IJMAAS International Journal of Microbiology and Applied Sciences
ISSN 1115-4004 Volume 4 issue 3 Sept, 2025 Research Article

Antimicrobial Susceptibility profile of *Enterococcus faecalis* obtained from blood samples correlated with molecular detection of microbial resistance genes

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ABSTRACT

Bloodstream infections resulting from *Enterococcus faecalis* pose significant therapeutic challenges due to emerging antimicrobial resistance. This study examines the characteristics profile and resistance patterns of *Enterococcus faecalis* from blood samples obtained from patients in a tertiary hospital in Lagos State, Nigeria. A total of 103 blood samples were aseptically collected and processed using standard microbiological techniques. The isolates were characterized presumptively identified based on cultural, morphological, and biochemical properties, while species confirmation was done using the API 20 Strep system and PCR amplification of the 16S rRNA gene. Antibiotic susceptibility testing was determined via the Kirby-Bauer disc diffusion method, and all isolates were screened for resistance genes. Sixteen *Enterococcus faecalis* isolates (15.5%) were recovered from the samples. Among these, four isolates (25%) exhibited resistance to all antibiotics tested. High levels of resistance were observed to vancomycin (100%), cefotaxime, tigecycline, norfloxacin, azithromycin, and cefoxitin (62.5% each), while the highest susceptibilities were recorded for ertapenem (75%), meropenem, and chloramphenicol (62.5% each). Multiplex PCR analysis detected sul2 and sul1 genes responsible for sulphonamide resistance in 81.3% and 31.3% of the isolates, respectively, and one isolate tested positive for the blaZ gene, which encodes β-lactamase, conferring resistance to penicillins. Sequence analysis confirmed bacterial identities, and phylogenetic analysis revealed the evolutionary relationships among the strains. The detection of multidrug-resistant *E. faecalis* in bloodstream infections is alarming, as it limits therapeutic opinions, increases the risk of treatment failure and adverse outcomes. It highlights urgent need for antibiotic stewardship/effective infection control measures.

Keywords: Blood Infection, *Enterococcus faecalis,* Multidrug Resistance, Antibiotic Resistance Genes, PCR, Lagos State.

Introduction

Enterococcus species are Gram-positive, facultative anaerobic cocci that are part of the normal flora of the human gastro-intestinal tract. They are opportunistic pathogens capable of causing infections like bloodstream infections (also called bacteraemia), urinary tract infections, wound infections, intra-abdominal infections, and endocarditis. Among them, *Enterococcus faecalis* is most frequently implicated in human infections, especially in immunocompromised individuals. Reports show that *Enterococcus* is the second leading cause of urinary tract and wound infections and the third leading cause of bacteraemia (Wada *et al*, 2020).

Their inherent to several antibiotics and the ability to resistance readily acquire further resistance via mobile elements have made them important nosocomial pathogens. The rise of multidrug-resistant (MDR) Enterococci is of greatest concern as they are difficult to treat and associated with high mortality. A recent meta-analysis by Wada *et al.* (2020) estimated that approximately 25% of clinical *Enterococcus* isolates in Nigeria are vancomycin-resistant. Globally, antimicrobial resistance (AMR) in Enterococci has been increasing, with systematic reviews showing rising resistance over time to drugs like chloramphenicol, linezolid, and tetracycline with differences in resistance rates across regions.

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Nigeria faces a heavy AMR burden. A systematic analysis of the global burden of bacterial AMR published by the Lancet in 2022 shows an estimate of 1.27 million deaths directly attributed to AMR, and 4.95 million deaths associated with AMR. Western sub-Saharan Africa had the highest burden of all regions, with 27.3 deaths per 100,000 attributable to AMR. Recent reviews note that Nigeria's extensive antibiotic use have led to widespread resistant infections in hospitals, communities, animals and the environment (Alabi *et al*, 2025; Amupitan *et al,* 2025).

Despite this, clinical capacity is limited. Many hospitals lack routine susceptibility data, and bloodstream infections are often treated empirically without laboratory guidance.

Therefore, the purpose of this study is to provide comprehensive insight on *Enterococcus* species, particularly *E. faecalis*, as a human pathogen from blood samples as well as to characterise the antibiotic susceptibility to commonly used antibiotics and link the phenotypes to specific resistance genes extracted from the isolates, which is critical in informing treatment and control measures.

Materials and Methods

This study employed a laboratory-based cross-sectional design. Blood samples were obtained from consenting patients at Lagos University Teaching Hospital (LUTH), Idi-Araba, Lagos State, between September 23 and November 24, 2024. Sample processing, antibiotic susceptibility testing and molecular analysis were all carried out at the FOWM laboratory, Sabo Yaba, Lagos Nigeria.

Ethical Approval

The research protocol, informed consent documents, and all related study materials were reviewed and approved by the Health Research Ethics Committee of Lagos University Teaching Hospital, Lagos, Nigeria (Ref. No.: 19/12/2008a), prior to the commencement of the study.

The study was conducted in accordance with institutional guidelines and the ethical principles outlined in the Declaration of Helsinki.

