, classified by four phylogenetic groups: A1 (n = 39), B1 (n = 19), B2 (n = 22), and D (n = 58).

Table 4: Distribution of Resistance Genes and Phylogenetic Markers by *E. coli* Phylogenetic Group

Gene / Marker	Group A1 (n = 39)	Group B1 (n = 19)	Group B2 (n = 22)	Group D (n = 58)	p-value
ESBL (%)	46.2	68.4	59.1	58.6	0.397
blaTEM (%)	46.2	84.2	63.6	34.5	0.001
blaSHV (%)	5.1	26.3	4.5	10.3	0.090
blaTEM/SHV (%)	48.7	84.2	68.2	41.4	0.005
blaCTX-M-1 (%)	53.8	21.1	13.6	24.1	0.002
blaCTX-M-2 (%)	17.9	21.1	18.2	31.0	0.413
blaCTX-M-9 (%)	5.1	0.0	4.5	5.2	0.940
CTX-M group (%)	69.2	42.1	36.4	48.3	0.049
chuA (%)	0.0	0.0	100	100	<0.001
yjaA (%)	100	15.8	100	0.0	<0.001
TspE4C2 (%)	0.0	100	45.5	32.8	<0.001
p-value	<0.001	<0.001	<0.001	<0.001	—

Although the overall frequency of ESBL production was comparable across groups (p = 0.397), the prevalence of specific *β*-lactamase genes varied significantly. The *blaTEM* gene was most commonly detected in groups B1 (84.2%) and B2 (63.6%), and significantly less frequent in group D (34.5%) (p = 0.001). The *blaSHV* gene was generally uncommon but more frequently observed in group B1 (26.3%) than others, although the difference was not statistically significant (p = 0.090). The combined presence of *blaTEM/blaSHV* was significantly more common in groups B1 (84.2%) and B2 (68.2%) than in A1 (48.7%) or D (41.4%) (p = 0.005).

Among the *CTX-M* gene family, *blaCTX-M-1* was significantly enriched in group A1 (53.8%), compared to lower frequencies in B1 (21.1%), B2 (13.6%), and D (24.1%) (p = 0.002). In contrast, *blaCTX-M-2* and *blaCTX-M-9* were detected at low and relatively uniform rates across all groups, with no significant differences (p = 0.413 and p = 0.940). When considering all CTX-M variants collectively, group A1 had the highest overall frequency (69.2%) (p = 0.049).

In terms of phylogenetic marker genes, highly specific distributions were observed, consistent with group-defining characteristics. The *chuA* gene was detected exclusively in groups B2 and D (100% in both; p < 0.001), reinforcing their classification as extraintestinal pathogenic *E. coli* (ExPEC) lineages. The *yjaA* gene was present in 100% of isolates from groups A1 and B2 but absent in group D and rare in group B1 (15.8%) (p < 0.001). *TspE4C2* was universally detected in group B1 (100%), moderately present in B2 (45.5%) and D (32.8%), and absent in A1 (p < 0.001). All within-group distributions were statistically significant (p < 0.001), highlighting strong genetic divergence across phylogroups.

These results emphasize the existence of distinct resistance gene patterns and phylogenetic signatures among *E. coli* lineages. The predominance of *blaTEM* in B1 and B2, and *blaCTX-M-1* in A1, suggests lineage-specific acquisition and maintenance of ESBL determinants. The alignment of phylogenetic marker profiles with resistance gene patterns further supports the use of molecular phylogrouping as a tool for epidemiological surveillance and risk assessment. Group D, despite its relatively lower carriage of *blaTEM*, exhibited the highest PDR rate, indicating that resistance burden cannot be explained by a single gene alone but rather a complex genomic context.

The high carriage rate of ESBL-producing *E. coli* among healthy individuals in rural Thai Binh underscores a significant epidemiological shift in antimicrobial resistance within Vietnam. Rather than being confined to hospital settings, these resistant organisms are now firmly established in the community. This trend is likely driven by widespread over-the-counter antibiotic use, inadequate sanitation infrastructure, and the routine use of antimicrobials in agriculture and animal husbandry. The presence of such strains in asymptomatic individuals suggests that the human gastrointestinal tract may serve as a silent reservoir, facilitating ongoing environmental dissemination and inter-host transmission.

