



OPEN Extended Spectrum Beta-Lactamase-Producing Bacterial Clones in West Africa: A Systematic Review and Meta-Analysis from a One Health Perspective

Samuel Nee-Amugie Yartey, Frederick Kungu, Anastasia Akosua Asantewaa & Eric S. Donkor✉

Extended-spectrum beta-lactamase (ESBL)-producing bacteria are associated with life-threatening infections with limited treatment options worldwide. In this review, we assess the circulating ESBL-producing bacterial clones from human, animal, and environmental sources in West Africa, through a One Health approach. A systematic search in PubMed, Scopus, and Web of Science identified 485 records, with 38 studies analysed. Data were organized thematically and pooled prevalence estimates calculated through a random-effects meta-analysis. Results showed a 16.8% (95% CI [12.1; 22.1]) prevalence of ESBL-producing bacteria, with *Escherichia coli* (64%) being the most frequently detected species. Humans were the most affected (20.4%), followed by animals (12.8%) and the environment (9.3%). Among the 153 identified bacterial clones, ST10, ST410, ST58, ST155, ST4684, ST2178, and ST37, found majorly in *E. coli*, occurred across multiple sources, suggesting cross-sectoral transmission. The *bla*CTX-M gene, prevalent with several fluoroquinolone and aminoglycoside resistance genes, was often on conjugative IncF plasmids (50.0%), suggesting high horizontal transmission potential. The emerging ESBL gene, GES (1.2%) was also detected in *Pseudomonas aeruginosa*. The findings reinforce the need for robust antimicrobial resistance monitoring and intervention strategies in West Africa. A regional One Health-based approach is necessary to curtail the spread of resistant bacterial clones and safeguard the efficacy of available treatment options.

Keywords ESBL, Clones, One health, West Africa

Abbreviations

bla	Beta-lactamase
CI	Confidence interval
CMY	Cephamecinase
Col	Colicinogenic
CTX-M	Cefotaximase
DDST	Double-disk synergy test
ESBL	Extended-spectrum beta-lactamase
EUCAST	European Committee on Antimicrobial Susceptibility Testing
GCC	Gulf Cooperation Council
GES	Guiana extended-spectrum
ICU	Intensive Care Unit
IncF/H/I/K/N/Q/R/X/Y	Incompatibility group F/H/I/K/N/Q/R/X/Y
JB1	Joanna Briggs Institute
MLST	Multilocus sequence typing
NICU	Neonatal intensive care unit
OXA	Oxacillinase

Department of Medical Microbiology, University of Ghana Medical School, Korle Bu, P. O. Box KB 4236, Accra, Ghana. ✉email: esampene-donkor@ug.edu.gh

PRISMA	Preferred reporting items for systematic reviews and meta-analyses
PROSPERO	International Prospective Register of Systematic Reviews
QRDR	Quinolone resistance determining region
RIS	Research information systems
SHV	Sulfhydryl-variable
ST	Sequence type
TEM	Temoneira
UTI	Urinary tract infection
VEB	Vietnamese extended-spectrum beta-lactamase
WGS	Whole genome sequencing

Extended-spectrum beta-lactamases (ESBLs) are enzymes primarily produced by *Enterobacteriaceae*, conferring resistance to a wide range of broad spectrum of beta-lactam antibiotics, including penicillins and cephalosporins¹. This has intensified concerns about antimicrobial resistance among human and animal populations worldwide². The growing prevalence of ESBL-producing bacteria is now a major public health challenge, making common treatments less effective and facilitating spread beyond healthcare facilities into community settings, with implications for both human and animal health.

ESBLs are primarily plasmid-mediated enzymes that hydrolyse the beta-lactam ring of penicillins and cephalosporins¹. Among the plethora of identified ESBL types, *bla*CTX-M, *bla*SHV, and *bla*TEM are the most prevalent. Initially, TEM (Temoniera) and SHV (Sulfhydryl-variable) were predominantly found in hospital-acquired infections, especially in resistant *Klebsiella pneumoniae*. However, CTX-M (Cefotaximase) enzymes have recently become the most widespread globally, appearing in both hospital and community settings, with *Escherichia coli* now the most commonly associated bacterium³.

The most commonly identified ESBL-producing bacteria are *Escherichia coli* and *Klebsiella pneumoniae*⁴. Although these organisms are naturally part of the human gut flora, they can cause severe infections when they invade other parts of the body, such as the urinary tract, bloodstream, and lungs^{1,5}. A study focused on the Gulf Cooperation Council (GCC) countries reported that the prevalence of ESBL-producing bacteria, particularly among *Enterobacteriales*, in clinical samples ranged from 21.6% to 29.3%⁶. The rates were notably higher in intensive care unit (ICU) patients, with prevalence ranging from 17.3% to 31.3%. Similarly, urinary tract infections had prevalence rates of 25.2%–31.7%⁶. This clearly demonstrates the important struggle caused by ESBL-producing pathogens in healthcare settings.

In addition to resistance to beta-lactam antibiotics, ESBL-producing bacteria frequently exhibit resistance to other classes of antibiotics, including aminoglycosides and fluoroquinolones⁷. This co-resistance complicates treatment options and poses a significant challenge in clinical settings, especially for last-resort carbapenems⁸.

The prevalence of ESBL-producing bacteria varies significantly across regions. Approximately 16.5% of the global population is affected by these resistant bacteria, with particularly alarming rates observed in many developing countries⁹. For instance, studies have shown that the prevalence of ESBL-producing *E. coli* can exceed 50% in certain populations^{10–12}. Countries such as India, China, and parts of Africa, including Ethiopia, have reported especially high rates of these resistant strains, indicating a rise in public health crises^{13–15}.

Clonal dissemination is a well-known factor in the spread of ESBL-producing bacteria, with specific clones identified across various reservoirs, indicating potential transmission pathways between humans, animals, and the environment¹⁶. Despite the global focus on ESBL-producing bacteria, a significant gap remains in our understanding of their clonal interactions, especially in West Africa. Comprehensive studies on the spread of these clones between humans, animals, and the environment in this region are lacking, leaving critical questions about their public health implications unanswered. This knowledge gap raises concerns about the role of animals and the environment as reservoirs and vectors of these resistant strains, which could have serious implications for human health.

This systematic review investigated the distribution and interconnections of ESBL-producing bacterial clones among humans, animals, and the environment in West Africa. We focused on the shared characteristics and possible transmission pathways of these clones in order to lay emphasis on the urgent need for additional research to address this pressing public health challenge.

Methods

Study design and systematic review protocol

This study was registered in the International Prospective Register of Systematic Reviews (PROSPERO) with registration ID: CRD42024608157 and followed the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) guidelines¹⁷.

Search strategy

We searched several databases, including PubMed, Scopus, and Web of Science, to retrieve relevant articles, applying no restrictions on publication year. The search terms included keywords such as “ESBL, Extended Spectrum Beta Lactamase,” “One Health,” “Human,” “Animal,” “Environment,” and specific countries in West Africa (Supplementary data 1; Table 1). The last database search was conducted on October 28, 2024.

Study selection

The studies identified from individual databases were first exported and compiled in Zotero (Version 6.0.30), where they were merged into a single RIS file. This file was then imported into Rayyan, an online platform designed for systematic review screening, where duplicate studies were manually identified and resolved. Titles and abstracts were screened to determine their relevance based on the inclusion and exclusion criteria. Three

reviewers independently analyzed all the studies from the search terms' title, abstract, and selected full texts. A fourth reviewer addressed any inconsistencies or disagreements.

Eligibility criteria

Studies were included if they met the following criteria:

1. Reported both phenotypic and genotypic identification of ESBL-producing bacteria from human, animal, or environmental sources.
2. Conducted antimicrobial susceptibility testing of bacterial isolates.
3. Were based on research conducted in West Africa.

Studies were excluded if they were:

1. Abstracts, review articles, letters to editors, case reports, research notes, systematic reviews.
2. Laboratory-based studies focusing only on resistance gene transfer.
3. Studies conducted during outbreaks.
4. Studies focusing solely on the phenotypic or genotypic identification of ESBL-producing bacteria.

Data extraction

The following data were extracted and summarized from the final selected studies: author name and year of publication, study period, study design, country, population, sample size, settings, sample types, ESBL diagnostic method, ESBL bacterial species, ESBL positive isolates, phenotypic resistance to Ciprofloxacin (CIP), Nalidixic acid (NAL), Chloramphenicol (CHL), Imipenem/Meropenem (IPM/MEM), Tobramycin (TOB), Gentamicin (GEN), Tetracycline/Doxycycline (TET/DOX), Trimethoprim-Sulfamethoxazole (TMP/SXT), ESBL and other co-resistance genotypes, ESBL clones (sequence types), and associated ESBL plasmid types. The extracted data were compiled in Microsoft Excel for further analysis.

Quality assessment

A critical appraisal tool developed by the Joanna Briggs Institute (JBI) was used to assess the risk of bias among the included studies¹⁸. There are nine questions in this JBI instrument. Each answer was scored 0 or 1 based on whether the answer was “yes” or “no”. NA was used to indicate that the question was not relevant. Studies with scores of 7–8 indicated a low risk of bias, those with scores of 5–6 indicated a moderate risk of bias, and those with scores less than 5 indicated a high risk of bias (Supplementary data 1; Table 2).

Data analysis

Data were organized in narratives, figures, and tables. The meta-analysis of the prevalence of ESBL-positive isolates in humans, animals, and the environment was conducted using RStudio software version 4.4.2. This analysis included data from 38 studies included in the systematic review. The meta-package was used to estimate pooled prevalence using the DerSimonian-Laird method. To ensure consistency in variances across studies and facilitate pooled prevalence computation, the Freeman-Tukey double arcsine transformation was applied. Furthermore, 95% confidence intervals for individual studies were calculated using the Clopper-Pearson method.

The pooled prevalence of ESBL-positive isolates was measured, and subgroup analyses were performed according to country, setting, and population. Studies reporting on ESBL-positive *E. coli* and *K. pneumoniae* isolates were analyzed separately to determine their prevalence. Subgroup analyses were also conducted on the basis of the settings and sample types. This approach was adopted because *E. coli* and *K. pneumoniae* emerged as the most prevalent ESBL-positive bacteria across human, animal and environmental sources.