Sample Collection and Processing

A total of 103 blood samples were aseptically obtained from patients by health care personnel from different wards at Lagos University Teaching Hospital (LUTH). Each sample was obtained following standard venepuncture procedures into sterile EDTA bottles and transported promptly to the laboratory for immediate processing in coolers which contained ice packs to help maintain a temperature of approximately 4°C.

Organism isolation

The blood samples were centrifuged at 6,500rpm. From the resultant supernatant, 0.1mL was inoculated onto Tryptic Soy Agar (TSA), then spread evenly with a sterile glass spreader and incubated at 37°C for 24 hours under aseptic conditions. Following incubation, colonies were observed for growth and subsequently subjected to identification tests. Quantitative colony forming unit per millilitre (CFU/mL) enumeration was not performed as the study primarily focused on isolation and antimicrobial susceptibility profiling, rather than quantification.

Identification of Isolates

Gram staining, as described by Forbes *et al* (2007), was used to identify Gram-positive cocci, followed by a series of standard biochemical tests including catalase, haemolysis, oxidase and bile esculin test (MacFaddin, 2000). Species confirmation was achieved using the API 20 Strep system (bioMerieux, 2017) and PCR amplification of the 16S rRNA gene (American Society for Microbiology, n.d).

Antimicrobial Susceptibility Testing (AST)

The Kirby-Bauer disk diffusion method, as described by the National Committee for Clinical and Laboratory Standard (CLSI) (2023), was used to determine the antibiotic susceptibility profiles of the isolates. The isolates were first sub-cultured on Nutrient Agar for 24 hours at 37°C, after which a single well-isolated colony suspension was prepared in 5mL of sterile normal saline inside a test tube. The suspension was adjusted to 0.5 MacFarland turbidity standard and Mueller Hinton agar plates were evenly inoculated with the suspension using a sterile swab stick.

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Antibiotic discs were applied after a fifteen-minute rest at room temperature. The isolates were tested against a panel of 11 antibiotics. The plates were incubated at 37°C for 24 hours after which the zones of inhibition were measured and interpreted. The antibiotics used included: Tigecycline (15µg), Meropenem (10µg), Norfloxacin(10µg), Vancomycin (30µg), Chloramphenicol (30µg), Ertapenem (10µg), Cefotaxime (25µg), Sulfamethoxazole-thrimethoprim (25µg), Azithromycin (15µg), Linezolid (5µg) and Cefoxitin (30µg).

Molecular Analysis

Deoxyribonucleic Acid (DNA) Extraction by Boiling

Genomic DNA was extracted from pure isolates using the boiling method. Bacterial cells grown overnight were suspended into sterile water, vortexed until completely dissolved, centrifuged for 5 minutes at 10,000rpm, and the pellet resuspended in sterile water. The suspension was boiled for 10 minutes at 100°C in a heat block incubator and immediately cooled on ice. The solutions were centrifuged for 5 minutes at 10,000rpm to separate cellular debris and any remaining cell fragments from the DNA. The resulting supernatants, containing the DNA, were carefully decanted into fresh Eppendorf tubes and stored at 4°C to preserve stability for subsequent PCR amplification (Dashti *et al*., 2009).

Multiplex Polymerase Chain Reaction (PCR) Amplification of Resistance Genes

The PCR reaction was carried out using the Soils Biodyne 5x FIREPol Master Mix. PCR was performed in 25µL of a reaction mixture, and the reaction concentration was brought down from 5x concentration to 1x concentration so that each 25µL reaction contained 1x Master Mix buffer (Soils Biodyne, Estonia), 1.5mM $MgCl_2$, 200µM of each deoxynucleoside triphosphates (dNTPs), 25pMol of each primer (Stab Vida, Portugal), 2 unit of Hot FIREPol DNA polymerase (Soild Biodyne), Proofreading Enzyme and 5µL the extracted DNA.

Thermal cycling was conducted in a Techne Prime thermal cycler for an initial denaturation of 95°C for 15mins, followed by 35 amplification cycles of 30 seconds at 95°C, and then 1 minute primer-specific annealing and 1 minute 30 seconds extension at 72°C extension.

A final 10minute extension was done at 72°C (American Society for Microbiology, n.d.).

Table 1 presents the Antibiotics resistance primers used for this studyand the conditions for the Amplification of Resistance Genes.

Table 1: Antibiotics resistance primers used and the conditions for Amplification of Resistance Genes

Target Gene	Primer Name	Sequence (5'-3')	Annealing Temp (°C)	Base pair (bp)	Reference
Tetracycline	TetA	F: GCTACATCCTGCTTGCCTTC R: CATAGATCGCCGTGAAGAGG	55	210	Bacci *et al*., 2012
	TetB	F: TTGGTTAGGGGCAAGTTTTG R: GTAATGGGCCAATAACACCG		659	
β-lactamase	BlaZ	F: CAAAGATGATATAGTTGCTTATTCTCC R: TGCTTGACCACTTTTATCAGC		421	Kaase *et al*., 2008
	Sul1	F: CGGCGTGGGCTACCTGAACG R: GCCGATCGCGTGAAGTTCCG		433	Taban *et al*., 2013
Sulfonamides	Sul2	F: GCGCTCAAGGCAGATGGCATT R: GCGTTTGATACCGGCACCCGT	52	293	Taban *et al*., 2013

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Polymerase Chain Reaction (PCR) Amplification for 16S rRNA genes (27F and 1492R)

Polymerase chain reaction was carried out to amplify the 16SrRNA gene of the bacteria using the primer pair 27F- 5′-AGAGTTTGATCCTGGCT CAG -3', and 1492R 5'- GGTTACCTTGTTACGACTT -3' as described by (Lane 1991).

