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Short Communication: Characterization of phenotypic and genotypic antibiotic resistance patterns in *Escherichia coli* isolates from bloodstream infections

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Abstract. *Tjampakasari CR, Stevina L, Paramita RI. 2026. Short Communication: Characterization of phenotypic and genotypic antibiotic resistance patterns in* Escherichia coli *isolates from bloodstream infections. Biodiversitas 27 (3): d270320. https://doi.org/10.13057/biodiv/d270320.* Extraintestinal pathogenic *Escherichia coli* (ExPEC) is a leading cause of bloodstream infections (BSIs) worldwide and contributes substantially to the burden of antimicrobial resistance. The aim of this study was to evaluate the concordance between phenotypic antimicrobial susceptibility testing (AST) and genotypic resistance determinants identified by whole-genome sequencing (WGS) in *E. coli* BSI isolates. During the study period, 194 blood culture samples from patients with suspected BSIs were processed, of which 92 yielded positive results; 13 non-duplicate *E. coli* isolates (14.13% of positive cultures) were included in this single-center, exploratory study. Phenotypic resistance profiles were determined using the VITEK®2 Compact system, while WGS was performed to identify antimicrobial resistance genes, sequence types (STs), and resistance-associated mutations. Phenotypic resistance showed that resistance to ampicillin (7/13, 53.8%), ciprofloxacin (6/13, 46.2%), and ceftriaxone (5/13, 38.5%). Genomic analysis revealed β-lactam resistance genes predominantly associated with blaTEM-1B and its variants (blaTEM-116, blaTEM-141, and blaTEM-206), along with blaCTX-M-55; blaTEM-1B was the most frequently detected gene (4/13), followed by blaCTX-M-55 (2/13). Fluoroquinolone resistance was mainly linked to mutations within quinolone resistance-determining regions of *gyrA* and *parC*, as well as alterations affecting the AcrAB-TolC efflux system. Multilocus sequence typing using the Achtman scheme demonstrated high genetic diversity, with 12 distinct STs identified among 13 isolates, including ST73, ST117, ST410, ST95, and ST127. High concordance between phenotypic β-lactam resistance and genotypic findings was observed, as all β-lactam-resistant isolates carried at least one corresponding β-lactamase gene. Overall, these findings indicate that WGS can reliably confirm β-lactam resistance mechanisms and provide useful molecular epidemiological insights into *E. coli* isolates causing BSIs; however, conclusions are limited by the small sample size and single-center design, and larger multicenter studies are warranted to support routine surveillance applications.

Keywords: Antibiotic resistance, bloodstream infection, *Escherichia coli*, genotypic, phenotypic

INTRODUCTION

Bloodstream infections (BSIs) are severe clinical conditions that occur when microorganisms, most commonly bacteria, enter the bloodstream and trigger a systemic inflammatory response. If not promptly and effectively treated, BSIs can rapidly progress to sepsis or septic shock, particularly among immunocompromised patients, individuals with chronic comorbidities, neonates, the elderly, and those undergoing invasive medical procedures (Forde et al. 2023; Korotetskiy et al. 2023). BSIs are associated with high morbidity and mortality rates, prolonged hospitalization, increased need for intensive care, and substantial healthcare costs, placing a significant burden on healthcare systems worldwide (Kwiecinski and Horswill 2020; Beshah et al. 2022).

Bloodstream infection management is increasingly complicated by rising antimicrobial resistance, particularly multidrug-resistant Gram-negative bacteria that restrict treatment options and worsen outcomes. Delayed or inappropriate therapy increases mortality, while invasive procedures such as central venous catheterization, mechanical ventilation, and hemodialysis further elevate BSI risk. These concerns underscore the urgent need for stronger surveillance, prudent antibiotic use, and advanced diagnostics (Schöneweck et al. 2021; Sora 2021; Afza et al. 2022).

Among Gram-negative pathogens, *Escherichia coli* is one of the leading causes of BSIs in both community and hospital settings (Endraswari et al. 2023). Resistance in *E. coli* is mediated through multiple mechanisms, including β-lactamase production, target-site mutations, efflux pump overexpression, and plasmid-mediated resistance genes, all of which compromise the effectiveness of commonly used antimicrobials (Di Franco et al. 2021; Lutwick et al. 2021). Extended-spectrum β-lactamase (ESBL)-producing *E. coli*, particularly those harboring bla_TEM_ and bla_CTX-M_ genes, have been increasingly reported worldwide, conferring resistance to third-generation cephalosporins. Fluoroquinolone resistance is frequently associated with mutations in

quinolone resistance-determining regions (QRDRs) of *gyrA* and *parC*, as well as plasmid-mediated mechanisms such as *aac*(6′)-Ib-cr (Li et al. 2021).