The random-effects model was used to generate forest plots showing the study-specific effect sizes with a 95% confidence interval (CI) for the pooled prevalence. The I^2 statistic was used to measure the heterogeneity among the studies. A value close to 0% indicates no heterogeneity, whereas values close to 25%, 50%, and 75% correspond to low, moderate, and high heterogeneity, respectively. The p-values correspond to the heterogeneities between studies from the chi-squared test of the null hypothesis that there is no heterogeneity. The distribution of clones circulating across human, animal, and environmental sources was analysed. Publication bias was measured using funnel plots to test for symmetry and was further complemented using Egger's regression test.

We also conducted sensitivity and meta-regression analyses to explore potential sources of heterogeneity. Statistical significance was determined using a p-value threshold of <0.05.

Results

Search and screening results

A total of 485 studies were retrieved after the initial search across three databases, including 183 from PubMed, 243 from Scopus, and 59 from Web of Science. After 335 duplicate studies were detected and resolved, titles and abstracts of 293 articles were screened based on the eligibility criteria. A total of 212 studies which were initially considered eligible and were subjected to full-text evaluation. After full-text examination, 38 articles were eligible for inclusion [humans ($n = 21$), animals ($n = 7$), environment ($n = 2$), Interdisciplinary studies ($n = 8$)] (Fig. 1).

Characteristics of included studies

All included studies were published between 2011 and 2024, with most of them (63.2%) having been conducted between 2015 and 2019. Studies that met the inclusion criteria were from only 9 out of 16 countries in West Africa. Nigeria had the highest number of individual studies ($n = 17$), followed by Ghana ($n = 13$), Senegal ($n = 3$), and Burkina Faso ($n = 2$). The remaining countries, Guinea-Bissau, Guinea, Niger, Gambia, and Benin, had one study each (Fig. 2) (Supplementary data 1; Table 3).

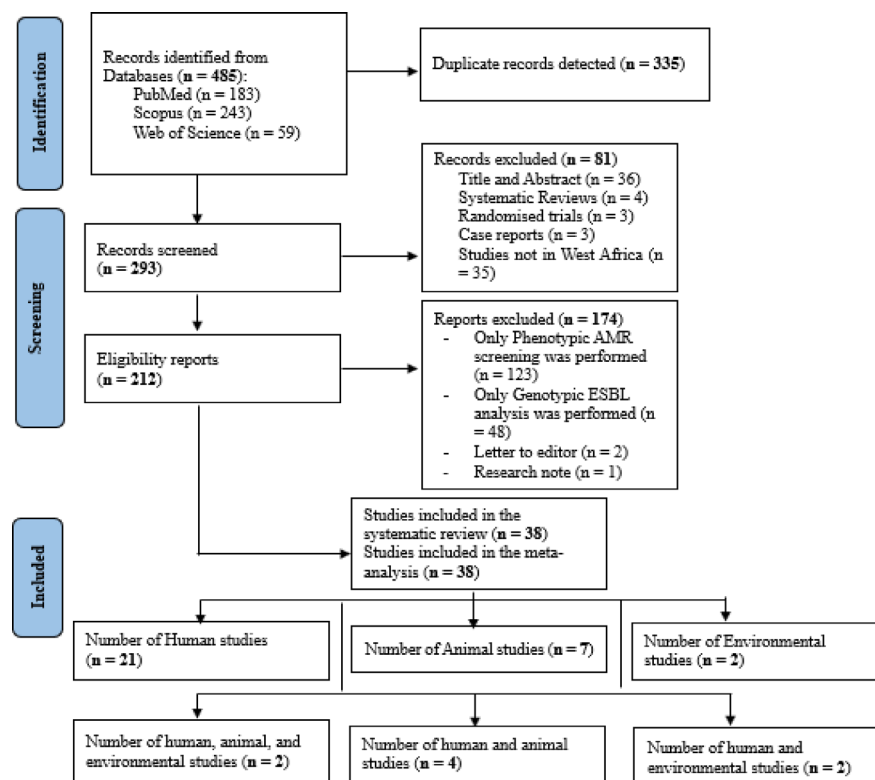


Fig. 1. PRISMA flowchart of study selection.

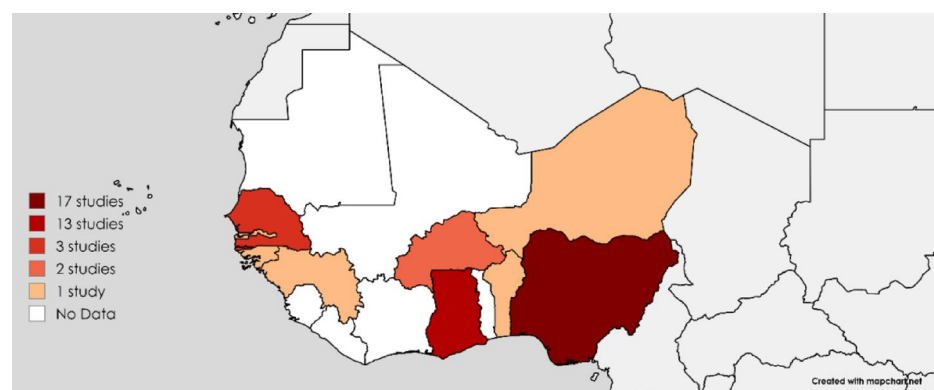


Fig. 2. Distribution of studies conducted in West Africa.

The number of confirmed ESBL-positive isolates across the studies ranged from 1 to 1937 and comprised 25 different bacterial isolates across the nine West African countries, encompassing various populations, settings, and sample types (Fig. 3) (Supplementary data 2; Table 1).

A majority of the studies were conducted on humans (76.3%, $n = 29$), followed by animals (34.3%, $n = 13$) and the environment (15.6%, $n = 6$) (Supplementary data 1; Table 3). Most studies isolated ESBL-positive bacteria from hospital-based settings (68.2%, $n = 24$), followed by communities (15.8%, $n = 6$), farms (10.5%, $n = 4$), and markets (2.6%, $n = 1$). Some isolated the bacteria from a combination of sources, such as markets and farms (7.9%, $n = 3$) and communities and hospitals (5.3%, $n = 2$). Most studies isolated the bacteria from two major sample types: clinical specimens (39.5%, $n = 15$) and faeces (31.6%, $n = 12$) (Supplementary data 1; Table 3).

Among the 25 ESBL-positive isolates, 18 were identified as *Enterobacteriaceae* and 7 were classified as non-*Enterobacteriaceae* (Supplementary data 2; Table 1).

The most commonly used ESBL diagnostic methods were EUCAST disk diffusion (18 studies), Double-Disk Synergy Test (DDST) (18 studies), Whole Genome Sequencing (WGS) (30 studies), and Multilocus Sequence Typing (MLST) (34 studies) (Supplementary data 1; Table 3).

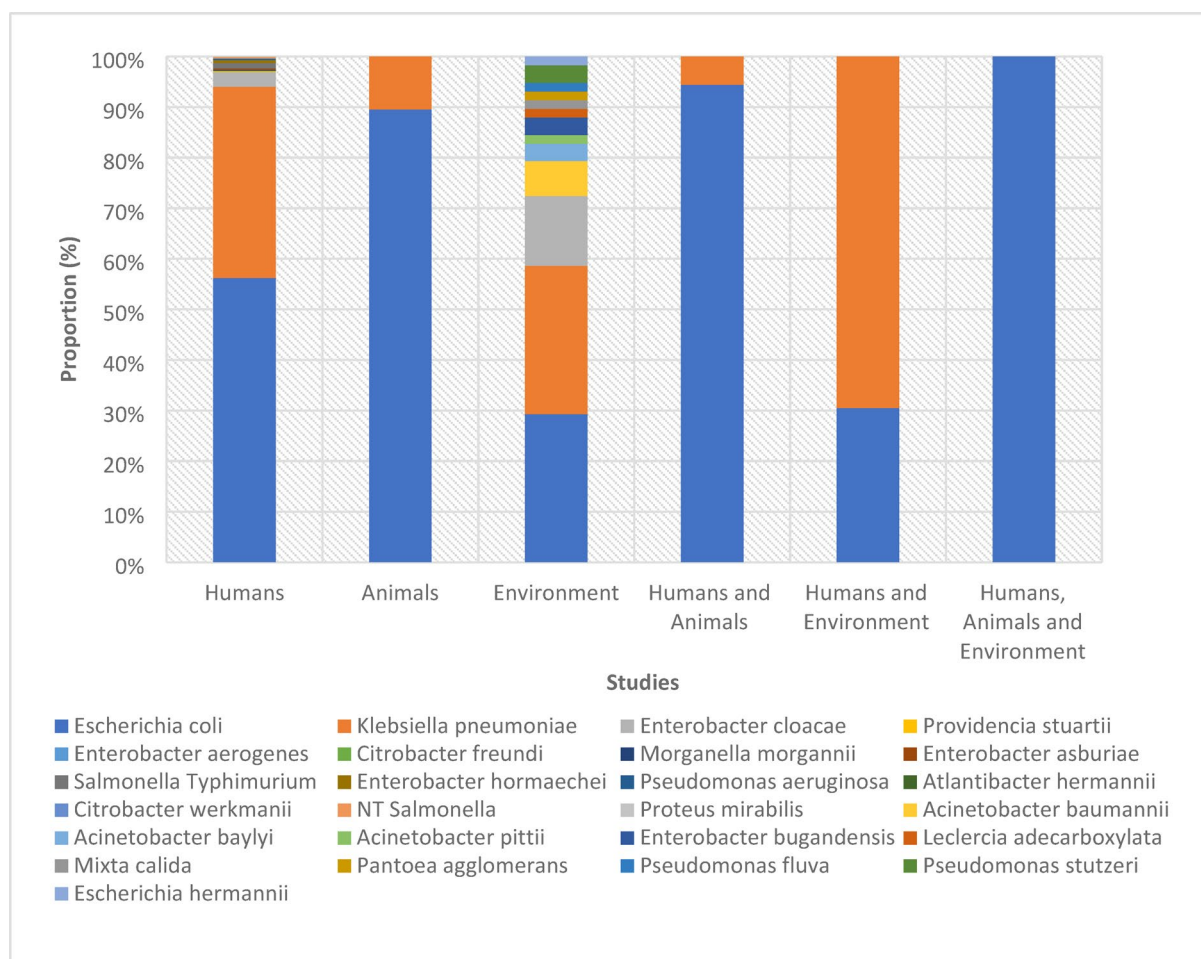


Fig. 3. ESBL positive bacterial isolates from different studies.