PCR was performed in 25 µl of a reaction mixture, and the reaction concentration was brought down from 5x concentration to 1X concentration containing 1X Blend Master mix buffer Buffer (Solis Biodyne, Estonia), 1.5 mM MgCl2, 200µM of each deoxynucleoside triphosphates (dNTP)(Solis Biodyne), 25pMol of each primer (Stab Vida, Portugal), 2 unit of Hot FIREPol DNA polymerase (Solis Biodyne), Proofreading Enzyme, 5µl of the extracted DNA, and sterile distilled water was used to make up the reaction mixture.

Thermal cycling was conducted in a Techne thermal cycler (3 prime series) for an initial denaturation of 95°C for 15 minutes followed by 35 amplification cycles of 30 seconds at 95°C; 1 minute at 61°C and 1 minute 30 Seconds at 72°C. This was followed by a final extension step of 10 minutes at 72°C.

Agarose Gel Electrophoresis

The amplification products were resolved on 1.5% agarose gels, using a 100bp DNA ladder as molecular weight marker. Electrophoresis was carried out at 80V for 1hour 30 minutes, after which, DNA bands were visualised by ethidium bromide staining under UV illumination (Lee *et al.*, 2012).

Sanger Sequencing

The 16s rRNA amplicons were purified using the EXOSAP- IT reagents according to Manufacturer's instructions and the purified PCR products were sent to EPOCH life Science USA for sanger sequencing.

The corresponding sequences were identified using the online blast search at http://blast.ncbi.nlm.nih.gov/ Blast.cgi and then submitted at the NCBI GenBank to obtain accession numbers.

Results

A total of 103 blood samples from Lagos State Teaching Hospital (LUTH) were analysed to for this study, among which 16 isolates (15.5%) were identified to be *Enterococcus faecalis*, which were confirmed through Gram staining, biochemical characterisation, morphological observation. The identity of the isolates was further validated using the API 20 Strep kit.

Antibiotic susceptibility testing on the 16 isolates revealed that four isolates (25%) were multidrug-resistant, showing resistance to all antibiotics tested. All isolates exhibited absolute resistance to Vancomycin (100%) according to CLSI (2023) interpretative standards. Ten isolates (62.5%) were resistant against Tigecycline, Norfloxacin, Cefotaxime, Azithromycin and Cefoxitin and a 37.5% susceptibility rate was recorded in these antibiotics.

Resistance was observed in nine isolates (56.3%) against Sulfamethoxazole-trimethoprim and Linezolid. The isolates showed highest susceptibility to Ertapenem (75%), followed by Meropenem and Chloramphenicol (62.5%). Overall, resistance patterns varied across antibiotic classes, with carbapenems (Meropenem and Ertapenem) and Chloramphenicol showing comparatively higher activity against the isolates (Figure 1).

Multiplex PCR analysis of the 16 isolates revealed that five isolates (31.3%) were positive for the *sul1* gene and 13 isolates (81.3%) carried the *sul2* gene, which is indicative of resistance to sulphanomides (Plate 1). One isolate (6.3%) was positive for the *blaZ* gene, while none of the isolates tested positive for *tetA* and *tetB* genes (Plate 2). The detection of *sul1* and *sul2* genes corresponded with the phenotypic resistance observed against Sulfamethoxazole-trimethoprim, whereas the single *blaZ*-positive isolate reflected plasmid-mediated β-lactam resistance.

Agarose gel electrophoresis verified successful amplification of the 16S rRNA gene across all isolates, and subsequent phylogenetic analysis illustrated their evolutionary relationships (Figure 2).