The predominance of phylogenetic group D among isolates, particularly those recovered from extraintestinal specimens such as pus and sputum, aligns with its known role in extraintestinal pathogenic *E. coli* (ExPEC). Group D strains possess enhanced virulence characteristics that enable them to colonize mucosal surfaces and invade host tissues.[10] In this study, the statistically significant association between group D and clinical specimens (p = 0.020), especially pus (p = 0.031) and sputum (p = 0.013), reinforces the hypothesis that this lineage exhibits dual functionality—as both a silent colonizer and an opportunistic pathogen—thus contributing to community-based transmission and clinical infection.

Although group B2 is also recognized for its involvement in invasive infections, its relatively lower prevalence in this study may reflect local ecological or host-specific factors. Notably, B2 strains retained strong associations with blood and respiratory specimens, consistent with prior studies. In contrast, group B1—commonly regarded as commensal—demonstrated the highest rate of multidrug resistance (52.6%), challenging traditional assumptions about its clinical relevance. This suggests that horizontal gene transfer, particularly via mobile genetic elements such as plasmids, plays a major role in the dissemination of resistance traits irrespective of inherent virulence levels.

The antibiotic resistance profiles observed further reflect the selective pressure exerted by both clinical and non-clinical antibiotic use. Nearly all isolates exhibited resistance to ampicillin and third-generation cephalosporins, while over 70% were resistant to ciprofloxacin—one of the *β*most commonly used fluoroquinolones. These trends support growing concerns about the widespread misuse of antibiotics in human medicine and livestock management.[7, 14] Although amikacin remained effective against most isolates (97.1% susceptibility), the observed gentamicin resistance (34.8%) highlights the risk of diminishing treatment options in the near future.

Molecular characterization provided deeper insights into the genetic context of resistance. The predominance of *blaTEM* in groups B1 and B2 suggests long-term integration of this gene within these lineages. In

contrast, the high prevalence of *blaCTX-M-1* in group A1 (53.8%) indicates recent acquisition, likely via plasmid-mediated transfer mechanisms. The co-detection of multiple *β*-lactamase genes in single isolates may enhance redundancy and phenotypic expression, complicating detection and contributing to treatment failure. These findings corroborate prior studies highlighting the importance of gene cassettes and integrons in sustaining resistance.[2, 15]

Phylogenetic marker gene analysis further confirmed the identity and evolutionary background of the isolates. The exclusive detection of *chuA* in groups B2 and D, both associated with ExPEC, and *yjaA* in groups A1 and B2, aligned well with canonical classification. Similarly, *TspE4C2* was a defining marker for group B1. These distinct gene signatures validated the PCR-based phylogrouping approach and support its continued use in resistance surveillance programs.

From a public health perspective, the convergence of multidrug resistance and virulence in community-derived strains—particularly those belonging to group D—presents a considerable threat. Healthy carriers may unknowingly serve as long-term reservoirs, perpetuating environmental contamination and increasing the risk of difficult-to-treat infections in both hospital and household settings. This risk is further amplified in resource-limited areas where antibiotic regulation is minimal and sanitation is suboptimal.

In summary, the high prevalence of ESBL-producing, multidrug-resistant *E. coli*—with a predominance of phylogenetic group D—among healthy individuals in Can Tho highlights an urgent public health concern. The significant correlation between phylogenetic background and resistance phenotype emphasizes the importance of integrating molecular epidemiology into AMR monitoring systems. Future studies should expand the sample scope and explore longitudinal dynamics of colonization to better inform targeted interventions.

Conclusion

This study highlights the high prevalence of ESBL-producing *E. coli* among healthy individuals and the significant role of phylogenetic group D in extraintestinal infections. Resistance to commonly used antibiotics, including *β*-lactams and fluoroquinolones, was widespread, while aminoglycosides remained **more** effective. Distinct resistance profiles and gene distributions across phylogenetic groups suggest lineage-specific reservoirs of resistance. These findings underscore the need for enhanced community-based surveillance and rational antibiotic use to limit the spread of resistant *E. coli* strains. Future research should focus on expanding sample size and geographic coverage, incorporating longitudinal surveillance and genomic analyses to track the evolution and dissemination of resistance genes in both community and clinical settings.

Conflict of interest

The author`s declare no conflict of interest.

Authors' Declaration

The authors hereby declare that the work presented in this article is original and that any liability for claims relating to the content of this article will be borne by them.

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