In Indonesia and other Southeast Asian countries, BSIs caused by resistant *E. coli* represent a growing clinical concern. Several studies have reported high rates of resistance to ampicillin, third-generation cephalosporins, and fluoroquinolones among *E. coli* isolates from blood cultures, reflecting widespread antibiotic use and limited routine molecular surveillance (Nelwan et al. 2020). However, most available data in Indonesia are based on phenotypic antimicrobial susceptibility testing, with limited integration of genomic data. Consequently, information on the molecular epidemiology, resistance gene distribution, and sequence type (ST) diversity of *E. coli* causing BSIs at the local hospital level remains scarce.

Globally, many *E. coli* isolates causing extraintestinal infections are categorized as extraintestinal pathogenic *E. coli* (ExPEC), a group often associated with specific phylogenetic backgrounds and virulence traits. Certain lineages, such as ST73, ST95, ST127, and the globally disseminated ST131 clone, have been linked to BSIs and elevated antimicrobial resistance (Avershina et al. 2023). Nevertheless, formal classification of ExPEC typically requires analysis of defined virulence gene profiles, which are not routinely assessed in all clinical genomic studies. Therefore, in the absence of comprehensive virulence gene characterization, it is more appropriate to describe isolates based on their clinical source as *E. coli* isolates from bloodstream infections.

Whole-genome sequencing (WGS) enables high-resolution characterization of bacterial pathogens, including resistance genes, sequence types, and phylogenetic relatedness, beyond conventional phenotypic methods (Bortolaia et al. 2023). When integrated with phenotypic susceptibility testing, WGS helps assess concordance between observed resistance and underlying genetic determinants, supporting its diagnostic and surveillance value (MacKinnon et al. 2021). However, such integrated baseline data for *E. coli* BSI isolates in Indonesia, especially at single-center hospitals, remain scarce. Existing studies are limited in number and often focus on either phenotypic resistance or genomic characterization alone. Moreover, few studies have explored the concordance between phenotypic antimicrobial resistance and WGS-detected resistance determinants in small, hospital-based cohorts.

Therefore, this exploratory, single-center study aimed to evaluate the concordance between phenotypic antimicrobial susceptibility testing and genotypic resistance determinants identified by WGS in *E. coli* isolates recovered from bloodstream infections. In addition, this study describes the distribution of sequence types and phylogenetic relationships within this limited dataset, providing baseline molecular epidemiological data for one hospital setting. Although constrained by a small sample size, the findings contribute preliminary insights into resistance mechanisms and genetic diversity of *E. coli* BSI isolates and may inform future larger-scale surveillance and antimicrobial stewardship efforts.

MATERIALS AND METHODS

Study design, isolate collection, and ethical approval

This study was designed as a retrospective, descriptive laboratory-based study. During the study period (January 2023-January 2024), a total of 194 blood culture samples from suspected bloodstream infection (BSI) cases were processed at the microbiology laboratory of the Department of Microbiology, Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia. The study was conducted at Dr. Cipto Mangunkusumo National Central General Hospital, a tertiary referral hospital in Jakarta, Indonesia. The isolates were obtained from adult patients (≥18 years old). Of these, 92 samples yielded positive blood culture results, and 13 isolates were identified as *Escherichia coli*. These 13 non-duplicate *E. coli* isolates were included in the study. Only the first bloodstream isolate per patient was included to avoid duplication. Given the small number of isolates (n = 13), the study was descriptive in nature.

This study was approved by the Ethics Committee of Dr. Cipto Mangunkusumo National Central General Hospital, Faculty of Medicine, Universitas Indonesia, approval no: Ket.414/UN2.F1/ETIK/PPM.06.02/2024. All procedures complied with ethical standards for research on clinical isolates.