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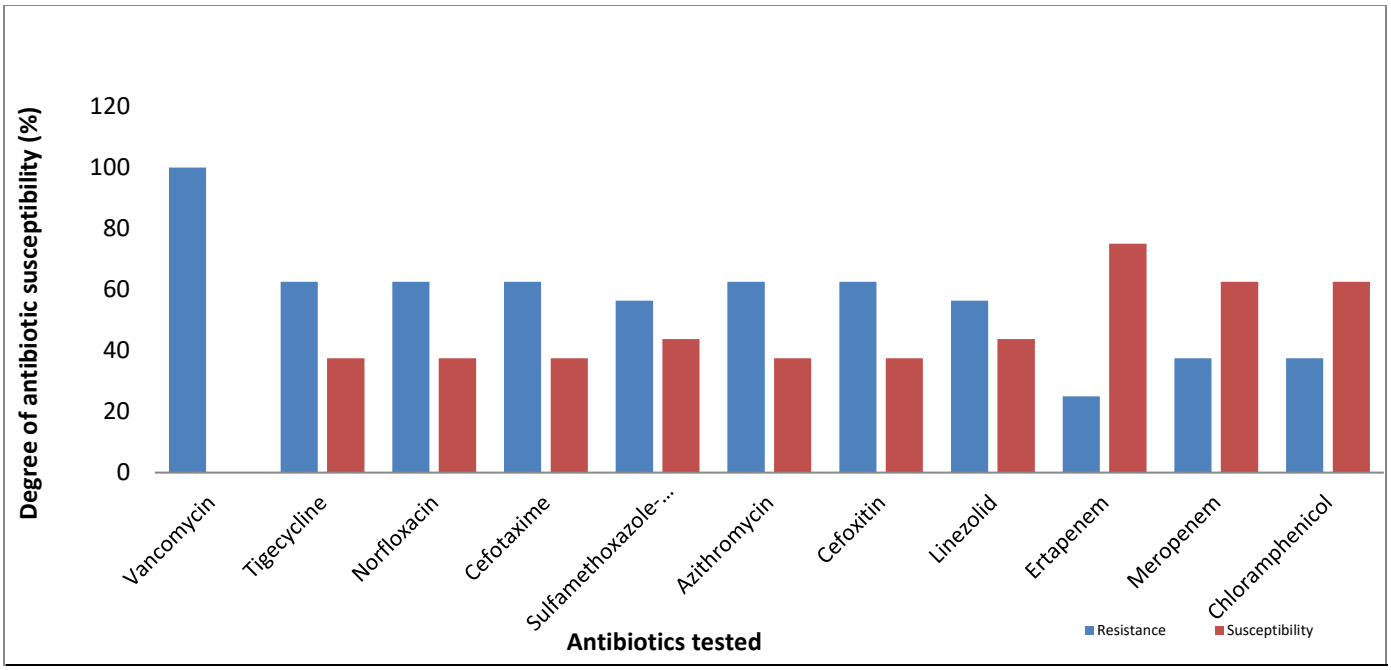

Figure 1: Antibiotic susceptibility patterns of the isolates

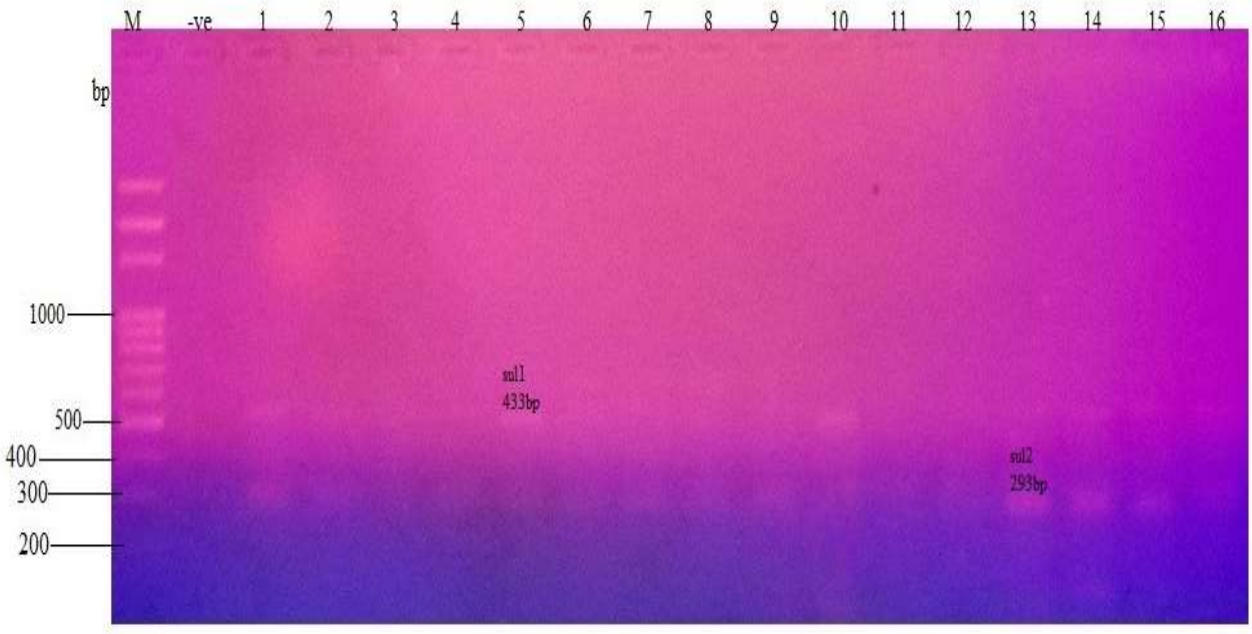

Plate 1: Distribution of *sul1* and *sul2* genes of the isolates

Gel electrophoresis showing 16 *Enterococcus faecalis* isolates amplification of Sulphonamides genes (*Sul1*, *Sul2*) amplified at 433bp and 293bp respectively. M- 100bp DNA ladder, -ve- Negative control. Lane 1,5,10,14,15 was positive for *Sul1* while Lane 1-9, 13-16 were positive for *Sul2*

