

Analysis of Antimicrobial Resistance Patterns in *Escherichia coli* And Molecular Characterization Across Karachi's Different Districts

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ABSTRACT

Background: Antimicrobial resistance (AMR) among *Escherichia coli* is an increasing clinical and public-health concern, particularly in settings where antimicrobial use and resistance surveillance remain challenging. Local phenotypic and molecular data are important for guiding empirical treatment and antimicrobial stewardship.

Objective: To characterize antimicrobial resistance patterns among clinical *E. coli* isolates from Karachi, Pakistan, and detect selected antimicrobial-resistance genes using polymerase chain reaction (PCR).

Methods: A laboratory-based observational study was conducted from January to December 2025 using 177 non-duplicate clinical *E. coli* isolates recovered from urine (n=145), pus (n=21), and blood (n=11) specimens. Antimicrobial susceptibility was determined using the Kirby-Bauer disk diffusion method and interpreted according to applicable Clinical and Laboratory Standards Institute criteria. Resistance patterns were analyzed according to specimen type, age group, sex, and residential district. Selected isolates were subjected to PCR targeting *tetA*, *sul1*, and *sul2*, with amplicons of 502, 430, and 190 bp, respectively.

Results: Among 2,210 antimicrobial susceptibility test results, resistance was highest to cefuroxime (89.3%), ampicillin (85.3%), and ciprofloxacin (76.0%). Resistance was comparatively low to fosfomycin (2.0%), meropenem (4.7%), and nitrofurantoin (6.7%), while no resistance was detected against amikacin or polymyxin B/colistin. Pus isolates demonstrated high resistance to ampicillin and ceftazidime (90.5% each), while blood isolates showed substantial resistance to ampicillin (81.8%). District-level differences were not statistically significant ($\chi^2=0.49$, $p=0.92$). PCR detected *tetA*, *sul1*, and *sul2* resistance determinants.

Conclusion: Clinical *E. coli* isolates in Karachi demonstrated substantial resistance to commonly used antibiotics, while selected agents retained comparatively good activity. Continued local surveillance integrating phenotypic and molecular approaches is warranted to support antimicrobial stewardship and evidence-based empirical therapy.

Keywords: Antimicrobial Resistance, *Escherichia coli*, Karachi, Multidrug Resistance, Clinical Microbiology, Public Health.

INTRODUCTION

Antimicrobial resistance (AMR) has emerged as one of the most serious public health challenges of the twenty-first century.^{1,2} It threatens the effective treatment and prevention of bacterial infections and undermines major advances in modern medicine.¹ The widespread and often inappropriate use of antimicrobial agents in human medicine, veterinary practice, and food-animal production has accelerated the emergence and dissemination of resistant bacterial strains.^{1,3,4} Additional factors, including inappropriate antibiotic access, self-medication, inadequate adherence to treatment regimens, and environmental dissemination, further contribute to the development and spread of resistance.^{2,5} Consequently, AMR is associated with increased

morbidity and mortality, prolonged healthcare utilization, treatment failure, and substantial economic costs worldwide.^{6,7} Moreover, antimicrobial resistance threatens the safety of critical medical interventions, including surgery, organ transplantation, and cancer chemotherapy, which depend on effective antimicrobial therapy.^{1,7}

Among bacterial pathogens, *Escherichia coli* (*E. coli*) is particularly important in AMR research because of its widespread distribution, diverse pathogenic mechanisms, and substantial clinical significance.^{8,9} *E. coli* is a Gram-negative bacterium that commonly inhabits the human gastrointestinal tract as part of the normal intestinal microbiota.⁸ Although many strains are commensal, pathogenic variants can cause urinary tract infections, bloodstream infections, neonatal meningitis, intra-



abdominal infections, and gastrointestinal diseases.^{8,9} The emergence of multidrug-resistant (MDR) *E. coli*, including extended-spectrum β -lactamase (ESBL)-producing and carbapenem-resistant strains, has become a major global concern because resistance can substantially reduce therapeutic options and increase the risk of treatment failure.^{10,11}