Bacterial rejuvenation and phenotypic identification

Escherichia coli isolates were revived by subculturing onto Nutrient Agar (NA) and Blood Agar (BA) plates and incubated at 37°C for 24-48 hours to obtain pure cultures. Phenotypic identification was performed using selective and differential media, including Eosin Methylene Blue Agar (EMB) and MacConkey Agar (MCA). Colony morphology was assessed, followed by Gram staining to confirm Gram-negative, rod-shaped bacteria. Species identification and antimicrobial susceptibility testing were previously confirmed using the VITEK® 2 Compact automated system (bioMérieux, France).

Antimicrobial susceptibility testing

Antimicrobial susceptibility testing (AST) was performed on all non-duplicated *E. coli* isolates recovered from blood cultures using the VITEK® 2 Compact system with AST GN93 cards, which included antibiotics, such as ampicillin, ampicillin sulbactam, piperacillin-tazobactam, ceftazidim, ceftriaxone, cefepime, aztreonam, ertapenem, meropenem, amikacin, gentamicin, ciprofloxacin, tigecycline, nitrofurantoi and trimethoprim-sulfamethoxazole. Minimum inhibitory concentrations (MICs) were interpreted according to CLSI guidelines (2024) and categorized as susceptible (S), intermediate (I), or resistant (R). Extended-spectrum β-lactamase (ESBL) production was determined based on phenotypic AST results following CLSI criteria.

DNA extraction

Genomic DNA was extracted from fresh bacterial colonies using the QIAamp DNA Mini Kit (Qiagen, Germany) following the manufacturer's protocol for Gram-negative bacteria. Briefly, bacterial pellets were re-suspended in phosphate-buffered saline (PBS), lysed using Proteinase K

and buffer ATL, and incubated at 56°C. DNA purification was performed using silica-based spin columns, followed by washing and elution. DNA quantity and quality were assessed using Qubit fluorometry and NanoDrop spectrophotometry, respectively.

Whole genome sequencing

Whole genome sequencing (WGS) libraries were prepared from purified DNA using the Nextera XT DNA Library Preparation Kit (Illumina) and sequenced on the Illumina NovaSeq 6000 platform (Illumina, USA), generating paired-end reads in FASTQ format.

Bioinformatics analysis

Raw sequencing reads were assessed for quality using FastQC. Reads with a Phred quality score <28 were trimmed using Trimmomatic. High-quality reads were assembled de novo using SPAdes assembler with default parameters. Assemblies were evaluated for quality prior to downstream analyses.

Genotypic characterization

Multilocus sequence typing (MLST) was performed using the Achtman MLST scheme via the Pathogenwatch platform. Sequence types (STs) were assigned based on the allelic profiles of seven housekeeping regulatory genes according to the Achtman scheme. Antimicrobial resistance genes were identified using ResFinder (version 4.1) with a minimum identity threshold of 90% and a minimum coverage of 60%.

Point mutations associated with fluoroquinolone resistance in gyrA and parC genes were identified using PointFinder. Phylogenetic relationships were inferred based on core-genome single nucleotide polymorphism (SNP) analysis using BV-BRC, and phylogenetic trees were visualized with iTOL.

Assessment of phenotype-genotype concordance

The relationship between phenotypic and genotypic antimicrobial resistance was assessed descriptively as concordance, defined as agreement between phenotypic resistance (R) and the presence of corresponding resistance genes or mutations identified by WGS. Discrepancies between phenotypic and genotypic results were recorded and described. No inferential statistical tests were performed due to the limited sample size.

RESULTS AND DISCUSSION

Inoculation and phenotypic testing

A total of 194 blood culture samples from suspected bloodstream infections (BSI) were processed, of which 92 were positive. Thirteen non-duplicate *E. coli* isolates (14.13%) were recovered used in the study. Isolates were cultured on Eosin Methylene Blue (EMB) and MacConkey Agar (MCA), showing metallic green colonies on EMB and pink colonies on MCA, indicative of lactose fermentation. Gram staining results confirmed that bacteria was Gram-negative, short rod-shaped (coccobacilli), non-spore-forming. Identification was further validated using the VITEK®2 Compact system (bioMérieux, France) (Figure 1), and species confirmation was performed by whole-genome sequencing (WGS).