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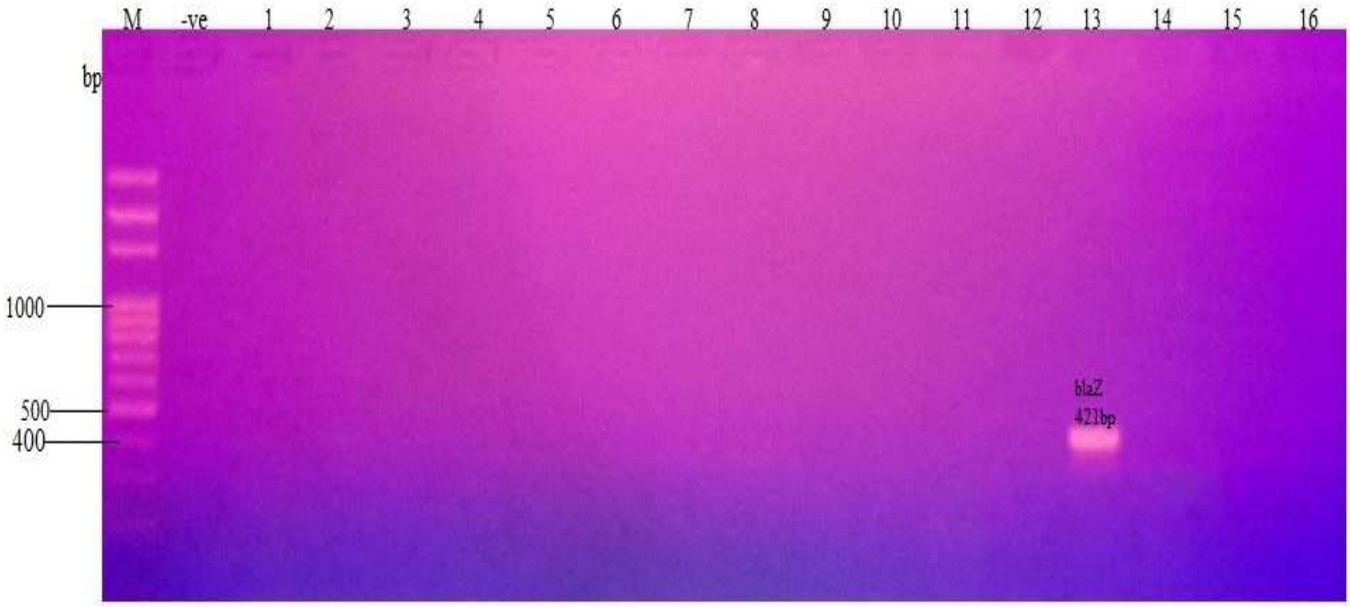

Plate 2: Distribution of *tetA, tetB* and *bla* genes of the isolates

Gel electrophoresis showing 16 *Enterococcus faecalis* isolates amplification of (*tetA, tetB* and *blaZ*) genes amplified at 210bp 659bp and421 bp respectively. M- 100bp DNA ladder, -ve- Negative control. Lane 13 was positive for *blaZ*. All samples were negative for *tetA* and *tetB*.

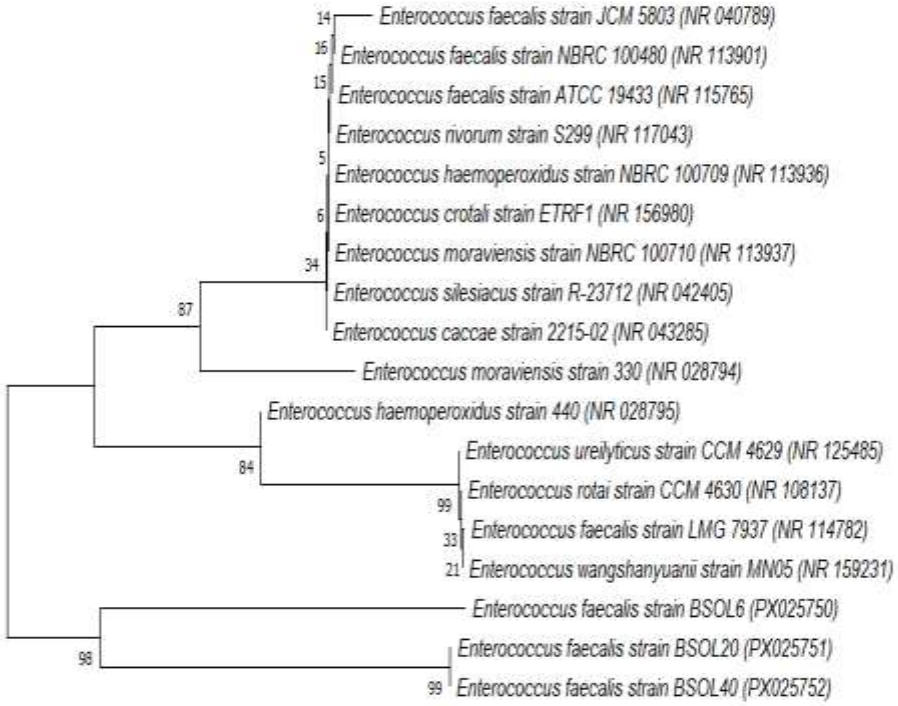

Figure 2: Phylogenetic relationships of *Enterococcus faecalis* isolates from blood samples