The burden of antimicrobial-resistant *E. coli* is particularly concerning in low- and middle-income countries, where inappropriate antimicrobial use, inadequate stewardship, limited diagnostic capacity, and weaknesses in infection prevention and control can facilitate the selection and dissemination of resistant organisms.^{6,12} The global spread of resistant organisms and the associated clinical and economic consequences highlight the importance of systematic surveillance and locally relevant antimicrobial-resistance data.^{6,13} Resistance patterns may vary according to geographic location, healthcare setting, patient population, antimicrobial prescribing practices, and circulating bacterial lineages, emphasizing the need for context-specific surveillance.^{10,14} Such data are essential for guiding antimicrobial stewardship, informing empirical treatment decisions, and strengthening infection-prevention strategies.^{12,15}

Pakistan represents an important setting for antimicrobial-resistance surveillance because the effective implementation of antimicrobial stewardship, infection prevention, and resistance monitoring remains essential for controlling the spread of resistant organisms.^{12,13} Local surveillance is particularly valuable because resistance patterns cannot necessarily be extrapolated from international or even national datasets to individual healthcare settings.^{13,14} Karachi therefore provides a relevant setting for investigating antimicrobial-resistance patterns among clinical *E. coli* isolates and for generating locally applicable evidence. Such surveillance can contribute to evidence-based empirical treatment, antimicrobial stewardship, and public health strategies aimed at limiting the emergence and dissemination of resistant organisms.^{12,15}

Molecular approaches have become increasingly valuable for characterizing the genetic mechanisms underlying antimicrobial resistance.¹¹ Polymerase chain reaction (PCR)-based methods can facilitate the detection of specific resistance determinants, including genes encoding β -lactamases and carbapenemases, and can complement phenotypic antimicrobial susceptibility testing.^{10,11} Molecular characterization can therefore provide additional insight into resistance mechanisms and support epidemiological surveillance of multidrug-resistant organisms.^{11,14}

Therefore, the present study aims to investigate antimicrobial resistance patterns among *E. coli* isolates obtained from clinical samples across Karachi and to detect associated resistance genes using PCR and gel electrophoresis. By integrating phenotypic antimicrobial susceptibility testing with molecular characterization, this study seeks to generate comprehensive local evidence that can support antimicrobial stewardship, improve empirical clinical decision-making, and strengthen public health interventions aimed at controlling antimicrobial resistance in Pakistan.

METHODOLOGY

A laboratory-based observational study was conducted to characterize antimicrobial resistance among clinical *Escherichia coli* isolates obtained from patients in Karachi, Pakistan, between January and December 2025. Clinical specimens were processed in collaboration with participating healthcare and diagnostic laboratories. A total of 177 non-duplicate *E. coli* isolates were included, comprising 145 isolates from urine specimens, 21 from pus specimens, and 11 from blood specimens. Duplicate isolates

obtained from the same patient were excluded to minimize overrepresentation. Patient-related information, including age, sex, and residential district, was recorded where available. Age was categorized into 0–18, 19–40, 41–60, and ≥ 61 years, while geographical analysis included the North, East, South, and West districts of Karachi.

Clinical specimens were processed using standard microbiological procedures according to the participating laboratories' validated standard operating protocols. Specimens were cultured on appropriate routine bacteriological media and incubated under recommended conditions. Suspected *E. coli* colonies were identified based on characteristic colony morphology and conventional biochemical characteristics and/or validated laboratory identification procedures. Confirmed isolates were subsequently subjected to antimicrobial susceptibility testing. Antimicrobial susceptibility was determined using the Kirby–Bauer disk diffusion method, and inhibition zones were interpreted according to applicable Clinical and Laboratory Standards Institute (CLSI) criteria. The antimicrobial panel included commonly used β -lactams, fluoroquinolones, aminoglycosides, carbapenems, urinary antimicrobials, and polymyxins. Urine isolates were tested against cefuroxime, ampicillin, ciprofloxacin, amikacin, polymyxin B/colistin, fosfomycin, nitrofurantoin, and meropenem. Pus isolates were tested against ampicillin, ceftazidime, ceftriaxone, cefixime, ciprofloxacin, meropenem, polymyxin B/colistin, amikacin, and chloramphenicol, whereas blood isolates were tested against ampicillin, ceftazidime, cefixime, ceftriaxone, polymyxin B, amikacin, meropenem, and chloramphenicol. Results were categorized as susceptible, intermediate, or resistant, and resistance proportions were calculated for each antimicrobial and specimen category. Overall, 2,210 antimicrobial susceptibility test results were evaluated.