DNA isolation and quality assessment

DNA was successfully isolated from all 13 isolates using the QIAamp DNA Mini Kit, following manufacturer protocols. DNA concentrations measured by Qubit ranged from 150.2 to 316.9 ng/µL, while Nanodrop measurements showed 260/280 ratios between 1.62-1.99, within the acceptable range for downstream WGS. All isolates met quality and quantity thresholds for library preparation (Table 1), ensuring reliable genomic analysis.

Sequence typing and phylogenetic analysis

Multilocus sequence typing (MLST) via Pathogenwatch classified the 13 isolates into 12 STs: ST73, ST117, ST10, ST410, ST83, ST169, ST95, ST1844, ST101, ST457, ST744, ST127. Phylogenetic analysis using BV-BRC and iTOL revealed three major clades, indicating close genetic relatedness among certain isolates (Figure 2). Notably, high-risk ExPEC clones such as ST73, ST95, ST127, and ST410 were identified.

Phenotypic antimicrobial resistance

Phenotypic testing using VITEK-2 revealed the following resistance rates among isolates: ampicillin: 7 of 13 isolates (53.8%), ciprofloxacin: 6 isolates (46.2%), ceftriaxone and aztreonam: 5 isolates respectively (38.5%), ampicillin-sulbactam and gentamicin: 4 isolates respectively (30.8%) and ceftazidime, piperacillin-tazobactam, cefepime, trimethoprim-sulfamethoxazole: 2 isolates respectively (15.4%). Five isolates (38.5%) were ESBL-positive.

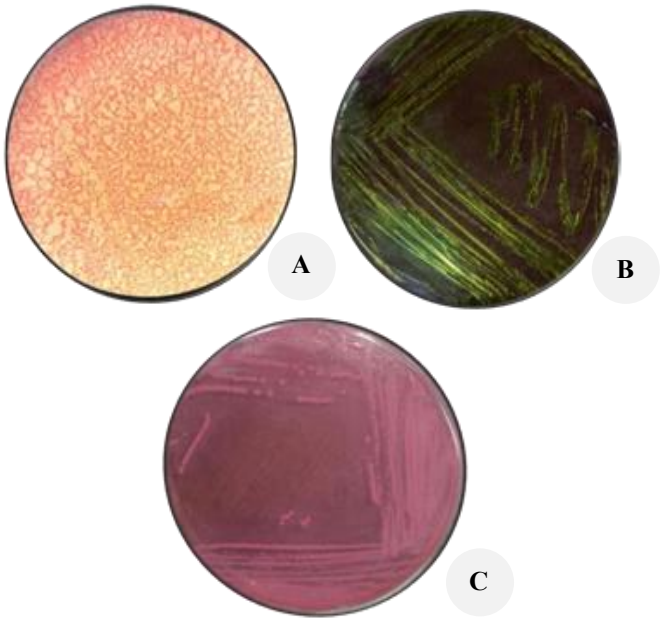

Figure 1. Phenotypic results. A. Gram stain, B. Colonies on EMB agar, C. Colonies on MacConkey agar

Table 1. Quantity and quality of isolated DNA

Isolates	Final volume (µL)	DNA quantity	DNA quality
		Qubit (ng/µL)	Nanodrop A 260/280
EC1-LGC9125	35	202.7	1.74
EC2-LGC9126	37	170.1	1.79
EC3-LGC9127	35	187.8	1.62
EC4-LGC9128	35	168.5	1.68
EC5-LGC9129	35	150.2	1.66
EC6-LGC9130	36	224.2	1,78
EC7-LGC9131	36	220.8	1.78
EC8-LGC9132	37	187.1	1.77
EC9-LGC9133	35	298.5	1.74
EC10-LGC9134	36	204.1	1.99
EC11-LGC9135	36	316.9	2.0
EC12-LGC9136	35	150.8	1.76
EC13-LGC9137	35	162.9	1.62

Genotypic antimicrobial resistance

Analysis of genome assemblies identified 38 resistance genes in all isolates. Key findings include: Beta-lactam resistance: blaTEM-1B, blaTEM-116, blaTEM-141, blaCTX-M-15, blaCTX-M-55, blaOXA-1, blaOXA-320, blaLAP-2. Fluoroquinolone resistance: Mutations in gyrA, parC, parE, and efflux-related genes AcrAB-TolC.

Specific gene variants corresponded to STs, e.g., ST73 carried blaTEM-1, ST1844 carried blaTEM-116, and ST457 carried blaTEM-1B. Resistance mechanisms included beta-lactamase production, target site mutations, and efflux pumps.