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Discussion

In this study, *Enterococcus faecalis* accounted for 15.5% of bloodstream isolates, and the isolates demonstrated notable multidrug-resistance patterns. All isolates showed resistance to vancomycin, even though it is not routinely prescribed in this environment. This observation may be related to indirect selective pressure from other commonly used antibiotics or circulation of resistant strains within the healthcare setting. Similar findings have been reported in Lagos by Salami *et al.* (2025), who also described high vancomycin resistance among Enterococcus species from different sample sources. Studies from other regions, such as South Africa (Founou *et al.*, 2024) and East Africa (Khatiebi *et al.,* 2022), have also documented increasing multidrug resistance in *E. faecalis*, supporting the wider pattern observed in our study.

Resistance to tigecycline, norfloxacin, cefoxitin, cefotaxime and azithromycin was observed in 62.5% of isolates, and 56.3% were resistant to sulphamethoxazole–trimethoprim and linezolid. These findings are in line with reports from other African and international studies, which have described reduced susceptibility to several antibiotic classes (Guang *et al.,* 2024; Sangiorgio *et al.,* 2024). While some earlier Nigerian studies reported better activity for carbapenems and chloramphenicol, the moderate susceptibility observed in our isolates is similar to more recent data (Dauphin *et al*., 2020), suggesting evolving patterns over time.

The resistance genes detected provide supportive evidence for the phenotypic results. The high detection rates of sul2 and sul1 are consistent with findings from Egypt and Europe, where these genes often coexist with determinants for resistance to other antibiotic classes (Geraldes *et al*., 2022; Said *et al.,* 2024). The presence of blaZ in one isolate agrees with previous reports of β–lactamase activity in *E. faecalis*. The absence of tetA/B genes is also in keeping with expectations for this species.

Phylogenetic analysis showed that all isolates clustered closely with established *E. faecalis* reference strains with high bootstrap support. This pattern of relatedness is similar to reports by Morgan *et al.* (2011) and reflects limited genetic divergence, possibly due to isolates originating from a similar clinical environment or circulating within a related transmission network.

From a clinical standpoint, the resistance patterns observed here are comparable to those documented in other recent studies and serve as useful information for guiding therapy. Although some antibiotics remain effective, the variation in susceptibility highlights the importance of using local data when making treatment decisions. Additionally, because Enterococci are known to persist in hospital environments and can spread through several routes, the findings further emphasize the value of routine antimicrobial surveillance and adherence to standard infection-control practices, as also recommended by Adesida *et al.* (2017) and Alabi *et al.* (2025). Overall, our results align well with local, regional and international reports and contribute to the growing body of evidence on *E. faecalis* resistance patterns. They highlight the need for continued monitoring and careful antibiotic selection to support effective patient management in the clinical setting.

Conclusion

The high levels of vancomycin resistance and resistance to several antibiotic groups observed in this study suggest that some of the usual treatments for enterococcal bloodstream infections may soon become less effective locally. Misuse and overuse of antibiotics are likely contributing to the spread of these resistant *Enterococcus* strains. For this reason, routine antibiotic susceptibility testing of enterococcal isolates should be encouraged. In addition, regular molecular monitoring of *E. faecalis* in hospitals, along with strengthened infection-control measures such as proper hand hygiene, thorough environmental cleaning, and appropriate patient isolation, will be helpful in reducing transmission. Establishing strong antimicrobial stewardship programmes will also support better antibiotic use. Together, these measures aligned with the One Health approach are important for limiting the spread of multidrug-resistant *E. faecalis* and ensuring that effective treatment options remain available.

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Acknowledgements

We appreciate the Health Research Ethics Committee of Lagos University Teaching Hospital, Lagos, Nigeria for granting us the Ethical approval (Ref. No.: 19/12/2008a) to conduct this study.

Conflicts of interests: None declared by authors.

Authors' contributions: Egwuatu, T. O. G. conceptualized and designed the study, supervised the project, critically reviewed and vetted the manuscript, and approved the final version for publication. Olaogun, O. O. was responsible for sample collection, microbial isolation, and antibiotic susceptibility testing. Onyeaghasiri F. U. and Obioma P. C. performed the molecular analyses. All authors participated in drafting and revising the manuscript and approved the final submission.

Funding: None declared by authors.

References

Adesida, A.S., Ezenta C.C., Adagbada O.A., Aladesokan A.A. & Coker O.A. (2017). Carriage of multidrug resistant *Enterococcus faecium* and *Enterococcus faecalis* among apparently healthy humans. *African J Infect Dis, 11(2),* 83–89.