ESBL-producing bacterial species across human, animal, and environmental sources in West Africa

E. coli and *K. pneumoniae* accounted for 64% and 31% of the ESBL-positive isolates, respectively. Other *Enterobacteriaceae* species collectively comprised 4% of the isolates, whereas non-*Enterobacteriaceae* constituted 1% of the total isolates (Supplementary data 2; Table 1A).

ESBL genes and clones

A total of 171 ESBL genes were identified, with *bla*CTX-M being the most predominant gene (70.8%) across humans, animals, and the environment. The most frequent variant, *bla*CTX-M-15 (31.6%), was detected in all three sources, with greater occurrence in humans (21.1%) than in animals (7.6%) or the environment (2.9%) (Fig. 4) (Supplementary data 2; Table 2–10).

Multilocus sequence typing (MLST) was performed on 7 of 25 isolates, identifying 153 unique sequence types (STs) (Supplementary data 2; Table 14). Among these, Nigeria accounted for 133 clones from 5 isolates, with ST10, ST131, and ST410 being the most frequently observed. Ghana contributed 139 clones from 3 isolates, predominantly featuring ST131 and ST617, whereas 64 clones were identified from 4 isolates in other countries (Supplementary data 2; Table 11–13).

In terms of species-specific observations, *E. coli* exhibited considerable differences in sequence types among the different sources. In humans, ST131, ST10, ST410, and ST617 were prominent, whereas animals predominantly exhibited ST48, ST10, and ST38. Environmental samples were marked by the prevalence of ST10, ST58, and ST155. For *K. pneumoniae*, the predominant human STs were ST17, ST36, ST530, ST15, and ST14. In animals, ST307 was observed, and ST152 was most frequently observed in the environment. Non-*Enterobacteriaceae* displayed notable source-specific trends, with *Pseudomonas aeruginosa* ST1469 and ST235 linked to human samples and *Acinetobacter baumannii* ST109, ST1, ST78, ST132, and ST1136 linked to environmental samples (Supplementary data 2; Table 11–13).

Clonal distribution analysis showed that 71 clones were unique to humans, 27 to animals, and 14 to the environment. The number of shared clones between humans and animals was 25, between humans and the environment was 4, and between animals and the environment was 5. Notably, seven clones including ST10,

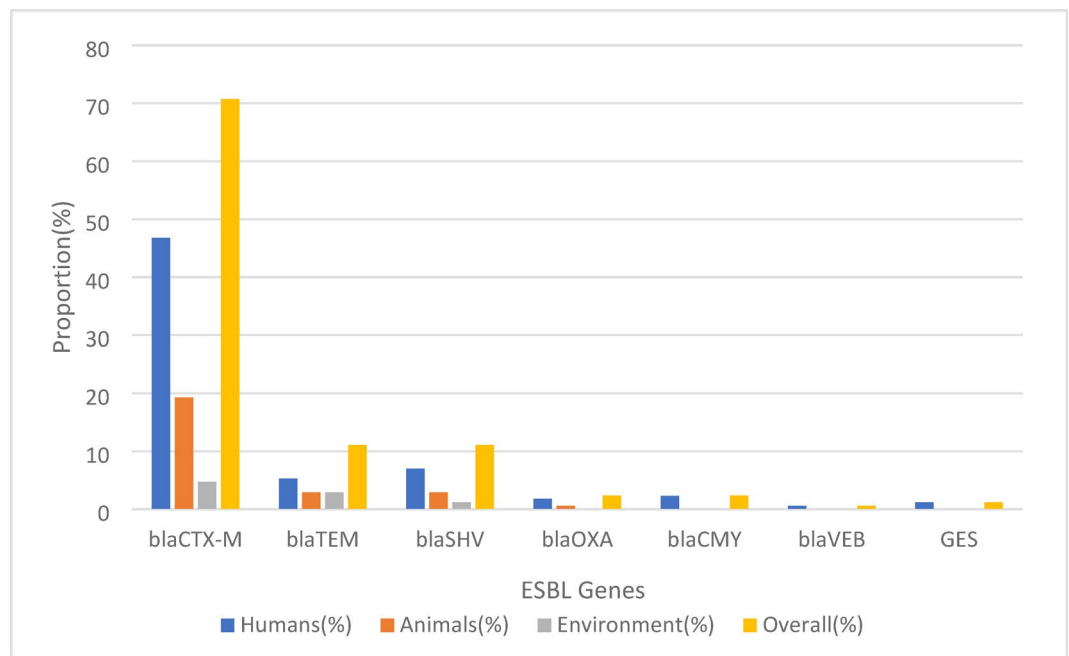


Fig. 4. Prevalence of ESBL genes across human, animal, and environmental sources.

ST410, ST58, ST155, ST4684, ST2178, and ST37 were detected predominantly in *E.coli* across all three sources. ST37 was also observed in *K. pneumoniae* (Fig. 5) (Supplementary data 2; Tables 11–14; Fig. 1).

Potential transmission routes of ESBL clones

Data from 7 of 38 studies (18.4%) were analysed for possible transmission routes of ESBL clones across human, animal and environmental sources. These studies used a combination of sources to detect the prevalence of ESBL-producing bacteria in hospitals, markets, and farms (Supplementary data 1; Table 3). These studies included Humans-Animals, Humans-Environment, and Humans-Animals-Environment studies (Table 1). Although the studies did not clearly state the transmission routes, they acknowledged that because of the common ESBL clones detected, the transmission routes were possible.

Antimicrobial resistance patterns

An analysis of 24 studies covering 1215/1937 ESBL-producing bacterial isolates revealed significant resistance patterns against non-beta-lactam antibiotics (Supplementary data 1; Table 4) (Supplementary data 2; Table 15–15A). The results show that bacteria from humans are much more resistant than those from animals and the environment. Phenotypically, the bacteria were particularly resistant to commonly prescribed antibiotics such as ciprofloxacin (71%), gentamicin (58%), and trimethoprim-sulfamethoxazole (53%). Resistance to tobramycin (39%) and tetracycline/doxycycline (28%) was moderate, whereas nalidixic acid (17%), imipenem/meropenem (16.7%), and chloramphenicol (14.1%) showed lower resistance (Fig. 6).

Co-resistance genes

The ESBL-producing bacterial isolates also exhibited the genotypic presence of co-resistance genes. Aminoglycoside and fluoroquinolone/quinolone resistance genes were the most frequently reported, each appearing in 15 studies, with *aac(6′)-Ib-cr* identified as the predominant resistance gene. Folate pathway antagonists’ resistance genes were reported in 12 studies, where *dfrA14* was the most common resistance gene. This was followed by sulfonamides (11 studies), where *sul2* was the most common gene, and tetracyclines (10 studies), with *tet(A)* as the predominant resistance gene. Phenicol was reported in 9 studies, with *catA1* being the most prevalent gene. Finally, carbapenem resistance genes were documented in 8 studies, with *blaNDM*, *blaOXA-48*, and *blaOXA-181* identified as the predominant resistance genes. Additionally, five studies reported mutations within the quinolone resistance-determining regions (QRDR) of *gyrA*, *parC*, *parE*, and *parC* (Table 2). (Other reported co-resistance genes are present in the supplementary data 2; Table 16).

Mobile genetic elements

A total of 22 studies performed mobilome analyses to identify the plasmid types carrying ESBL and other co-resistance genes. IncF plasmids emerged as the most prevalent, found across human, animal, and environmental sources, with an overall prevalence of 50.0%. Col plasmids were the second most common, accounting for 16.4%. Other plasmid types, including IncH, IncQ, and IncI, were detected at lower frequencies, with prevalence rates ranging from ~4.0% to ~7.0%. (Fig. 7) (Supplementary data 2; Table 17–17A).

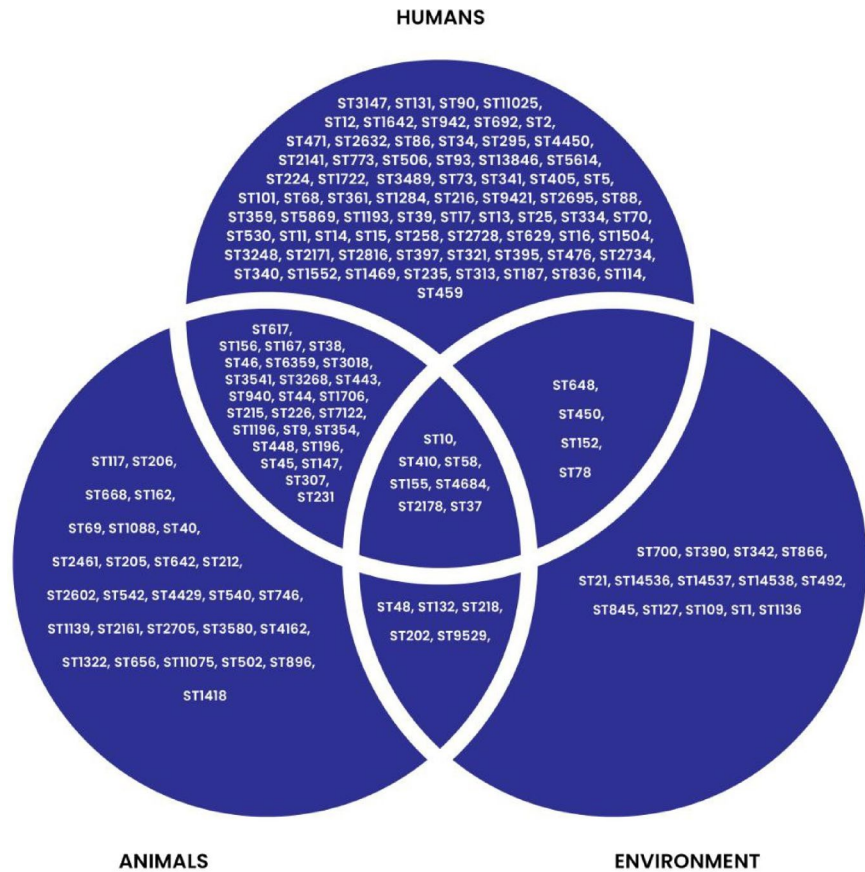


Fig. 5. Clonal distribution across West Africa.