Correlation between phenotypic and genotypic resistance

A strong correlation was observed between phenotypic resistance and genotypic data. All beta-lactam-resistant isolates carried corresponding resistance genes. Among ESBL-positive 5 isolates (38.46%), blaTEM-1B and blaCTX-M-15 were consistently detected.

The study demonstrates that phenotypic and genotypic analysis complement each other in understanding antimicrobial resistance in *E. coli* BSIs. Phenotypic testing identified resistance profiles, while WGS provided detailed insights into resistance genes, sequence types, and high-risk ExPEC clones present in the dataset (ST73, ST95, ST127, ST410). ST131 was not detected in any isolate. The phylogenetic relationships among isolates are illustrated in Figure 2, which shows substantially genetic diversity.

Genotypic profiling revealed diverse resistance mechanisms, including beta-lactamase enzymes, target mutations, and efflux pumps, which explained the phenotypic resistance patterns observed. Importantly, concordance analysis confirmed the reliability of WGS for predicting phenotypic resistance, highlighting its potential as a tool for precision antimicrobial therapy and infection control in hospital settings.

Discussion

This study provides an exploratory whole-genome sequencing (WGS)-based characterization of phenotypic and genotypic antimicrobial resistance among *E. coli* BSI isolates in a single-center setting in Indonesia. Although limited by a small sample size, the findings offer preliminary insights into resistance patterns, molecular epidemiology, and genotype-phenotype concordance that are relevant for local antimicrobial surveillance and infection control efforts. The study also demonstrates the feasibility and added value of integrating WGS with routine laboratory diagnostics to improve understanding of invasive *E. coli* infections.

Resistance patterns in the local and regional context

The resistance profiles observed in this study reveal substantial reduced susceptibility to first- and second-line antibiotics commonly used for BSI management, particularly β-lactams and fluoroquinolones (Grøv et al. 2022). Comparable trends have been reported across Southeast Asia and other low- and middle-income regions, where extended-spectrum β-lactamase (ESBL)-producing *E. coli* is increasingly prevalent. Five isolates (38.5%) were ESBL-positive. These findings align with previous studies showing high resistance rates to beta-lactams and Fluoroquinolones in ExPEC isolates (Gambushe et al. 2022; Hapsari et al. 2024).

While this study is not designed to estimate prevalence, the detection of resistance to first- and second-line agents underscores the ongoing clinical challenge of antimicrobial resistance in invasive *E. coli* infections. These preliminary observations should be interpreted cautiously, as they are based on a small, single-center dataset and do not represent population-level prevalence.

Molecular epidemiology and sequence type diversity

WGS revealed substantial genetic heterogeneity among the isolates, with 12 different sequence types (STs) identified across 13 isolates. This diversity indicates the absence of a single dominant clone, consistent with the polyclonal nature of *E. coli* BSI reported in other settings. Several STs detected, including ST73, ST95, ST127, and ST410, are recognized extraintestinal pathogenic *E. coli* (ExPEC) lineages associated with invasive disease globally (Shahbazi et al. 2023).

Importantly, the high-risk clone ST131, frequently reported in global ExPEC outbreaks, was not identified in this collection. This absence may reflect local epidemiological variation or be influenced by the limited sample size. The presence of multiple ExPEC-associated STs nonetheless indicates that diverse pathogenic lineages contribute to bloodstream infections, emphasizing the need for continued genomic surveillance rather than reliance on a single dominant clone (Chmielarczyk et al. 2021; Pokharel et al. 2023).