Alabi, D.E., Rabiu, G.A. & Adesoji, T.A. (2025). A review of antimicrobial resistance challenges in Nigeria: the need for a one health approach. *One Health*, 20, 101053.

American Society for Microbiology. (n.d.). Polymerase chain reaction (PCR) protocol. ASM. https://asm.org/media/protocol-images/polymerase-chain-reaction-protocol.pdf

Amupitan, A.A., Adeyemo, T.A, Amupitan, O.A, Obadare, O.T. & Aboderin, O.A. (2025). Bacteraemia in a Nigerian hospital: implementing antimicrobial resistance surveillance. *J Public Health Afr, 16(1),* a665.

Antimicrobial Resistance Collaborators (2022). Global burden of bacterial antimicrobial resistance in 2019; a systematic analysis. *Lancet*, *399(10325),* 629-655.

Bacci, C., Boni, E., Alpigiani, I., Lanzoni, E., Bonardi, S., & Brindani, F. (2012). Phenotypic and genotypic features of antibiotic resistance in Salmonella enterica isolated from chicken meat and chicken and quail carcasses. *Inter J Food Microbiol, 160(1),* 16-23.

BioMérieux. (2017). Your Expert in Infectious Diseases at ECCMID 2017. https://www.Rapid microbiology. com/news /biomerieux-your-expert-in-infectious-diseases-at-eccmid-2017.

CLSI. (2023). Performance Standards for Antimicrobial Susceptibility Testing.33rd Edition. Clinical and Laboratory Standards Institute. CLSI document M100. Wayne, PA: CLSI.

Dashti, A.A., Jadaon, M.M., Abdulsamad., A.M., & Dashti, H.M. (2009). Heat treatment of bacteria: a simple method of DNA extraction for molecular techniques. *Kuwait Med J.*, *41(2),* 117–122.

Dauphin, D.M. & Oluwolwe, M.D. (2020). Prevalence, antibiotic susceptibility pattern and risks of multiple drug resistant Enterococcus species in Ojo, Lagos, Nigeria. *New York Sci J., 13(7),* 25–32.

Forbes, B.A., Sahm, D.F., & Weissfeld, A.S, Bailey & Scott's diagnostic microbiology, 12th ed, Vol 1, Mosby Elsevier, St. Louis, 2007, pp. 80–83.

Founou, R.C., Founou, L.L., Allam, M., Ismail, A. & Essack, Y.S. (2024). Genome analysis of multidrug resistant *Enterococcus faecium* and *Enterococcus faecalis* circulating among hospitalised patients in uMgungundlovu District, KwaZulu-Natal, South Africa. *BMC Infect Dis*, *24*, 671.

Fraser, S.L., Donskey, C.J. & Salata, R.A. (2024). Enterococcal infections clinical presentation. Medscape: Infectious Diseases. https://emedicine. medscape.com/article/216993-clinical?form=fpf#b1

Georges M., Odoyo E., Matano D., Tiria F., Kyany'a C., Mbwika D. *et al*. (2022). Determination of *Enterococcus faecalis* and *Enterococcus faecium* antimicrobial resistance and virulence factors and their association with clinical and demographic factors in Kenya. *J Pathog*, *2022 (1),* 1–10.

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Geraldes C., Tavares L., Gil S. & Olivera M. (2022). *Enterococcus* virulence and resistant traits associated with its permanence in the hospital environment. *Antibiotics, 11(7),* 857.

Guang, L., Beig M., Wang, L., Navidifar, T., Moradi, S., Faezah, M.T *et al.* (2024). Global status of antimicrobial resistance in clinical *Enterococcus faecalis* isolates: systematic review and meta-analysis. *Ann Clin Microbiol Antimicrob, 23(80).*

Kaase, M., Lenga, S., Friedrich, S., Szabados, F., Sakinc, T., Kleine, B., & Gatermann, S.G. (2008). Comparison of phenotypic methods for penicillinase detection in Staphylococcus aureus. *Clin Microbiol Infect, 14(10),* 951–955.

Khatiebi S., Kiprotich K., Oyando Z., Mwaura J., Wekesa C., Chi, N.C. *et al*. (2022). High-throughput metagenomics of microbial footprints uncovers a cocktail of noxious antibiotic resistance genes in the Winam gulf of lake Victoria, Kenya. *J trop med*, *2024(1),* 7857069.

Kilpper-Baiz, R. & Schleifer K.H. (1984). Transfer of *Streptococcus faecalis* and *Streptococcus faecium* to the genus *Enterococcus* nom.rev. as *Enterococcus faecalis* comb. nov. and *Enterococcus faecium* comb. nov. *International Journal of Systematic Bacteriology 34(1),* 31–34.

Lane, D. J. (1991). 16S/23S rRNA sequencing. In: Stackebrandt, E. & Goodfellow, M. (eds.), Nucleic Acid Techniques in Bacterial Systematics. John Wiley & Sons, New York, pp. 115–175.

Lee, P.Y., Costumbrado, J., Hsu, Y., & Kim, Y. H. (2012). Agarose gel electrophoresis for the separation of dna fragments. *J Vis Exp.*, *(62),* 3923.