Interface	Number of studies	Common ESBL clones detected
Human-animal	3	ST38, ST6359, ST58, ST3018, ST3541, ST9, ST44, ST443, ST231, ST10, and ST226
Human-environment	2	ST648, and ST37
Human-animal-environment	2	ST1196, ST10, ST155, ST48, ST4684, ST2178, ST58, ST226, ST7122, ST46, and ST9529

Table 1. Transmission interfaces and common ESBL clones.

Meta-analysis

Prevalence of ESBL

The overall pooled prevalence of ESBL in West Africa was 16.8% (95% CI [12.1; 22.1]. Nigeria and Ghana reported the highest prevalence at 18.3% and 17.3%, respectively, while Burkina Faso and Senegal had low prevalences at 6.0% and 1.2%, respectively (Fig. 8).

Humans had the highest prevalence at 20.4%, followed by animals at 12.8% and the environment at 9.3% (Fig. 9). Among settings, market-based environments recorded the highest prevalence at 23.6%, followed by hospitals at 21.8% and community-based settings at 7.5% (Supplementary data 1; Fig. 1).

Prevalence of ESBL-producing E. coli and K. pneumoniae

The study explored ESBL-producing *E. coli* and *K. pneumoniae* in 34 of 38 and 20 of 38 studies, respectively, across humans, animals, and the environment (Supplementary data 2; Tables 18-23). The prevalence of ESBL *E. coli* was 15.9% (95% CI: [9.8; 23.1], 26 studies) in humans, 15.0% (95% CI: [6.5; 26.0], 11 studies) in animals, and 6.1% (95% CI: [1.8; 12.4], 5 studies) in the environment (Table 3). The prevalence of ESBL *K. pneumoniae* was 8.2% (95% CI: [3.9; 13.8], 16 studies) in humans, 2.0% (95% CI: [0.4; 4.3], 4 studies) in animals, and 6.6% (95% CI: [4.4; 9.2], 2 studies) in the environment (Table 4).

Subgroup analysis The subgroup analysis of pooled prevalence estimates for ESBL-positive *E. coli* and *K. pneumoniae* across humans, animals, and the environment revealed distinct patterns based on settings and sample types.

For *E. coli*, human studies showed the highest prevalence in hospital-based settings (17.6%) and faecal samples (23.2%). Among animals, faecal samples have the highest prevalence (27.0%), and farm-based settings

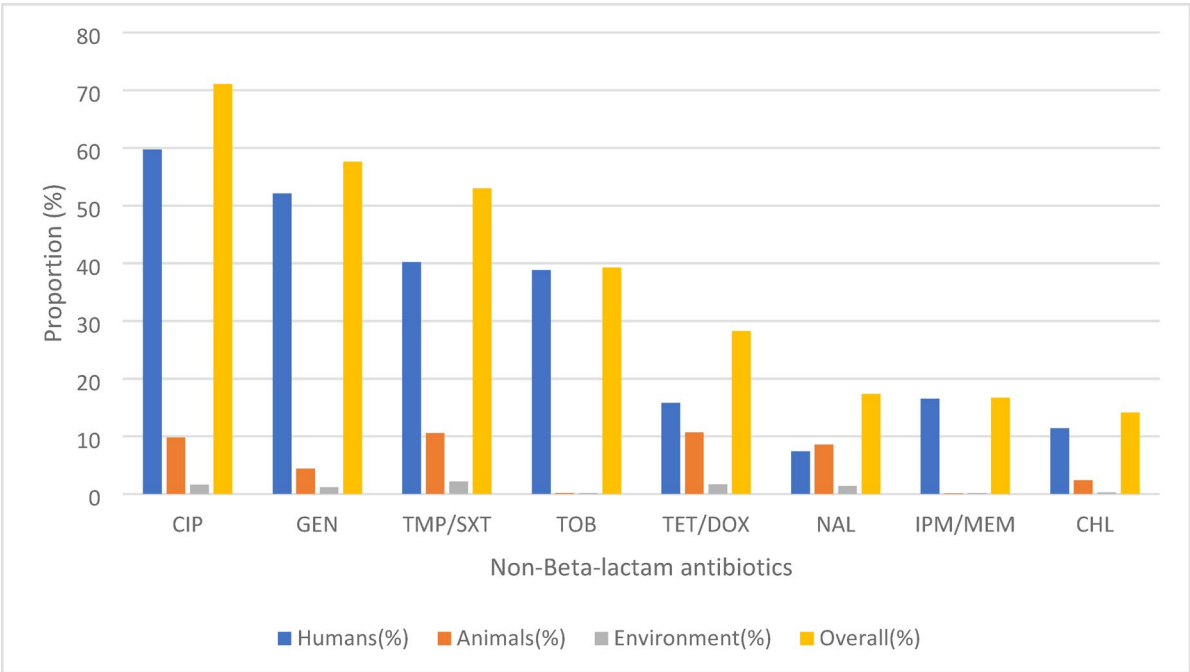


Fig. 6. Antimicrobial resistance of ESBL bacterial isolates to non-beta-lactam agents.

Antibiotic class	No of studies	Resistance genes (frequency)
Fluoroquinolone/quinolone	15	<i>aac(6′)-Ib-cr</i> (12), <i>qnrS1</i> (7), <i>qepA4</i> (4), <i>qnrB</i> (2), <i>qnrB1</i> (2)
Aminoglycoside	15	<i>aac(6′)-Ib-cr</i> (8), <i>aph(6)-Id</i> (8), <i>aadA1</i> (6), <i>aadA5</i> (6), <i>aac(3)-IId</i> (5)
Folate pathway antagonist	12	<i>dfrA14</i> (7), <i>dfrA1</i> (6), <i>dfrA12</i> (6), <i>dfrA17</i> (6), <i>dfrA7</i> (4),
Sulfonamide	11	<i>sul2</i> (11), <i>sul1</i> (9), <i>sul3</i> (5), <i>sul3-v1</i> (1)
Tetracycline	10	<i>tet(A)</i> (8), <i>tet(B)</i> (7), <i>tet(M)</i> (5), <i>tet(L)</i> (1), <i>tet(R)</i> (1)
Phenicol	9	<i>catA1</i> (6), <i>catB3</i> (5), <i>flor</i> (4), <i>catA2</i> (3), <i>cmlA1</i> (3)
Carbapenem	8	<i>blaNDM</i> (3), <i>blaOXA-48</i> (3), <i>blaOXA-181</i> (3), <i>blaNDM-1</i> (2), <i>blaKPC</i> (1), <i>blaNDM-5</i> (1), <i>blaNDM-7</i> (1), <i>blaVIM</i> (1)
Mutations		
QRDR	5	<i>gyrA</i> (6), <i>parC</i> (5), <i>parE</i> (3)

Table 2. Co-resistance genes in ESBL isolates according to antibiotic classes. Mutations in QRDR. QRDR – Quinolone Resistance Determining Region.

(21.7%) were particularly affected. In environmental studies, the abattoir environment has the highest rate (22.4%), whereas hospital-based environments have much lower rates (1.7%) (Table 3).

Clinical specimens: urine, ear swab, sputum, blood culture, eye swab, wound swab, urethral swab, endocervical swab, stool, high vaginal swab, nasal swab, gastric lavage, pus, tracheal aspirates, semen, axilla, groin, perianal region, cerebrospinal fluid, lower respiratory tracts, urinary tract infections (UTIs), ascitic fluid.

Patient environment: swabs of high- and low-touch areas, beds, taps, and drip stand.

NICU environment: swabs of incubator doors, cots, nurse trolley handle, weighing scales, tables, and desks.

For *K. pneumoniae*, human studies revealed a dominance of hospital-based settings (10.4%) and clinical specimens (9.5%) prevalence, with wound swabs reporting the highest prevalence in individual samples (57.1%). In animals (4 studies, 2.0%), community-based settings (2.5%), and rectal swabs (10.3%) showed higher prevalence rates. Environmental studies for *K. pneumoniae* (6.6% prevalence) indicated that patient environments (7.4%) had a slightly higher prevalence than NICU environments (5.8%) (Table 4).

Heterogeneity was significant in human studies on both isolates ($I^2 \sim 99\%$), indicating variability across settings and sample types, whereas environmental studies on *K. pneumoniae* showed minimal variability ($I^2 = 0\%$). This subgroup analysis indicated significant differences in the prevalence of ESBL *E. coli* and *K. pneumoniae* depending on the host/source, settings, and sample type.

Heterogeneity and publication bias

The funnel plot illustrates a publication bias, which was confirmed by Egger’s regression test ($p < 0.0001$) (Supplementary data 1; Fig. 2). Estimated prevalences were significantly heterogeneous across countries,

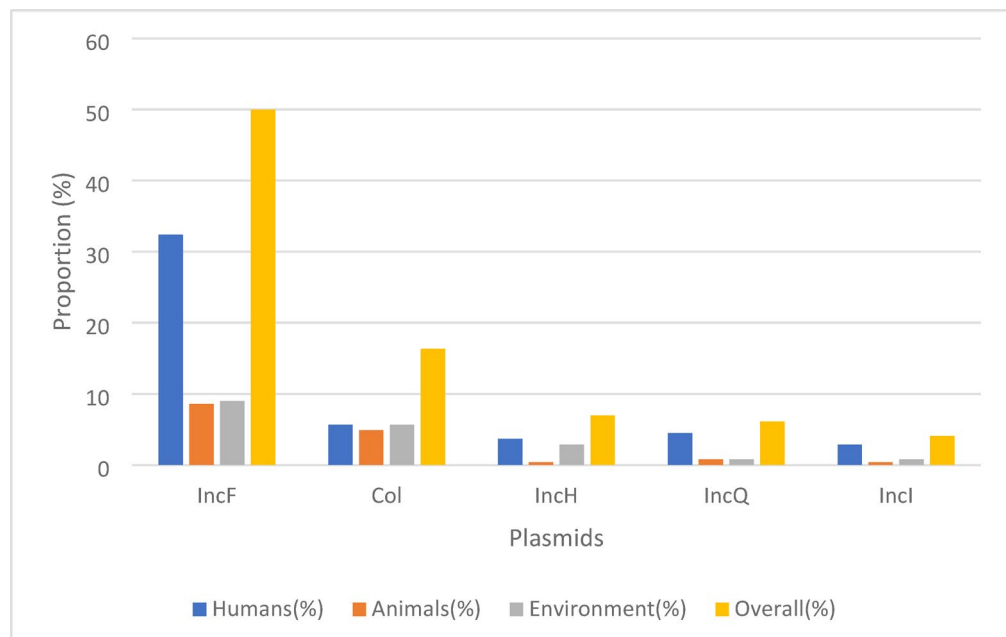


Fig. 7. Prevalence of ESBL conjugative plasmids across human, animal, and environmental sources.