For molecular characterization, selected confirmed *E. coli* isolates were subjected to genomic DNA extraction using a validated extraction procedure. PCR assays were performed using gene-specific primers targeting *tetA*, *sul1*, and *sul2*. Expected amplicon sizes were 502 bp, 430 bp, and 190 bp, respectively. PCR products were separated by agarose gel electrophoresis alongside a 100-bp DNA molecular-weight ladder. Positive and negative controls were included to verify amplification specificity and exclude contamination. A resistance gene was considered detected when a band corresponding to the expected molecular size was observed.

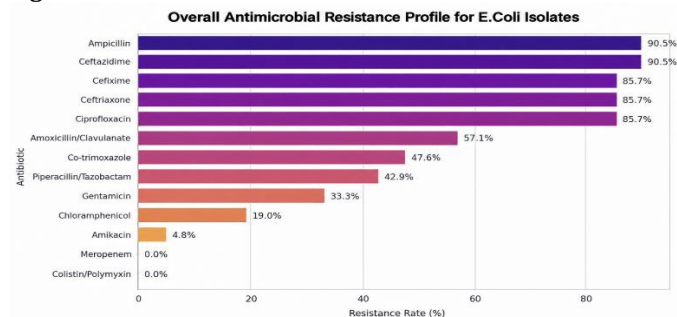
Data were summarized using frequencies, percentages, and resistance proportions. Descriptive comparisons were performed across specimen type, age group, sex, and residential district. Associations between categorical demographic/geographical variables and antimicrobial resistance were assessed using the chi-square test of independence. Statistical significance was set at $p < 0.05$. The overall association analysis yielded $\chi^2 = 0.49$ and $p = 0.92$. Statistical analyses were performed using SPSS version 25.0.

RESULTS

A total of 177 clinical *E. coli* isolates and 2,210 antimicrobial susceptibility test results were evaluated. The overall antimicrobial resistance profile demonstrated substantial resistance to several commonly used antibiotics. The highest resistance was recorded against ampicillin (90.5%) and ceftazidime (90.5%), followed by cefixime, ceftriaxone, and ciprofloxacin (85.7% each). Resistance to amoxicillin/clavulanate was 57.1%, while co-trimoxazole and piperacillin/tazobactam showed resistance rates of 47.6% and 42.9%, respectively. Resistance to gentamicin (33.3%) and chloramphenicol (19.0%) was comparatively lower. Amikacin showed 4.8% resistance,

whereas meropenem and colistin/polymyxin showed no resistance (0.0%) among the tested isolates. (Figure 1)

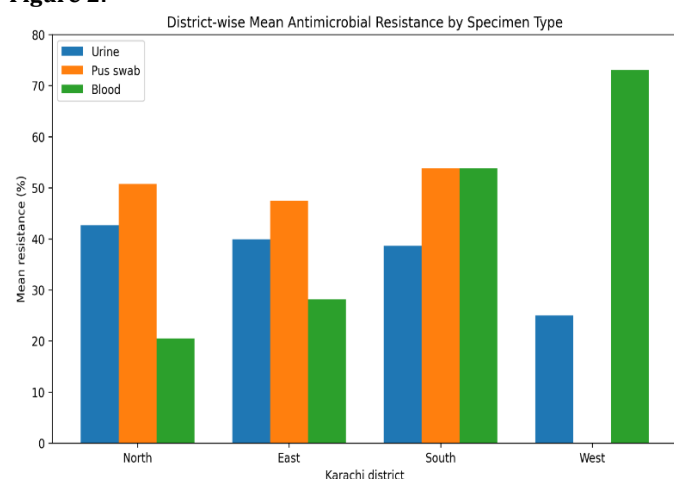
Figure 1:



Specimen- and District-specific Resistance

Resistance patterns varied according to both specimen type and geographical location. Among urine isolates, the highest mean resistance was observed in the **North district (42.7%)**, followed by East (39.9%) and South (38.6%), whereas West demonstrated the lowest mean resistance (25.0%). Pus swab isolates showed comparatively higher resistance, with the highest mean resistance in **South (53.8%)**, followed by North (50.8%) and East (47.4%). Blood isolates demonstrated the greatest geographical variation, with resistance reaching **73.1% in West** and **53.8% in South**, compared with 28.2% in East and 20.5% in North (Figure 2).