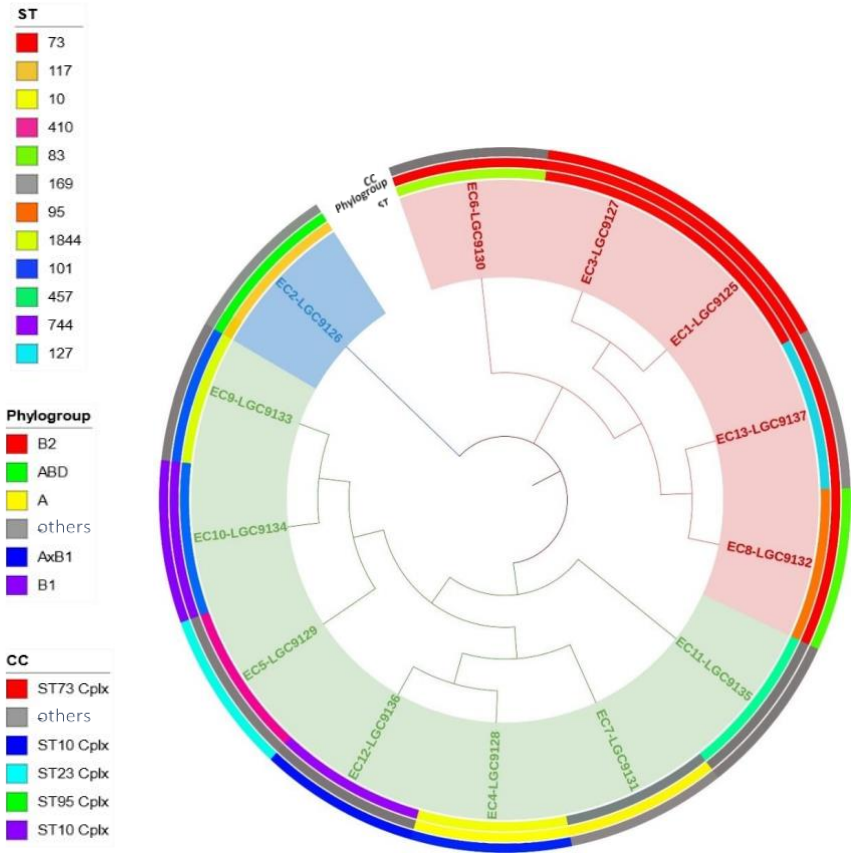

Figure 2. Phylogenetic tree of *Escherichia coli* isolates. ST: Sequence Type, CC: Clonal Complex

Genotype-phenotype concordance for β-lactam resistance

A primary objective of this study was to assess the concordance between phenotypic antimicrobial susceptibility testing (AST) and WGS-derived resistance determinants. For β-lactam antibiotics, a high level of concordance was observed, as all resistant isolates carried one or more corresponding resistance genes, primarily blaTEM and blaCTX-M variants. This observation aligns with global evidence identifying these genes as major contributors to β-lactam and ESBL-mediated resistance. Minor discordance between genotype and phenotype may arise from regulatory mechanisms, gene expression differences, or resistance determinants not captured by current databases. Given the descriptive nature of the study, the findings are best interpreted as concordance rather than statistical correlation (Geurtsen et al. 2022). These results are consistent with studies by Tyson et al. (2015) and Weerdenburg et al. (2023), reporting high concordance between WGS-predicted and phenotypic profile.

Fluoroquinolone resistance mechanisms and clinical relevance

Fluoroquinolone resistance was primarily mediated by mutations in quinolone resistance-determining regions of gyrA and parC, which modify DNA gyrase and topoisomerase IV, reducing drug binding. These mutations are clinically significant, as fluoroquinolones are commonly used empirically or as step-down therapy for invasive *E. coli* infections. Detection of such mutations highlights the potential for WGS to identify high-risk resistance mechanisms prior to clinical failure and underscores the need for cautious use of fluoroquinolones in settings with established resistance (Mostafa et al. 2020).

Clinical and surveillance implications of WGS

The integration of WGS with routine phenotypic testing demonstrated added value by providing detailed insights into resistance mechanisms, clonal diversity, and ExPEC lineages. Clinically, these data can inform empirical therapy selection, support antimicrobial stewardship, and guide infection prevention strategies. From a surveillance perspective, WGS enables the tracking of circulating lineages and resistance determinants, early detection of emerging high-risk clones, and identification of potential outbreak clusters (Neidhöfer et al. 2023). Although WGS is currently limited by cost and infrastructure, targeted use for research and surveillance purposes may enhance understanding of resistance dynamics in bloodstream infections. Furthermore, the absence of ST131 in this small collection highlights the need for region-specific genomic data to guide local infection control and empiric treatment recommendations, rather than relying solely on global prevalence data.