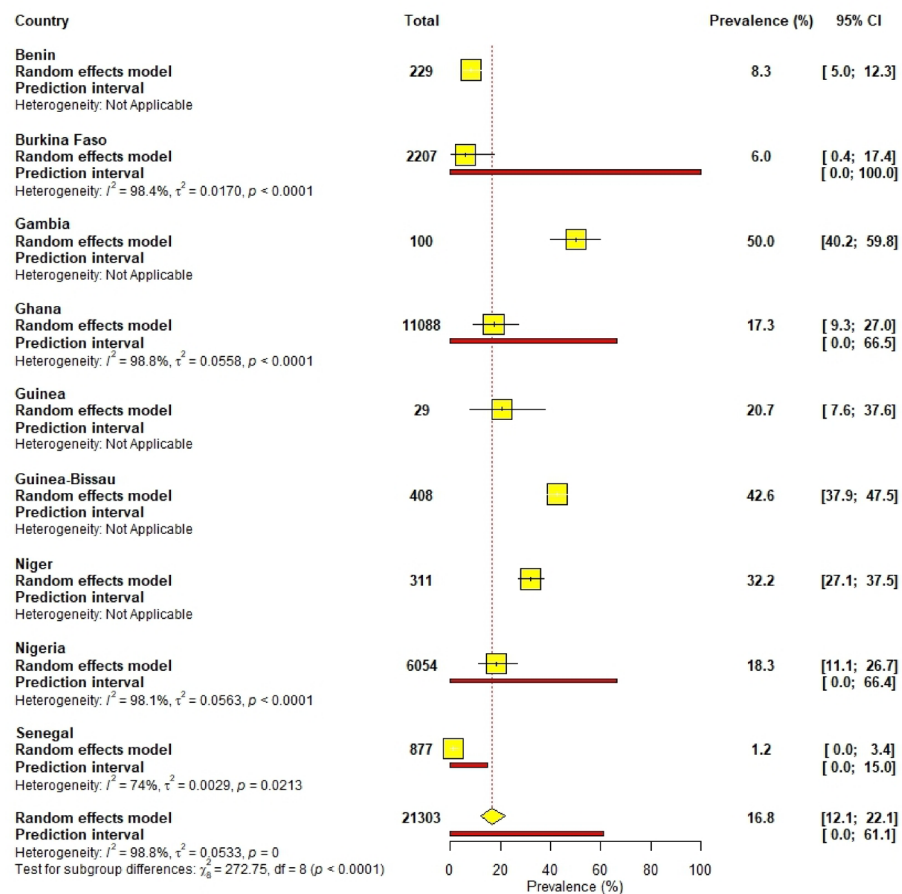


Fig. 8. Pooled prevalence of ESBL-producing bacteria according to countries in West Africa.

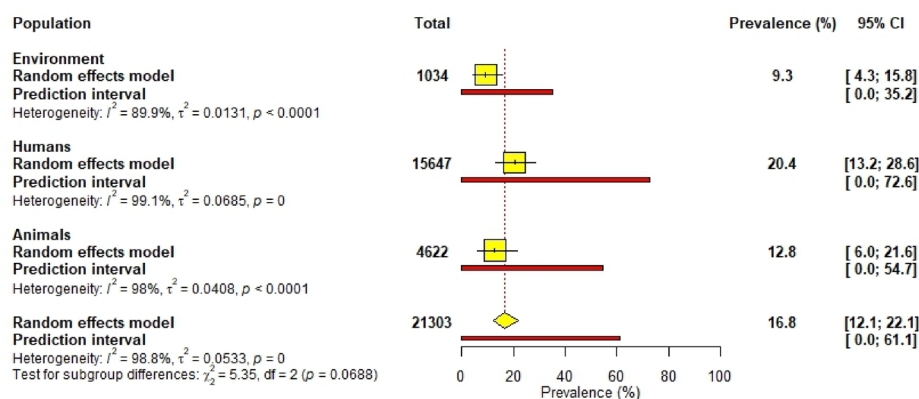


Fig. 9. Pooled prevalence of ESBL-producing bacteria across human, animal, and environmental sources.

populations, and settings ($H > 1$ and $I^2 \geq 75\%$). The overall prevalence also showed significant heterogeneity ($H = 8.98$ [8.51; 9.49] and $I = 98.8\%$ [98.6%; 98.9%]).

Sensitivity analysis and meta-regression

The impact of each study on the pooled prevalence of ESBL-producing bacteria was assessed using a leave-one-out sensitivity analysis. The sensitivity analysis revealed that excluding individual studies had a minimal impact on the pooled prevalence of 16.8%, with most exclusions resulting in prevalence values ranging from 16.0 to 17.4%. Only one study reported a slightly lower prevalence of 15.8%. These findings indicate that the pooled prevalence was stable and not overly influenced by any single study, demonstrating the robustness of the meta-analysis results (Supplementary data 1; Table 5).

A random effects meta-regression analysis was used to assess the effect of the three study characteristics on the observed wide variations in effect sizes in this study. Country, population, and settings were used as covariates. Gambia had a minimally significant negative association with heterogeneity. The remaining covariates did not significantly affect heterogeneity (Table 5).

Discussion

ESBL prevalence in West Africa is a growing concern, emphasising the need for a better understanding of its transmission across different populations and environments. While global and regional studies have documented the prevalence of ESBL, there is still a lack of detailed analyses of the movements of ESBL-producing clones between humans, animals, and the environment. This systematic review and meta-analysis filled this gap by synthesising findings from West African studies to provide essential evidence for addressing these clones and their genetic elements.

The overall prevalence of ESBL-producing bacteria in West Africa was 16.8%, which is notably lower than that reported in other countries, particularly in subregions of Africa where extreme pooled prevalences have been observed. For instance, countries such as Egypt, Ethiopia, and China have reported ESBL-producing bacterial prevalence rates exceeding 50%^{12,15,19}. Additionally, other subregions in Africa, specifically East, Central, and Southern Africa, have recorded pooled prevalence rates greater than 30%²⁰.

The predominant species observed in this review associated with ESBL were from the *Enterobacteriaceae* family, particularly *E. coli* and *K. pneumoniae*. These species have emerged as some of the most problematic *Enterobacteriaceae* species globally, associated with various infections that result in prolonged hospital stays, costly treatments, and increased mortality rates. This aligns with the findings of an epidemiological study by Quan et al., who noted a global increase in ESBL prevalence in *E. coli* and *K. pneumoniae*¹¹. Non-*Enterobacteriaceae* species such as *Pseudomonas aeruginosa* and *Acinetobacter baumannii* were also identified as contributors to ESBL prevalence, albeit at a very low prevalence (1%). This result is in contrast with the findings of Rameshkumar et al., who reported that *P. aeruginosa* accounted for 38% of ESBL-related cases, surpassing that of *E. coli* at 16.6%²¹. An epidemiological study in Egypt confirmed the ESBL-producing nature of *A. baumannii* in clinical settings²².

The acquisition of ESBL genes by non-*Enterobacteriaceae* may be attributed to their conjugative abilities, with horizontal gene transfer playing a crucial role in the dissemination of these genes. Notably, *A. baumannii* was observed in one of the environmental sources, suggesting a potential pathway for acquiring ESBL genes.

The emergence of ESBL-producing strains has created a crisis that renders traditional treatments ineffective against common infections. Initially, common beta-lactam antibiotics such as amoxicillin/clavulanic acid, ampicillin, cefotaxime, cefepime, and ceftazidime were employed; however, they have been proven ineffective due to the ability of ESBL enzymes to hydrolyse the beta-lactam rings of penicillins and cephalosporins.

Natural resistance is a plausible explanation for this phenomenon²³. This refers to the intrinsic ability of certain bacteria to resist specific antibiotics, allowing them to proliferate even at high concentrations that the human body can tolerate. Acquired resistance also plays a role, particularly in non-*Enterobacteriaceae* that

Organism	Number of studies	Pooled estimates			Measure of Heterogeneity			
		Sample size	ESBL positive	Prevalence (%)	I ² (%)	p-value	Q-value	95% CI
Overall human								
<i>E. coli</i>	26	14,583	829	15.9	98.9	0.00	2210.68	[9.8; 23.1]
Human settings								
Hospital based	21	13,773	749	17.6	99	0.00	2073.76	[10.4; 26.1]
Market based	1	118	40	33.9	–	–	0.00	[25.4; 43.2]
Market- and farm-based	2	342	32	9.3	2.1	0.3123	1.02	[6.4; 12.7]
Community- and hospital-based	1	250	4	1.6	–	–	0.00	[0.4; 4.0]
Community based	1	100	4	4.0	–	–	0.00	[1.3; 10.1]
Human samples								
Faeces	6	806	162	23.2	95.4	<0.0001	108.07	[10.6; 38.7]
Blood cultures	2	7240	40	15.7	99.4	<0.0001	161.85	[0.0; 84.9]
Clinical specimens	13	5590	491	13.3	98.5	<0.0001	816.17	[6.3; 22.3]
Wound swabs	2	236	22	18.5	81.1	0.0214	5.29	[0.0; 60.4]
Rectal swabs	1	408	83	20.3	–	–	0.00	[16.6; 24.6]
Rectal and hand swabs	1	83	13	15.7	–	–	0.00	[8.9; 25.5]
Hand swabs	1	220	18	8.2	–	–	0.00	[4.9; 12.6]
Overall animals								
<i>E. coli</i>	11	4059	372	15.0	98.2	<0.0001	562.06	[6.5; 26.0]
Animal settings								
Farm based	3	504	85	21.7	93.7	<0.0001	31.51	[5.6; 43.6]
Community based	4	2229	72	11.7	98.5	<0.0001	195.02	[0.0; 40.4]
Market- and farm-based	3	1054	171	14.1	74.1	0.0211	7.72	[9.4; 19.6]
Market based	1	272	44	16.2	–	–	0.00	[12.1; 21.2]
Animal sample type								
Caecal content	2	626	81	13.1	77.3	0.0358	4.41	[8.0; 19.2]
Intestinal culture	1	2000	15	0.8	–	–	0.00	[0.2; 1.0]
Cloacal swabs and meat	1	188	20	10.6	–	–	0.00	[5.5; 15.0]
Rectal swabs	1	29	3	10.3	–	–	0.00	[3.3; 27.9]
Faeces	5	1116	249	27.0	93	<0.0001	56.85	[14.9; 41.1]
Whole cockroach homogenates	1	100	4	4.0	–	–	0.00	[1.5; 10.3]
Overall environment								
<i>E. coli</i>	5	843	45	6.1	89.7	<0.0001	39.01	[1.8; 12.4]
Environmental settings								
Hospital based	2	439	8	1.7	58	0.1227	2.38	[0.3; 4.2]
Market based	1	58	13	22.4	–	–	0.00	[12.9; 35.4]
Market- and farm-based	1	196	9	4.6	–	–	0.00	[2.1; 8.5]
Community- and hospital-based	1	150	15	10.0	–	–	0.00	[5.8; 16.0]
Environment sample type								
Patient environment	2	439	8	1.7	58	0.1227	2.38	[0.3; 4.2]
Abattoir environment	1	58	13	22.4	–	–	0.00	[12.9; 35.3]
The farm and market environments	1	196	9	4.6	–	–	0.00	[2.1; 8.5]
Water and sediments	1	150	15	10.0	–	–	0.00	[5.8; 16.0]