Figure 2:

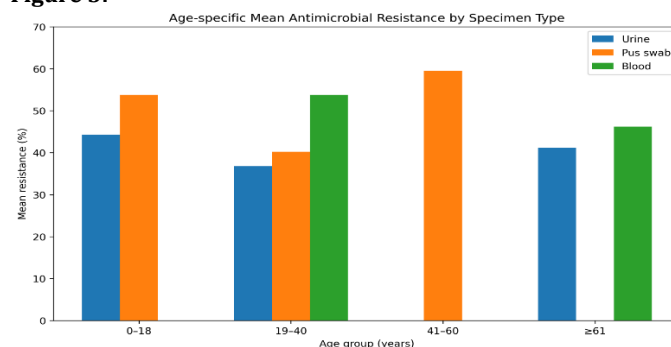


The antimicrobial profile of pus swab isolates was particularly notable, with **90.5% resistance to both ampicillin and ceftazidime**, and **85.7% resistance to ceftriaxone, cefixime, and ciprofloxacin**. Conversely, meropenem and polymyxin B/colistin showed 0% resistance, while amikacin demonstrated 4.8% resistance. Blood isolates also showed substantial resistance to ampicillin (81.8%), ceftazidime (72.7%), cefixime (63.6%), and ceftriaxone (63.6%).

Age-specific Resistance Patterns

Age-stratified analysis demonstrated heterogeneous resistance patterns across specimen types. For urine isolates, mean resistance was highest among patients aged **0–18 years (44.3%)**, followed by those aged ≥ 61 years (41.2%), while the 19–40-year group had the lowest resistance (36.8%). In pus swabs, resistance was highest among patients aged **41–60 years (59.6%)**, followed by those aged 0–18 years (53.8%), whereas the 19–40-year group showed 40.2% resistance. Among blood isolates, the highest mean resistance occurred in the **19–40-year group (53.8%)**, followed by patients aged ≥ 61 years (46.2%) (Figure 3).

Figure 3:



Statistical Association

Despite the observed geographical variation, the Chi-square test of independence showed no statistically significant association between district/social class and antimicrobial resistance ($\chi^2 = 0.49$, $p = 0.92$). Thus, the differences observed between districts did not reach statistical significance in the analyzed dataset.

Molecular Characterization of Resistance

PCR analysis identified three antimicrobial resistance-associated genes: tetA, sul1, and sul2. The corresponding amplicons were detected at the expected sizes of 502 bp, 430 bp, and 190 bp, respectively. Amplification was confirmed using positive controls and a 100-bp DNA ladder, supporting the successful detection of these resistance determinants (Figure 4, 5).

Figure 4:

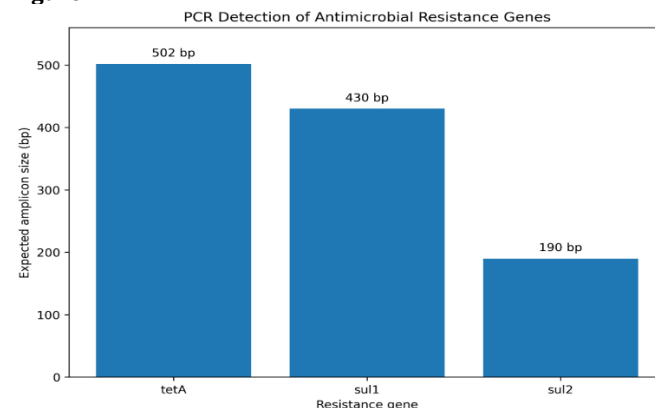
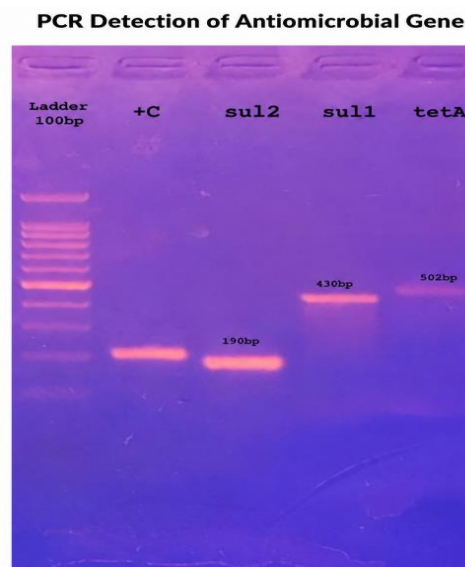


Figure 5:



DISCUSSION

The present study demonstrates a substantial burden of antimicrobial resistance among *Escherichia coli* isolates obtained from clinical specimens across Karachi. The overall resistance profile was characterized by particularly high resistance to several commonly used β -lactam antibiotics and fluoroquinolones, whereas lower resistance was observed against fosfomycin, nitrofurantoin, amikacin, and meropenem. These findings are consistent with the broader global concern that increasing antimicrobial resistance is progressively narrowing effective therapeutic options and complicating the empirical management of bacterial infections.^{1,16,22}

The high resistance observed against cefuroxime (89.3%), ampicillin (85.3%), and ciprofloxacin (76.0%) in urine-derived isolates is clinically important because these agents have historically been widely used for the treatment of urinary tract infections (UTIs). High resistance to fluoroquinolones and β -lactam agents has been reported internationally and is increasingly recognized as an important challenge in the treatment of *E. coli* infections.^{16,23} The observed pattern suggests that empirical treatment based solely on commonly used oral agents may be unreliable in this study population and reinforces the importance of culture-directed therapy and local antimicrobial-resistance surveillance.

The comparatively low resistance to fosfomycin (2.0%) and nitrofurantoin (6.7%) is an important finding for urinary isolates. Both agents retain activity against many uropathogenic *E. coli* strains and are widely recognized as useful therapeutic options for uncomplicated lower UTIs when clinically appropriate.^{15,18} However, susceptibility findings from a single laboratory population should not automatically be interpreted as evidence that these agents are universally suitable as first-line therapy, because treatment selection should also consider infection site, patient characteristics, susceptibility testing standards, and clinical guidelines.¹⁵

Meropenem showed relatively low resistance in urine (4.7%), pus (0%), and blood isolates (9.1%), while amikacin also demonstrated comparatively low resistance. These findings indicate preservation of activity against the tested isolates, but carbapenems and other reserve agents should be protected through antimicrobial stewardship because excessive use can promote selection and dissemination of carbapenem-resistant organisms.^{10,11,19} The absence of detected resistance in a particular subgroup should therefore be interpreted cautiously, particularly where the number of isolates is small.

The resistance profile of pus isolates was particularly concerning, with ampicillin and ceftazidime showing 90.5% resistance and ceftriaxone, cefixime, and ciprofloxacin each showing 85.7% resistance. Such extensive resistance among isolates associated with skin and soft-tissue infections substantially limits conventional antimicrobial choices and may increase the need for microbiological investigation and susceptibility-guided treatment. Antimicrobial resistance in clinical infections is associated with poorer treatment outcomes, increased healthcare utilization, and greater economic burden, particularly where diagnostic and therapeutic resources are constrained.^{6,25}

The relatively preserved activity of meropenem and colistin/polymyxin against pus isolates, together with the low resistance observed against amikacin, is noteworthy. Nevertheless, these findings should not be interpreted as justification for routine use of last-resort agents. Colistin, in particular, is generally reserved for infections caused by organisms resistant to safer and more routinely recommended alternatives, and susceptibility results should be interpreted using appropriate laboratory standards.^{10,20} The relatively low

resistance observed in this study therefore supports continued surveillance rather than unrestricted use of reserve antibiotics.

Blood isolates demonstrated high resistance to ampicillin (81.8%), ceftazidime (72.7%), cefixime (63.6%), and ceftriaxone (63.6%). Because bloodstream infections require prompt effective antimicrobial therapy, such resistance patterns have important clinical implications. Resistance to third-generation cephalosporins may be compatible with the presence of ESBL-producing organisms; however, phenotypic resistance alone cannot establish ESBL production unless appropriate confirmatory testing was performed.^{10,17} Therefore, the present findings should be interpreted as evidence of substantial β -lactam resistance rather than definitive evidence of ESBL production.

The relatively low resistance to meropenem (9.1%) and amikacin (9.1%) among blood isolates suggests that these agents retained considerable in-vitro activity during the study period. However, the blood-isolate subgroup appears substantially smaller than the urine and pus groups, as indicated by the 9.1% increments, and the estimates may therefore be statistically unstable. Consequently, these findings should be considered preliminary and should be validated using larger numbers of bloodstream isolates before informing empirical treatment protocols.