In conclusion, this study demonstrates the feasibility and utility of WGS in characterizing antimicrobial resistance and molecular epidemiology of *E. coli* BSI isolates in Indonesia. Despite the small sample size, the high concordance between genotype and phenotype, the diversity of sequence types, and the absence of ST131 provide valuable preliminary data for local surveillance and antimicrobial stewardship programs. Continued WGS-based monitoring is warranted to detect emerging high-risk clones, guide empirical therapy, and inform infection control strategies.

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REFERENCES

Afzal A, Gutierrez VP, Gomez E, Mon AM, Sarmiento CM, Khalid A, Polishchuk S, Al-Khateeb M, Yankulova B, Yusuf M, de Castro YS. 2022. Bloodstream infections in hospitalized patients before and during the COVID-19 surge in a community hospital in the South Bronx. Intl J Infect Dis 116: 43-46. https://doi.org/10.1016/j.ijid.2021.12.349.

Avershina E, Khezri A, Ahmad R. 2023. Clinical diagnostics of bacterial infections and their resistance to antibiotics—current state and whole-genome sequencing implementation perspectives. Antibiotics 12 (4): 781. https://doi.org/10.3390/antibiotics12040781.

Beshah D, Desta A, Belay G, Abebe T, Gebreselasie S, Tessema TS. 2022. Antimicrobial resistance and associated risk factors of Gram-negative bacterial bloodstream infections in Tikur Anbessa Specialized Hospital, Addis Ababa. Infect Drug Resist 15: 5043-5059. https://doi.org/10.2147/IDR.S371654.

Vanstokstraeten R, Pierard D, Crombé F, De Geyter D, Wybo I, Muyldermans A, Seyler L, Caljon B, Janssen T, Demuyser T. 2023. Genotypic resistance determined by whole-genome sequencing versus phenotypic resistance in 234 *Escherichia coli* isolates. Sci Rep 13: 449. https://doi.org/10.1038/s41598-023-27723-z.

Chmielarczyk A, Pomorska-Wesołowska M, Romaniszyn D, Wójkowska-Mach J. 2021. Healthcare-associated laboratory-confirmed bloodstream infections—species diversity and resistance mechanisms. Intl J Environ Res Public Health 18: 2785. https://doi.org/10.3390/ijerph18052785.

Di Franco S, Alfieri A, Pace MC, Sansone P, Pota V, Fittipaldi C, Fiore M, Passavanti MB. 2021. Bloodstream infections from MDR bacteria. Life 11: 575. https://doi.org/10.3390/life11060575.

Endraswari P, Mertaniasih NM, Setiawan F, Paramita AL. 2022. Epidemiology of *Escherichia coli* as a critical pathogen of bloodstream infection patients in a tertiary referral hospital. Indones J Trop Infect Dis 10: 205-213. https://doi.org/10.20473/ijtid.v10i3.39494.

Forde BM, Bergh H, Cuddihy T, Hajkowicz K, Hurst T, Playford EG, Henderson BC, Runnegar N, Clark J, Jennison AV, Moss S. 2023. Clinical implementation of routine whole-genome sequencing for hospital infection control of multidrug-resistant pathogens. Clin Infect Dis 76: e1277-e1284. https://doi.org/10.1093/cid/ciac726.

Gambushe SM, Zishiri OT, El Zowalaty ME. 2022. Review of *Escherichia coli* O157:H7 prevalence, pathogenicity, heavy metal and antimicrobial resistance. Infect Drug Resist 15: 4645-4673. https://doi.org/10.2147/IDR.S365269.

Geurtsen J, de Been M, Weerdenburg E, Zomer A, McNally A, Poolman J. 2022. Genomics and pathotypes of the many faces of *Escherichia coli*. FEMS Microbiol Rev 46: fuac031. https://doi.org/10.1093/femsre/fuac031.

Grøv K, Håland E, Waagsbø B, Salvesen Ø, Damås JK, Afset JE. 2022. Empirical antimicrobial therapy for bloodstream infections not compliant with guidelines predicts poorer outcomes. Infect Dis (Lond) 54: 833-845. https://doi.org/10.1080/23744235.2022.2109208.

Hapsari A, Wijayana KA. 2024. Antibiotic therapy in neonatal sepsis. Med Health J 3: 249-256. https://doi.org/10.20884/1.mhj.2024.3.2.11437.