Table 3. Prevalence of ESBL *E. coli* in humans, animals, and the environment. 95% CI, 95% confidence interval. I^2 describes the proportion of total variation in the prevalence of ESBL isolates due to heterogeneity; a value > 50% indicates the presence of heterogeneity. Q-value shows how much the studies differ; a high Q with $p < 0.05$ means the differences are significant. p-value indicates whether the results are meaningful; $p < 0.05$ suggests that the differences do not occur simply by chance.

circulate among humans, animals, and the environment. Resistance can arise through chromosomal mutations, often due to antibiotic overuse or acquired resistant plasmids or insertion sequences²³.

Beta-lactam antibiotics have been used to confirm the presence of ESBL-producing bacteria as part of the identification of alternative treatment options. Our findings indicate that commonly used antibiotics for this purpose include those previously mentioned²⁴. However, alternative treatment options employed against these bacteria include ciprofloxacin, nalidixic acid, chloramphenicol, imipenem/meropenem, tobramycin, gentamicin, tetracycline/doxycycline, and trimethoprim-sulfamethoxazole. Unfortunately, due to co-resistance, these

Organism	Number of studies	Pooled estimates			Measures of Heterogeneity			
		Sample size	ESBL positive	Prevalence (%)	I ²	p-value	Q-value	95% CI
Overall human								
<i>K. pneumoniae</i>	17	14,052	551	8.2	98.7	<0.0001	1250.39	[3.9; 13.8]
Human settings								
Hospital based	14	13,366	537	10.4	98.9	<0.0001	1235.33	[4.9; 17.5]
Community- and hospital-based	1	250	12	4.8	–	–	0.00	[2.7; 8.4]
Community based	2	436	2	0.3	0.0	0.3365	0.92	[0.0; 1.3]
Human samples								
Clinical specimens	11	5718	392	9.5	98.7	<0.0001	741.15	[3.6; 7.7]
Wound swabs	1	7	4	57.1	–	–	0.00	[22.0; 85.8]
Blood cultures	1	7172	34	0.5	–	–	0.00	[0.2; 0.5]
Rectal swabs	2	744	92	7.5	99.3	<0.0001	134.48	[0.0; 41.3]
Faeces	2	411	29	4.4	90.6	0.0011	10.58	[0.0; 15.0]
Overall animals								
<i>K. pneumoniae</i>	4	692	17	2.0	45.0	0.1409	5.46	[0.4; 4.3]
Animal settings								
Community based	3	592	16	2.5	57.8	0.0937	4.74	[0.3; 6.2]
Farm based	1	100	1	1.0	–	–	0.00	[0.2; 5.8]
Animal sample types								
Rectal swabs	1	29	3	10.3	–	–	0.00	[2.2; 27.4]
Faeces	1	100	1	1.0	–	–	0.00	[0.0; 5.4]
Whole cockroach homogenates	1	100	1	1.0	–	–	0.00	[0.0; 5.4]
Faeces and termites	1	463	12	2.6	–	–	0.00	[1.3; 4.5]
Overall environment								
<i>K. pneumoniae</i>	2	422	28	6.6	0.0	0.5256	0.4	[4.4; 9.2]
Environmental settings								
Hospital based	2	422	28	6.6	0.0	0.5256	0.4	[4.4; 9.2]
Environmental sample types								
Patient environment	1	231	17	7.4	–	–	0.00	[4.3; 11.5]
NICU environment	1	191	11	5.8	–	–	0.00	[2.9; 10.1]

Table 4. Prevalence of ESBL *K. pneumoniae* in humans, animals, and the environment. 95% CI, 95% confidence interval. I^2 describes the proportion of total variation in the prevalence of ESBL isolates due to heterogeneity; a value > 50% indicates the presence of heterogeneity. Q-value shows how much the studies differ; a high Q with $p < 0.05$ means the differences are significant. p-value indicates whether the results are meaningful; $p < 0.05$ suggests that the differences do not occur simply by chance.

bacteria can acquire additional genes through horizontal gene transfer, enabling them to resist and neutralise these antibacterial agents.

In this study, the widespread presence of co-resistance genes in ESBL-producing bacteria across various sources was confirmed, with the findings being consistent with that of previous studies. For instance, Tacao et al. reported high levels of co-resistance to non-beta-lactam antibiotics in aquatic environments²⁵, while Athanasakopoulou et al. observed similar patterns in animal isolates²⁶. Moreover, Yang et al. identified co-resistance genes in clinical samples²⁷. A common feature found across these studies was the detection of fluoroquinolone/quinolone resistance genes, indicating their high prevalence in ESBL-producing bacteria. Similarly, in this analysis, fluoroquinolone/quinolone and aminoglycoside resistance genes were the most frequently encountered, with “*aac(6′)-Ib-cr*” emerging as a dominant genetic determinant. Notably, five studies reported mutations in genes encoding the quinolone resistance determining regions (QRDR) *gyrA*, *parC*, and *parE*, which are key contributors to fluoroquinolone/quinolone resistance²⁸.

Furthermore, the genes associated with ESBL-producing bacteria were predominantly *bla*CTX-M gene (70.8%), with its most troublesome variant being *bla*CTX-M-15 (31.6%). This finding aligns with a study conducted in China on the epidemiological characteristics of antimicrobial resistance in ESBL-producing bacteria, in which *bla*CTX-M was identified as the most prevalent ESBL gene¹⁵. Counter to our findings, their study identified another variant, *bla*CTX-M-14, as the most common but acknowledged the potential rise of *bla*CTX-M-15. Other genes, such as *bla*SHV, *bla*TEM, *bla*OXA, and *bla*VEB, although present at lower proportions in this study, have been recognised in other studies as highly prevalent under certain conditions^{29,30}. For instance, a systematic review by Ghaderi et al. identified high prevalence rates of *bla*SHV (37%) and *bla*TEM (51%) in clinical isolates in Iran³¹.

Interestingly, a study in the early 2000’s reported a novel ESBL-associated gene called Guiana-Extended-Spectrum (GES), which was isolated from nine countries, including French Guiana, Brazil, Portugal, Argentina,

Covariates	Estimate	Standard Error	z-value	p-value	95% CI. LB	95% CI. UB
Intercept	0.0999	0.3123	0.3201	0.7489	-0.5121	0.7120
Country						
Burkina Faso	-0.0470	0.2801	-0.1676	0.8669	-0.5960	0.7120
Gambia*	0.7965	0.3544	2.2479	0.0246	0.1020	1.4911
Ghana	0.2438	0.2390	1.0200	0.3077	-0.2246	0.7121
Guinea	0.4950	0.3627	1.3649	0.1723	-0.2158	1.2059
Guinea-Bissau	0.4164	0.3236	1.2868	0.1982	-0.2178	1.0505
Niger	0.3081	0.3238	0.9514	0.3414	-0.3266	0.9428
Nigeria	0.2365	0.2387	0.9908	0.3218	-0.3266	0.9428
Senegal	0.0209	0.2790	0.0750	0.9402	-0.5259	0.5678
Population						
Humans	0.0396	0.1294	0.3062	0.7595	-0.2140	0.2933
Environment	-0.1359	0.1518	-0.8953	0.3706	-0.4335	0.1617
Settings						
Hospital based	0.1559	0.1751	0.8902	0.3734	-0.1873	0.4990
Market based	0.2058	0.2191	0.9397	0.3474	-0.2235	0.6352
Community based	-0.1111	0.2216	-0.5014	0.6161	-0.5453	0.3232
Farm based	0.0611	0.2399	0.2546	0.7990	-0.4090	0.5312
Market- and farm-based	0.0069	0.2006	0.0345	0.9725	-0.3862	0.4001

Table 5. Meta-regression of factors affecting heterogeneity. 95% confidence interval (CI) LB – 95% confidence interval lower bound, 95% confidence interval (CI) UB – 95% confidence interval upper bound.

South Africa, Japan, Korea, Greece, and France. This gene consisted of nine variants identified at that time: GES-1 through GES-9. These genes were found in common enterobacteria, such as *E. coli*, *K. pneumoniae*, and *Enterobacter cloacae*, as well as non-enterobacteria like *Pseudomonas aeruginosa*, which were associated with GES-1, GES-2, GES-8, and GES-9³². Our findings indicate that this emerging family of ESBLs has been isolated in Senegal and Nigeria, with GES-1 and GES-9 being the major variants discovered in *Pseudomonas aeruginosa*. Although the emergence rate in West Africa is low at 1.2%, it raises concerns about the future prominence of ESBL-associated genes, potentially surpassing that of *bla*CTX-M-15.