Lewis, S.J., Kirn, J.T., Weinstein, P.M., Limbago, B., Bobenchik, M.A, Mathers, J.A *et al*. *Performance Standards for Antimicrobial Susceptibility Testing, 33rd edition.,* Clinical and Laboratory Standard Institute. Wayne (PA), 2023, pp107–111.

MacFaddin J.F, *Biochemical Tests for Identification of Medical Bacteria, Vol. 1, 3rd edition,* Lippincott Williams & Wilkins, Philadelphia, 2000, pp. 8, 78, 368.

Melese, A., Genet, C & Andulem, T (2020). Prevalence of vancomycin resistant enterococci (VRE) in Ethiopia: a systematic review and meta-analysis. *BMC Infect Dis. 20*, 1–10.

Morgan A. Margie., Marlowe Elizabeth., Novak-Weekly Susan., Miller J.M., Painter T.M., Salimnia Hossein & Crystal Benjamin. (2011). A 1.5 hour procedure for identification of *Enterococcus* species directly from blood cultures. *J Vis Exp, 48*, e2456.

Nashibula, D & Mustapha, A. (2022). Prevalence of *Enterococci* infection in blood cultures based on age group at Makere university clinical microbiology laboratory: a cross-sectional study. *Stud J Health Research*, *5(2),* 45–53.

Ngbede, O.E., Issa, S., Akwuobu, A.C., Nanven, A.M., Adikwu, A.A., Abba, O.P *et al.* (2023). Carriage of linezolid-resistant enterococci (LRE) among humans and animals in Nigeria: coexistence of *the cfr, optrA* and *poxtA* genes in *Enterococcus faecium* of animal origin. *J Glob Antimicrob Resist, 34*, 234–239.

Ogunkunle T.O., Abdulkadir .M.B., Katibi O.S., Bello S.O., Raheem R.A. & Olaoshebikan, R. (2020). Paediatric blood culture isolates and antibiotic sensitivity in a Nigerian tertiary hospital. *Niger J Med*, *29(2),* 261–264.

Orababa, O.Q., Soriwei, J.D., Akinsuyi, S.O., Essiet, U.U. & Solesi, O.M. (2021). A systematic review and meta-analysis on the prevalence of vancomycin-resistant enterococci (VRE) among Nigerians. *Porto Biomed J., 6(1),* 125.

Ruth M., Husna I., Olga P & Sabelle J. (2022). A retrospective analysis of Culture-confirmed Enterococci bloodstream infections in South Africa, 2016–2020: a cross-sectional study. *Trop Med Infect Dis, 8(1),* 19.

Said, S.M., Tirthani, E. & Lesho, E. (2024). *Enterococcus* infections. *National Library of Medicine*.https://www.ncbi.nih.gov/books/NBK549803/

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Salami, O.W., Ajoseh, O.S., Lawal-Sanni, O.A., Ashraf, A.A., Heinrich, N., Gamal, W & Akinyemi, O.K. (2025). Prevalence, antimicrobial resistance patterns, and emerging carbapenemase-producing Enterococcus species from different sources in Lagos, Nigeria. *Antibiotics*. 14(4), 398.

Sangiorgio G., Calvo M., Migliorisi G., Campanile F. & Stefani S (2024). The impact of *Enterococcus* spp. in the immunocompromised host: a comprehensive review. *Pathogens, 13(5),* 409.

Shuwaram, A.S. & Kenneth, C.I. (2019). Antimicrobial resistance pattern of Enterococci isolated from stool samples in a tertiary hospital in Nigeria. *Ann Trop Pathol. 10(2),* 126–131.

Suhair, A.A., Jinan, M.H., Elham, J.K and Aseel, S.I. (2020). Bacterial study of *Enterococcus* bacteria isolated from different diseases in the female cows. *Biomed Pharm J., 13(2),* 643–50.

Taban, B.M., Aytac, S.A., Akkoc, N., & Akcelik, M. (2013). Characterization of antibiotic resistance in *Salmonella enterica* isolates determined from ready-to-eat (RTE) salad vegetables. *Brazilian J Microbiol*, *44*, 385-391.

Thomas, J.L. (2012). Choosing optimal antimicrobial therapies. *Med Clin North America, 96(6),* 1079–1094.

Wada, Y., Harun, A.B., Yean, C.Y. & Zaidah, A.R. (2020). Vancomycin-resistant enterococci (VRE) in Nigeria: the first systematic review and meta-analysis. *Antibiotics, 9(9),* 565.

Yusuf-Omoloye N.A., Adeyemi M.F., Sule F.W., Yusuf W., Ajigbewu H.O., Adegbite-Badmus K.M *et al.* (2023). Culture-dependent and molecular characterisation of vancomycin-resistant Enterococci from clinical, food, and water samples across Osun State, Southwest Nigeria. *Scientific African, 21*, e01813.

Citation: Egwuatu *et al*. (2025). Antimicrobial susceptibility profile of *Enterococcus faecalis* obtained from blood samples correlated with molecular detection of microbial resistance genes. *International Journal of Microbiology and Applied Sciences*. *4(3)*: 310 – 319.