Gender-based analysis showed relatively similar overall resistance levels in urine isolates, with females demonstrating a modestly higher average resistance than males. In contrast, resistance was higher among males in the blood-isolate analysis. These differences should not be interpreted as evidence of a direct biological effect of sex because the study did not establish the underlying mechanisms or adjust for potential confounding factors such as previous antibiotic exposure, hospitalization, comorbidities, infection severity, or healthcare-associated acquisition. Previous antimicrobial exposure and healthcare-related factors are recognized contributors to the selection and dissemination of resistant organisms.^{1,22}

Age-specific analysis also demonstrated variation in resistance across patient groups. Among urine isolates, the highest average resistance was observed in patients aged 0–18 years, whereas among blood isolates the highest average resistance occurred in the 19–40-year group. Pus isolates showed the highest resistance among adults aged 41–60 years. These differences may reflect variations in antimicrobial exposure, healthcare utilization, underlying disease, infection type, and the epidemiological characteristics of the sampled populations; however, these potential explanations were not directly evaluated in the present study. Such heterogeneity reinforces the importance of interpreting antimicrobial-resistance patterns within specific clinical and demographic contexts rather than assuming a uniform resistance profile across all patient groups.^{13,14}

Geographical analysis revealed considerable descriptive variation among Karachi districts. For urine isolates, the highest average resistance was observed in North district (42.7%), whereas West district had the lowest observed value (25.0%). For pus isolates, South district demonstrated the highest resistance (53.8%), while blood isolates showed the highest resistance in West district (73.1%). These differences are epidemiologically noteworthy because antimicrobial-resistance patterns can vary according to healthcare setting, antimicrobial exposure, patient characteristics, and circulating bacterial populations.^{10,14} However, the observed district-level differences should not be interpreted as evidence that a particular district caused or independently determined resistance.

Importantly, the chi-square analysis did not demonstrate a statistically significant association between district/social class and AMR ($\chi^2 = 0.49$, $p = 0.92$). Thus, although descriptive resistance percentages differed between districts, the available statistical evidence does not support a significant relationship

between the investigated geographical/social variables and resistance status. This distinction between descriptive variation and statistical association is important and prevents overinterpretation of apparent geographical differences. Larger, multicenter studies with stratification by specimen type, healthcare facility, antibiotic exposure, and patient characteristics would be useful to determine whether the observed geographical patterns are reproducible.

The molecular findings provide additional evidence that resistance in the studied *E. coli* isolates was associated with recognizable genetic determinants. Conventional PCR detected tetA at 502 bp, sul1 at 430 bp, and sul2 at 190 bp, with amplification confirmed by the expected bands, positive controls, and a 100-bp DNA ladder. The detection of these genes is consistent with established mechanisms of antimicrobial resistance involving altered tetracycline susceptibility and sulfonamide resistance.¹¹ Molecular characterization can complement phenotypic antimicrobial susceptibility testing by providing information about specific resistance determinants that cannot be obtained from susceptibility testing alone.^{11, 14}

The coexistence of substantial phenotypic resistance with detectable resistance genes highlights the value of integrating microbiological and molecular approaches in AMR surveillance. Nevertheless, detection of a resistance gene should not automatically be equated with phenotypic resistance to a particular antimicrobial in every isolate, because gene expression, genetic context, regulatory mechanisms, and additional resistance determinants may influence the observed phenotype.¹¹ Molecular findings should therefore be interpreted alongside antimicrobial susceptibility results rather than independently.

The findings of this study have several implications for antimicrobial stewardship in Karachi. The high resistance observed against commonly used antibiotics indicates that empirical treatment strategies should be informed by current local susceptibility data rather than relying exclusively on historical prescribing practices. Surveillance systems that combine phenotypic susceptibility testing with molecular characterization can provide more informative evidence for antimicrobial stewardship, infection-control planning, and empirical treatment guidelines.^{12, 16, 19}

The findings are also consistent with the broader international concern regarding increasing antimicrobial consumption and the selection of resistant organisms. Global increases in antimicrobial consumption, including use in food-producing animals, have been identified as important contributors to the wider AMR problem.^{26, 27} Although the present study did not investigate antibiotic consumption, prescribing behavior, or environmental reservoirs, the high resistance levels observed in clinical isolates emphasize the importance of addressing antimicrobial use through coordinated stewardship, prescription control, surveillance, and infection-prevention measures.^{19, 21, 22}

Several limitations should be considered when interpreting these findings. First, the study was based on clinical isolates obtained from selected laboratory and healthcare settings in Karachi and may not represent the entire city or the wider Pakistani population. Second, the number of isolates differed substantially between specimen types, and the relatively small blood-isolate subgroup limits the precision of its resistance estimates. Third, the observational nature of the analysis prevents causal conclusions regarding the effects of gender, age, district, socioeconomic factors, or presumed antibiotic exposure on resistance. Fourth, although PCR identified tetA, sul1, and sul2, the molecular analysis does not by itself establish the complete resistome or explain all phenotypic resistance observed in the isolates. Finally, the study did not report detailed information on

previous antimicrobial exposure, hospitalization status, comorbidities, infection severity, or healthcare-associated versus community-acquired infection, all of which could influence resistance patterns.