Studies have shown that ESBL-producing bacteria are highly susceptible to imipenem/meropenem; thus, these antibiotics are recommended as a last line of defence against such infections^{33,34}. Despite our findings indicating a relatively low rate of resistance developing across West Africa (16.7%), concerns remain regarding the growing threat posed by these bacteria's ability to neutralise these antibiotics. A systematic review by Khademi et al. reported a pooled prevalence of ESBL-producing bacteria resistant to imipenem and meropenem of 16.6% and 16.2%, respectively, indicating that resistance to these drugs is gradually developing²⁴.

Market-based settings were observed to have a higher prevalence of ESBL (23.6%) compared with hospital-based settings (21.8%). Hospitals are reservoirs for various resistant bacterial strains because of antibiotic overuse; however, market-based environments have emerged as significant reservoirs for ESBL bacteria across West Africa because of the poor hygiene conditions prevalent in many markets. In a study conducted in Mumbai which assessed the prevalence of ESBL *E. coli* in fresh seafood from retail markets and found it to be as high as 71.58%, researchers indicated a clear link between poor hygiene practices and elevated prevalence rates³⁵. Another contributing factor could be improper antimicrobial agent disposal practices leading to resistance acquisition across various interfaces within the market.

This review shows significant public health concerns regarding the prevalence of ESBL-producing bacteria in humans (20.4%), animals (12.8%), and environmental sources (9.3%). Humans are primary reservoirs, especially in healthcare settings, while farm animals such as poultry and cattle, also serve as carriers for resistant strains due to antibiotic misuse in agriculture. Environmental reservoirs, particularly healthcare-associated water systems, and inadequately disposed waste further facilitate bacterial spread. One study focused on the interconnectedness between strains found among patients and within neonatal intensive care unit (NICU) environments and indicated that neonates are at heightened risk due to their vulnerability during early life stages³⁶; this is likely exacerbated by poor sanitation practices and excessive antibiotic use within healthcare facilities. Another study emphasised market/farm-based interactions involving humans, animals, and environmental factors where faecal-oral transmission routes are probable pathways for spreading these bacteria³⁷.

Current findings confirm the ongoing circulation patterns for ESBL-associated clones across human, animal, and environmental sources within West Africa; specifically identifying ST131, ST410, ST10, and ST617 as predominant strains in *E. coli* isolated from humans and animals, consistent with findings from multiple studies confirming their presence across various reservoirs^{38,39}. *E. coli* clones ST10 and ST410 were also observed in the environmental samples. These clones can be transferred from one source to another enabling their presence across the three interfaces.

At the molecular level, the spread of these ESBL clones is plausibly driven by mobile genetic elements (MGEs). These elements include integrons, transposons, and insertion sequences, which facilitate the transfer of

genetic material between bacteria through mechanisms such as horizontal gene transfer, including conjugation, transformation, and transduction⁴⁰. MGEs are often carried on extrachromosomal DNA, such as plasmids, which harbor foreign genes that can be integrated into the genomes of other bacteria in the environment. These acquired genes frequently encode traits that enhance bacterial survival, most notably antibiotic resistance.

Evidently, this review reported on the presence of notable plasmids harbouring ESBL genes as well as co-resistance genes in ESBL-producing bacteria. The most prevalent plasmids discovered across all sources were IncF and Col conjugative plasmids, with IncF predominating across all sources. Tacao et al. confirms the successful mobilization of ESBL-producers containing ESBL genotypes as well as other co-resistance genes by the IncF plasmid types²⁵. These findings were also confirmed by other studies indicating the presence of these conjugative plasmids in ESBL-producing bacteria^{29,41}.

At a broader level, the dissemination of ESBL-producing clones can be significantly influenced by cross-border livestock trade. This form of trade, while economically beneficial by ensuring a steady supply of livestock to meet demand, also facilitates the translocation of antimicrobial-resistant organisms. Countries with a higher prevalence of ESBL-producing strains can inadvertently introduce these clones into regions with lower prevalence through the importation of colonized or infected animals. A study conducted in Gabon documented cross-border trade involving poultry products imported from industrialized countries, rather than live animals. The study revealed a 23% prevalence of ESBL-producing *E. coli* in these imported poultry products⁴². This suggests that, in addition to cross-border livestock trade serving as a potential transmission route, animal products themselves can also harbor ESBL-producing bacteria.

Within livestock systems, particularly in intensive farming operations where antibiotics are frequently used for growth promotion and disease prevention⁴³, ESBL-producing bacteria can thrive and spread rapidly. The interconnectedness of humans, animals, and the environment further amplifies this risk. Humans can acquire resistant bacteria through direct contact with animals, the handling or consumption of contaminated animal products, or through environmental exposure⁴³.

Animal faeces, often rich in resistant bacteria, can contaminate soil and water systems when used as manure or through uncontrolled runoff^{44,45}. Contaminated water bodies not only affect aquatic life but also pose risks to public health, especially when such water is used for irrigation or as a source of drinking water. Agricultural products irrigated or washed with these waters can act as secondary vehicles for human exposure.

Throughout West Africa, widely circulating clones such as ST10, ST410, ST155, ST58, ST4684, ST2178, and ST37 identified majorly in *E. coli*, indicate the pressing need for multisectoral, genomic-based strategic interventions utilising One Health approaches. This is particularly important given that approximately 16.7% of the identified ESBL isolates demonstrated resistance to imipenem/meropenem. This finding starkly contrasts with earlier reports that indicated full susceptibility among all isolates, as noted in a previous systematic review conducted in Tanzania by Seni et al.³³. It is unfortunate yet necessary to acknowledge current realities regarding rising resistance trends among these pathogens; however, rates remain comparatively lower than those observed for other antibiotic classes, suggesting potential avenues still exist for implementing strategic interventions aimed at curbing further proliferation among existing populations harbouring resistant strains.

We acknowledge some limitations in this review. The final analysis included only a limited number of animal and environmental studies, primarily because of the scarcity of relevant articles. As a result, our examination of the transmission dynamics of circulating genotypes was limited to human-associated studies. Another limitation of this review is that, due to the inclusion criterion requiring both phenotypic and genotypic detection of ESBLs, only 9 out of 16 West African countries were represented in the final analysis. This may limit the generalizability of the findings to the broader region. Furthermore, the present antimicrobial resistance patterns do not encompass the full range of studies. Some studies have focused solely on testing the total number of isolates without specifically addressing ESBL-positive isolates. Moreover, some studies employed graphical representations to convey their results, lacking numerical indicators that would clarify the presence of resistant ESBL isolates.

Conclusion

This review confirms that West Africa is experiencing a growing antimicrobial resistance crisis, with ESBL-producing bacteria showing a prevalence of 16.8% across human, animal, and environmental sources. Genetic analysis indicates a concerning convergence of resistance traits, with ESBLs, particularly the *bla*CTX-M-15 gene, frequently co-occurring with fluoroquinolone/quinolone and aminoglycoside resistance genes. These resistance determinants are frequently disseminated through mobile IncF plasmids and widespread ESBL clonal lineages such as *E. coli* ST10, ST410, ST155, ST58, ST4684, ST2178, and ST37 across human, animal and environmental sources.

Addressing this issue requires a One Health approach, beginning with the establishment of regional genomic surveillance networks to detect resistance hotspots and track transmission routes across sectors and borders. Integrating ESBL monitoring into national health systems through standardized protocols for hospitals, farms, and food supply chains could help identify hidden reservoirs and guide targeted interventions. Central to this effort is antimicrobial stewardship, particularly in the farming sector, where stricter enforcement of antibiotic regulations and investment in alternatives like vaccines or probiotics could reduce selective pressure.

At the same time, future research should focus on uncovering the transmission pathways of ESBL-producing bacteria between humans, livestock, and the environment, while economic studies are needed to evaluate the often-overlooked costs of these infections on healthcare systems and the livestock sector, particularly in West Africa.

Data availability

The original contributions presented in the study are included in the article/Supplementary Materials 1, 2, and 3. Further inquiries can be directed to the corresponding author.