Korotetskiy IS, Shilov SV, Kuznetsova T, Kerimzhanova B, Korotetskaya N, Ivanova L, Zubenko N, Parenova R, Reva ON. 2023. Analysis of whole-genome sequences of pathogenic Gram-positive and Gram-negative isolates. Microorganisms 11: 323. https://doi.org/10.3390/microorganisms11020323.

Kwiecinski JM, Horswill AR. 2020. *Staphylococcus aureus* bloodstream infections: Pathogenesis and regulatory mechanisms. Curr Opin Microbiol 53: 51-60. https://doi.org/10.1016/j.mib.2020.02.005.

Li D, Elankumaran P, Kudinha T, Kidsley AK, Trott DJ, Jarocki VM, Djordjevic SP. 2023. Dominance of *Escherichia coli* sequence types ST73, ST95, ST127 and ST131. Microb Genom 9 (7): 001068. https://doi.org/10.1099/mgen.0.001068.

Lutwick L, Shweta F, Radel K, Bearman G. 2021. Central line-associated bloodstream infections. International Society for Infectious Diseases (ISID). Retrieved from: https://arpsp. cdc. gov/p rofile/infections/clabsi7hidden. [July, 8, 2025]

MacKinnon MC, McEwen SA, Pearl DL, Lyytikäinen O, Jacobsson G, Collignon P, Gregson DB, Valiquette L, Laupland KB. 2021. Mortality in *Escherichia coli* bloodstream infections. BMC Infect Dis 21: 606. https://doi.org/10.1186/s12879-021-06326-x.

Mostafa HH, Cameron A, Taffner SM, Wang J, Malek A, Dumyati G, Hardy DJ, Pecora ND. 2020. Genomic surveillance of ceftriaxone-resistant *Escherichia coli*. Front Microbiol 11: 1747. https://doi.org/10.3389/fmicb.2020.01747.

Neidhöfer C, Neuenhoff M, Jozič R, Atangcho B, Unsleber S, Neder U, Grumaz S, Parčina M. 2023. Exploring clonality and virulence gene associations in bloodstream infections. Front Cell Infect Microbiol 13: 1274573. https://doi.org/10.3389/fcimb.2023.1274573.

Nelwan EJ, Puspandari N. Paramita RI, Erlina, Renesteen E, Fadilah F. 2020. Whole genome sequencing data of *Escherichia coli* isolated from bloodstream infection patients in Cipto Mangunkusumo National Hospital, Jakarta, Indonesia. Data in Brief 30: 105631. https://doi.org/10.1016/j.dib.2020.105631.

Pokharel P, Dhakal S, Dozois CM. 2023. Diversity of *Escherichia coli* pathotypes. Microorganisms 11: 344. https://doi.org/10.3390/microorganisms11020344.

Schöneweck F, Schmitz RP, Rißner F, Scherag A, Löffler B, Pletz MW, Weis S, Brunkhorst FM, Hagel S. 2021. Epidemiology of bloodstream infections in Germany. Antimicrob Resist Infect Control 10: 132. https://doi.org/10.1186/s13756-021-00997-6.

Shahbazi R, Salmanzadeh-Ahrabi S, Aslani MM, Alebouyeh M, Falahi J, Nikbin VS. 2023. Genotypic and phenotypic characteristics of *E. coli* O25-B2-ST131. BMC Pediatr 23: 386. https://doi.org/10.1186/s12887-023-03866-w.

Sora VM, Meroni G, Martino PA, Soggiu A, Bonizzi L, Zecconi A. 2021. Extraintestinal pathogenic *Escherichia coli*. Pathogens 10: 1355. https://doi.org/10.3390/pathogens10111355.

Tyson GH, McDermott PF, Li C, Chen Y, Tadesse DA, Mukherjee S, Bodeis-Jones S, Kabera C, Gaines SA, Loneragan GH, Edrington TS. 2015. Whole-genome sequencing accurately predicts antimicrobial resistance in *Escherichia coli*. J Antimicrob Chemother 70: 2763-2769. https://doi.org/10.1093/jac/dkv186.

Weerdenburg E, Davies T, Morrow B, Zomer AL, Hermans P, Go O, Spiessens B, Van Den Hoven T, Van Geet G, Aitabi M, DebRoy C. 2023. Global distribution of O-serotypes and antibiotic resistance in ExPEC. Clin Infect Dis 76: e1236-e1243. https://doi.org/10.1093/cid/ciac421.