Despite these limitations, the study provides useful local evidence of substantial antimicrobial resistance among clinical *E. coli* isolates from Karachi and demonstrates the value of combining phenotypic susceptibility testing with molecular detection of resistance determinants. The particularly high resistance observed against several commonly used β -lactams and fluoroquinolones, together with the relatively preserved activity of selected agents, underscores the need for continuous local surveillance. Future studies should include larger multicenter populations, standardized antimicrobial susceptibility testing, detailed patient-level risk factors, confirmatory ESBL and carbapenemase testing where indicated, and broader molecular characterization of resistance determinants.

Overall, the findings indicate that antimicrobial resistance among clinical *E. coli* isolates in Karachi represents an important public-health and clinical concern. The absence of a statistically significant district/social-class association despite substantial descriptive variation suggests that resistance should be addressed as a broad surveillance and antimicrobial-stewardship priority rather than being attributed to a single geographical or socioeconomic group. Continued culture-guided therapy, rational antibiotic prescribing, molecular surveillance, infection-prevention measures, and coordinated antimicrobial-stewardship interventions are warranted to limit further dissemination of resistant *E. coli* in Pakistan.^{12, 19, 24}

CONCLUSION

This study demonstrates a substantial burden of antimicrobial resistance among clinical *Escherichia coli* isolates in Karachi, particularly against cefuroxime, ampicillin, and ciprofloxacin. In contrast, fosfomycin, nitrofurantoin, meropenem, amikacin, and polymyxin B/colistin retained comparatively greater activity. Although resistance varied across specimen types, age groups, and residential districts, no statistically significant association was identified with district or social class. Detection of tetA, sul1, and sul2 further indicates the presence of important antimicrobial-resistance determinants. These findings underscore the need for strengthened antimicrobial stewardship, routine susceptibility-guided therapy, and continuous phenotypic and molecular surveillance to monitor emerging resistance and support evidence-based infection-control strategies in Karachi.

Ethical Approval

Ethical approval was not required for this study because it involved the analysis of antimicrobial resistance patterns and molecular characterization using previously collected data and did not involve direct intervention with human participants.

Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to applicable institutional and ethical restrictions

Author Contributions

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Writing – Review & Editing: Muhammad Owais Sarfaraz, Syeda Sadaf Akber, Farhan Essa, Muhammad Uneeb
All authors reviewed and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Informed Consent

Informed consent was not applicable to this study because it did not involve direct participation of human subjects or the collection of personally identifiable information.

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Conflict of Interest

The authors declare that they have no competing interests.

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Declaration of AI-Assisted Language Editing

Grammarly was used to correct language.