Received: 9 February 2025; Accepted: 4 July 2025

Published online: 13 August 2025

References

1. El Aila, N. A., Laham, A., Ayesh, B. M. & N. A. & Prevalence of extended spectrum beta lactamase and molecular detection of blaTEM, blaSHV and blaCTX-M genotypes among gram negative bacilli isolates from pediatric patient population in Gaza strip. *BMC Infect. Dis.* **23**, 99 (2023).
2. Ramatla, T. et al. One health perspective on prevalence of co-existing extended-spectrum β -lactamase (ESBL)-producing *Escherichia coli* and *Klebsiella pneumoniae*: a comprehensive systematic review and meta-analysis. *Ann. Clin. Microbiol. Antimicrob.* **22**, 88 (2023).
3. Negeri, A. A. et al. Antimicrobial resistance profiling and molecular epidemiological analysis of extended spectrum β -Lactamases produced by extraintestinal invasive *Escherichia coli* isolates from Ethiopia: the presence of international High-Risk clones ST131 and ST410 revealed. *Front. Microbiol.* **12**, (2021).
4. Kuralayanapalya, S. P. et al. Prevalence of extended-spectrum beta-lactamase producing bacteria from animal origin: A systematic review and meta-analysis report from India. *PLOS ONE*. **14**, e0221771 (2019).
5. Rock, C. & Donnenberg, M. S. Human pathogenic *Enterobacteriaceae*. In *Reference Module in Biomedical Sciences*. <https://doi.org/10.1016/B978-0-12-801238-3.00136-7> (Elsevier, 2014).
6. Hadi, H. A. et al. Prevalence and genetic characterization of clinically relevant extended-spectrum β -lactamase-producing enterobacteriales in the Gulf Cooperation Council countries. *Front. Antibiot.* **2**, (2023).
7. Yousefipour, M. et al. Bacteria producing extended spectrum β -lactamases (ESBLs) in hospitalized patients: prevalence, antimicrobial resistance pattern and its main determinants. *Iran. J. Pathol.* **14**, 61–67 (2019).
8. Tan, K. et al. Prevalence of the carbapenem-heteroresistant phenotype among ESBL-producing *Escherichia coli* and *Klebsiella pneumoniae* clinical isolates. *J. Antimicrob. Chemother.* **75**, 1506–1512 (2020).
9. Bezabih, Y. M. et al. The global prevalence and trend of human intestinal carriage of ESBL-producing *Escherichia coli* in the community. *J. Antimicrob. Chemother.* **76**, 22–29 (2021).
10. Coque, T. M., Baquero, F. & Cantón, R. Increasing prevalence of ESBL-producing *Enterobacteriaceae* in Europe. *Eurosurveillance* **13**, 19044 (2008).
11. Quan, J. et al. High prevalence of ESBL-producing *Escherichia coli* and *Klebsiella pneumoniae* in community-onset bloodstream infections in China. *J. Antimicrob. Chemother.* **72**, 273–280 (2017).
12. Teklu, D. S. et al. Extended-spectrum beta-lactamase production and multi-drug resistance among *Enterobacteriaceae* isolated in addis ababa, Ethiopia. *Antimicrob. Resist. Infect. Control.* **8**, 39 (2019).
13. Andrews, B., Joshi, S., Swaminathan, R., Sonawane, J. & Shetty, K. Prevalence of extended spectrum B-Lactamase (ESBL) producing Bacteria among the clinical samples in and around a tertiary care centre in nerul, navi mumbai, India. *Int. J. Curr. Microbiol. Appl. Sci.* **7**, 3402–3409 (2018).
14. Beyene, D., Bitew, A., Fantew, S., Mihret, A. & Evans, M. Multidrug-resistant profile and prevalence of extended spectrum β -lactamase and carbapenemase production in fermentative Gram-negative bacilli recovered from patients and specimens referred to National reference laboratory, Addis Ababa, Ethiopia. *PLOS ONE*. **14**, e0222911 (2019).
15. Xiao, Y. H. et al. Epidemiology and characteristics of antimicrobial resistance in China. *Drug Resist. Updat.* **14**, 236–250 (2011).
16. Schaufler, K. et al. Clonal spread and interspecies transmission of clinically relevant ESBL-producing *Escherichia coli* of ST410—another successful pandemic clone? *FEMS Microbiol. Ecol.* **92**, fiv155 (2016).
17. Page, M. J. et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. *Syst. Rev.* **10**, 89 (2021).
18. Moola, Z., Munn, S., Lisy, K., Riitano, D. & Tufanaru, C. Chapter 5: Systematic reviews of prevalence and incidence. In *JBIM Manual for Evidence Synthesis*, *JBIM* <https://doi.org/10.46658/JBIMES-20-06> (2020).
19. Azzam, A., Khaled, H., Samer, D. & Nageeb, W. M. Prevalence and molecular characterization of ESBL-producing *Enterobacteriaceae* in Egypt: a systematic review and meta-analysis of hospital and community-acquired infections. *Antimicrob. Resist. Infect. Control.* **13**, 1–14 (2024).
20. Onduru, O. G., Mkakosya, R. S., Aboud, S. & Rumisha, S. F. Genetic determinants of resistance among ESBL-producing *Enterobacteriaceae* in community and hospital settings in East, Central, and Southern Africa: A systematic review and meta-analysis of prevalence. *Can. J. Infect. Dis. Med. Microbiol.* **2021**, 5153237 (2021).
21. Rameshkumar, G. et al. Prevalence and antibacterial resistance patterns of extended-spectrum beta-lactamase producing Gram-negative bacteria isolated from ocular infections. *Indian J. Ophthalmol.* **64**, 303 (2016).
22. El-Baky, R. M. A., Farhan, S. M., Ibrahim, R. A., Mahran, K. M. & Hetta, H. F. Antimicrobial resistance pattern and molecular epidemiology of ESBL and MBL producing *Acinetobacter baumannii* isolated from hospitals in minia, Egypt. *Alex J. Med.* **56**, 4–13 (2020).
23. Harris, M., Fasolino, T., Ivankovic, D., Davis, N. J. & Brownlee, N. Genetic factors that contribute to antibiotic resistance through intrinsic and acquired bacterial genes in urinary tract infections. *Microorganisms* **11**, 1407 (2023).
24. Khademi, F., Vaez, H., Neyestani, Z. & Sahebkar, A. Prevalence of ESBL-producing *Enterobacter* species resistant to carbapenems in Iran: a systematic review and meta-analysis. *Int. J. Microbiol.* **2022**, 8367365 (2022).
25. Tacão, M., Moura, A., Correia, A. & Henriques, I. Co-resistance to different classes of antibiotics among ESBL-producers from aquatic systems. *Water Res.* **48**, 100–107 (2014).
26. Athanasakopoulou, Z. et al. Antimicrobial resistance genes in ESBL-producing *Escherichia coli* isolates from animals in Greece. *Antibiotics* **10**, 389 (2021).
27. Yang, H., Chen, H., Yang, Q., Chen, M. & Wang, H. High prevalence of Plasmid-Mediated quinolone resistance genes *Qnr* and *Aac (6')-Ib-cr* in clinical isolates of *Enterobacteriaceae* from nine teaching hospitals in China. *Antimicrob. Agents Chemother.* **53**, 847–847 (2009).
28. Kibwana, U. O. et al. Fluoroquinolone resistance among fecal extended spectrum β lactamases positive enterobacteriales isolates from children in Dar Es salaam, Tanzania. *BMC Infect. Dis.* **23**, 135 (2023).
29. Carattoli, A. Resistance plasmid families in *Enterobacteriaceae*. *Antimicrob. Agents Chemother.* <https://doi.org/10.1128/aac.01707-08> (2009).
30. Legese, M. et al. Molecular epidemiology of extended-spectrum beta-lactamase and AmpC producing *Enterobacteriaceae* among sepsis patients in Ethiopia: A prospective multicenter study. *Antibiotics* **11**, 131 (2022).
31. Ghaderi, R. S., Yaghoubi, A., Amirfakhrian, R., Hashemy, S. I. & Ghazvini, K. The prevalence of genes encoding ESBL among clinical isolates of *Escherichia coli* in Iran: A systematic review and meta-analysis. *Gene Rep.* **18**, 100562 (2020).
32. Weldhagen, G. F. GES: an emerging family of extended spectrum beta-lactamases. *Clin. Microbiol. Newsl.* **28**, 145–149 (2006).

33. Seni, J. et al. Preliminary insights into the occurrence of similar clones of extended-spectrum beta-lactamase-producing bacteria in humans, animals and the environment in tanzania: A systematic review and meta-analysis between 2005 and 2016. *Zoonoses Public Health*. **65**, 1–10 (2018).
34. Gharavi, M. J. et al. Comprehensive study of antimicrobial susceptibility pattern and extended spectrum beta-lactamase (ESBL) prevalence in bacteria isolated from urine samples. *Sci. Rep.* **11**, 578 (2021).
35. Singh, A. S., Nayak, B. B. & Kumar, S. H. High prevalence of multiple antibiotic-resistant, extended-spectrum β -lactamase (ESBL)-producing *Escherichia coli* in fresh seafood sold in retail markets of Mumbai, India. *Vet. Sci.* **7**, 46 (2020).
36. Labi, A. K. et al. High carriage rates of multidrug-resistant gram-negative bacteria in neonatal intensive care units from Ghana. *Open Forum Infect. Dis* **7**, (2020).
37. Aworh, M. K. et al. Extended-spectrum β -lactamase-producing *Escherichia coli* among humans, chickens and poultry environments in abuja, Nigeria. *One Health Outlook*. **2**, 8 (2020).
38. Hayer, J. et al. Multiple clonal transmissions of clinically relevant extended-spectrum beta-lactamase-producing *Escherichia coli* among livestock, dogs, and wildlife in Chile. *J. Glob Antimicrob. Resist.* **34**, 247–252 (2023).
39. Pietsch, M. et al. Molecular characterisation of extended-spectrum β -lactamase (ESBL)-producing *Escherichia coli* isolates from hospital and ambulatory patients in Germany. *Vet. Microbiol.* **200**, 130–137 (2017).
40. Partridge, S. R., Kwong, S. M., Firth, N. & Jensen, S. O. Mobile genetic elements associated with antimicrobial resistance. *Clin. Microbiol. Rev.* **31** <https://doi.org/10.1128/CMR.00088-17> (2018).
41. Johnson, T. J. et al. Separate F-type plasmids have shaped the evolution of the H30 subclone of *Escherichia coli* sequence type 131. *mSphere* **1**, e00121-16 (2016).
42. Schaumburg, F. et al. The risk to import ESBL-producing Enterobacteriaceae and *Staphylococcus aureus* through chicken meat trade in Gabon. *BMC Microbiol.* **14**, 286 (2014).
43. Founou, L. L., Founou, R. C. & Essack, S. Y. Antibiotic resistance in the food chain: A developing country-perspective. *Front. Microbiol.* **7**, (2016).
44. Venglovsky, J., Sasakova, N. & Placha, I. Pathogens and antibiotic residues in animal manures and hygienic and ecological risks related to subsequent land application. *Bioresour Technol.* **100**, 5386–5391 (2009).
45. Alegbeleye, O. O. & Sant'Ana, A. S. Manure-borne pathogens as an important source of water contamination: an update on the dynamics of pathogen survival/transport as well as practical risk mitigation strategies. *Int. J. Hyg. Environ. Health.* **227**, 113524 (2020).

Author contributions

Conceptualization, E.S.D. and S.N.-A.Y.; methodology, S.N.-A.Y., F.K., and A.A.A.; formal analysis, S.N.-A.Y., F.K., and A.A.A.; data curation, S.N.-A.Y., F.K., and A.A.A.; writing—original draft preparation, S.N.-A.Y., F.K., and A.A.A.; writing—review and editing of final manuscript, S.N.-A.Y. and E.S.D.; visualization, S.N.-A.Y., F.K., and A.A.A.; supervision, E.S.D.; project administration, E.S.D.; funding acquisition, E.S.D. All authors have read and agreed to the published version of the manuscript.

Funding

This review paper was supported by the Fogarty International Center of the National Institutes of Health through the Research and Capacity Building in Antimicrobial Resistance in West Africa (RECABAW) Training Programme hosted at the Department of Medical Microbiology, University of Ghana Medical School (Award Number: D43TW012487). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

Declarations

Competing interests

The authors declare no competing interests.

Additional information

Supplementary Information The online version contains supplementary material available at <https://doi.org/10.1038/s41598-025-10695-7>.

Correspondence and requests for materials should be addressed to E.S.D.

Reprints and permissions information is available at www.nature.com/reprints.

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Open Access This article is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License, which permits any non-commercial use, sharing, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if you modified the licensed material. You do not have permission under this licence to share adapted material derived from this article or parts of it. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by-nc-nd/4.0/>.

© The Author(s) 2025