REFERENCES

- Holmes AH, Moore LSP, Sundsfjord A, Steinbakk M, Regmi S, Karkey A, et al. Understanding the mechanisms and drivers of antimicrobial resistance. *Lancet*. 2016;387(10014):176-87. doi:10.1016/S0140-6736(15)00473-0.
- Kaper JB, Nataro JP, Mobley HLT. Pathogenic *Escherichia coli*. *Nat Rev Microbiol*. 2004;2(2):123-40. doi:10.1038/nrmicro818.
- Nordmann P, Naas T, Poirel L. Global spread of carbapenemase-producing *Enterobacteriaceae*. *Emerg Infect Dis*. 2011;17(10):1791-8. doi:10.3201/eid1710.110655.
- Tenover FC. Mechanisms of antimicrobial resistance in bacteria. *Am J Infect Control*. 2006;34(5 Suppl 1). doi:10.1016/j.ajic.2006.05.219.
- Madigan MT, Bender KS, Buckley DH, Sattley WM, Stahl DA. Brock biology of microorganisms. 16th ed. Pearson; 2021.
- Murray PR, Rosenthal KS, Pfaller MA. Medical microbiology. 9th ed. Elsevier; 2021.
- Willey JM, Sherwood LM, Woolverton CJ. Prescott's microbiology. 10th ed. McGraw-Hill Education; 2017.
- Todar K. Todar's online textbook of bacteriology. Madison (WI): University of Wisconsin-Madison, Department of Bacteriology; 2004-2006. Available from: <https://www.textbookofbacteriology.net>
- Tortora GJ, Funke BR, Case CL. Microbiology: an introduction. 13th ed. Pearson; 2020.
- World Health Organization. Antimicrobial resistance [Internet]. Geneva: World Health Organization; 2023 [cited 2026 Sep 7]. Available from: <https://www.who.int/news-room/fact-sheets/detail/antimicrobial-resistance>
- Croxen MA, Law RJ, Scholz R, Keeney KM, Wlodarska M, Finlay BB. Recent advances in understanding enteric pathogenic *Escherichia coli*. *Clin Microbiol Rev*. 2013;26(4):822-80. doi:10.1128/CMR.00022-13.
- Nataro JP, Kaper JB. Diarrheagenic *Escherichia coli*. *Clin Microbiol Rev*. 1998;11(1):142-201. doi:10.1128/CMR.11.1.142.
- Donnenberg MS, Whittam TS. Pathogenesis and evolution of virulence in enteropathogenic and enterohemorrhagic *Escherichia coli*. *J Clin Invest*. 2001;107(5):539-48. doi:10.1172/JCI12404.
- Wang X, Quinn PJ. Lipopolysaccharide: biosynthetic pathway and structure modification. *Prog Lipid Res*. 2010;49(2):97-107. doi:10.1016/j.plipres.2009.06.002.
- Foxman B. The epidemiology of urinary tract infection. *Nat Rev Urol*. 2010;7(12):653-60. doi:10.1038/nrurol.2010.190.
- World Health Organization. Antimicrobial resistance: global report on surveillance. Geneva: World Health Organization; 2014. Available from: <https://www.who.int/publications/i/item/9789241564748>
- Tarr PI, Gordon CA, Chandler WL. Shiga-toxin-producing *Escherichia coli* and haemolytic uraemic syndrome. *Lancet*. 2005;365(9464):1073-86. doi:10.1016/S0140-6736(05)71144-2.
- Johnson JR, Russo TA. Molecular epidemiology of extraintestinal pathogenic (uropathogenic) *Escherichia coli*. *Int J Med Microbiol*. 2005;295(6-7):383-404. doi:10.1016/j.ijmm.2005.07.005.
- World Health Organization. Global action plan on antimicrobial resistance. Geneva: World Health Organization; 2015. Available from: <https://www.who.int/publications/i/item/9789241509763>
- Centers for Disease Control and Prevention. Antibiotic resistance threats in the United States, 2019. Atlanta (GA): US Department of Health and Human Services, CDC; 2019.
- O'Neill J. Tackling drug-resistant infections globally: final report and recommendations. London: Review on Antimicrobial Resistance; 2016.
- Prestinaci F, Pezzotti P, Pantosti A. Antimicrobial resistance: a global multifaceted phenomenon. *Pathog Glob Health*. 2015;109(7):309-18. doi:10.1179/2047773215Y.0000000030.
- Ventola CL. The antibiotic resistance crisis: part 1: causes and threats. *P T*. 2015;40(4):277-83.
- Laxminarayan R, Sridhar D, Blaser M, Wang M, Woolhouse M. Achieving global targets for antimicrobial resistance. *Science*. 2016;353(6302):874-5. doi:10.1126/science.aaf9286.
- Founou RC, Founou LL, Essack SY. Clinical and economic impact of antibiotic resistance in developing countries: a systematic review and meta-analysis. *PLoS One*. 2017;12(12). doi:10.1371/journal.pone.0189621.
- Van Boeckel TP, Brower C, Gilbert M, Grenfell BT, Levin SA, Robinson TP, et al. Global trends in antimicrobial use in food animals. *Proc Natl Acad Sci U S A*. 2015;112(18):5649-54. doi:10.1073/pnas.1503141112.
- Klein EY, Van Boeckel TP, Martinez EM, Pant S, Gandra S, Levin SA, et al. Global increase and geographic convergence in antibiotic consumption between 2000 and 2015. *Proc Natl Acad Sci U S A*. 2018;115(15). doi:10.1073/pnas.1717295115.