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Systematic review on genomic insights into antimicrobial resistance in ESKAPE pathogens

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Background: Antimicrobial resistance (AMR) is a major global public health threat. ESKAPE pathogens *(Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa*, and *Enterobacter* spp.) pose a major threat owing to resistance to last-line antibiotics. Genomic surveillance is crucial to understanding global and regional antimicrobial resistance genes (ARGs) in AMR transmission.

Aim: This systematic review synthesised global genomic evidence to identify global and region-specific ARGs distribution among ESKAPE pathogens.

Methods: Following PRISMA guidelines, studies published January 2019 to December 2024 were identified from PubMed, Google Scholar, and Web of Science. Eligible studies reported genomic characteristics and resistance patterns of one or more ESKAPE pathogens from any source.

Results: Seventy-seven studies were included, with most originating from Asia, followed by Europe and Africa. Clinical isolates predominated *K. pneumoniae* was the most frequently investigated pathogen, followed by *S. aureus, P. aeruginosa*, and *A. baumannii*. The most reported resistance genes were *bla*CTX-M, *bla*NDM, and *bla*SHV. Distinct regional patterns of antimicrobial resistance gene (ARG) distribution were observed, with tetracycline and quinolone resistance genes prevailing in Africa and South America, and *bla*OXA variants dominating in Asia and Europe. Region-specific ARG patterns were identified through descriptive synthesis and comparative analysis of study-reported frequencies.

Conclusion: This review provides a synthesised global map of ARG distribution in ESKAPE pathogens, highlighting surveillance gaps in underrepresented regions and non-clinical settings. Addressing these gaps will support targeted genomic surveillance and stewardship programmes.

What this study adds: This study contributes to the body of knowledge by mapping global and regional antimicrobial resistance gene patterns in ESKAPE pathogens, identifying key surveillance gaps and informing targeted AMR monitoring and stewardship strategies.

Keywords: ESKAPE pathogens; antimicrobial resistance; antimicrobial resistance genes; genomic resistance patterns; global distribution.

Introduction

Global antimicrobial resistance burden

Antimicrobial resistance (AMR) is one of the top global public health and development threats.[1] To define it, AMR arises when microorganisms including bacteria, fungi, parasites, and viruses adapt and evolve to the point where antimicrobial medications that were previously effective become ineffective in treating infections.[1] Antimicrobial resistance is globally called the 'Silent Pandemic', requiring urgent attention and effective management.[2] The World Health Organization (WHO) formulated the Global Action Plan on AMR (GAP-AMR) to combat the rising challenge of AMR. In line with this initiative, in 2015, the WHO launched

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Note: Additional supporting information is available for download alongside the online version of this article as Online Supplementary Documents 1–4.

the Global Antimicrobial Resistance and Use Surveillance System (GLASS) to bolster surveillance efforts and promote data sharing.[3]

In 2021, an estimated 4.71 million deaths worldwide were linked to bacterial AMR, with 1.14 million deaths directly attributed to it.[4] However, recent research estimated that 1.91 million deaths attributable to AMR and 8·22 million deaths associated with AMR could occur globally in 2050.[4]

ESKAPE pathogens: A critical challenge

The word ESKAPE is an acronym given to six nosocomial pathogens that are known for their potential for multidrug resistance and virulence: *Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa,* and *Enterobacter* spp.[5] In 2024, the WHO published a list of pathogens of public health concern, and categorised them into critical, high-, and medium-priority levels, with carbapenem-resistant *A. baumannii*, third-generation cephalosporin-resistant Enterobacterales, and carbapenem-resistant Enterobacterales extended being part of the critical priority category, while vancomycin-resistant *E. faecium* (VRE), carbapenem-resistant *Pseudomonas aeruginosa,* along with methicillin-resistant *S. aureus* (MRSA) falls in the high-priority list.[6] ESKAPE pathogens are known to develop multidrug resistance and virulence through three primary mechanisms: they inactivate drugs (often via an enzyme-mediated irreversible cleavage); they modify the target site of the antibiotic; and they reduce drug accumulation either by decreasing permeability or by increasing drug efflux.[7] In addition to their multidrug resistance mechanisms, they can form biofilms that inhibit the immune response of the host and prevent antibiotics from targeting the pathogen effectively.

β-lactam antibiotics are still essential for treating infections caused by ESKAPE pathogens; however, resistance caused by β-lactamase production has become widespread. Key extended-spectrum β-lactamases (ESBLs), including TEM, SHV, and CTX-M, compromise the efficacy of β-lactams significantly. Although carbapenems are considered last-resort agents, the emergence of carbapenemase-producing bacteria carrying enzymes such as KPC, NDM, and OXA-48-like has reduced treatment options further.

Resistance to other major antibiotic classes is also prevalent among Gram-negative pathogens such as *Escherichia coli*, *K. pneumoniae*, and *A. baumannii*. Common tetracycline resistance determinants include *tetA, tetB, tetC, tetD*, and *tetG.* Fluoroquinolone resistance typically arises from mutations in the quinolone resistance-determining regions of *gyrA, gyrB, parC,* and *parE*, while aminoglycoside resistance is often mediated by aminoglycoside-modifying enzymes. In Gram-positive bacteria, notably *S. aureus*, methicillin resistance is driven by the *mecA* gene located in the SCCmec element, which encodes a low-affinity penicillin-binding protein.

Role of genomic surveillance

ESKAPE pathogens pose a serious global health threat owing to their ability to evade treatment through diverse, gene-driven resistance mechanisms. Their resistance is mediated by chromosomes or by an array of mobile genetic elements, such as plasmids, transposons, and integrons, which can spread genes mediating resistance to β-lactams, carbapenems, tetracycline, and aminoglycoside resistance genes across strains and environments through horizontal gene transfer. Despite the growing threat, existing genomic studies often focus on individual pathogens or selected resistance genes, leaving gaps in our understanding of the broader genomic landscape driving resistance across the entire ESKAPE group. This fragmented approach limits the ability to develop comprehensive interventions, as it overlooks the complex interplay of resistance mechanisms and their potential for global dissemination. While phenotypic surveillance shows what type of resistance a bacterium has, genomic surveillance helps us to understand how resistance works and where it comes from. By looking closely at bacterial DNA, we can identify the exact genes responsible for resistance, show how strains are related to each other, and track the spread of high-risk bacteria and plasmids across hospitals or even countries. This makes genomic surveillance a powerful early-warning tool for detecting new and emerging AMR threats.

Knowledge gaps and review objectives

A systematic review of genomic data from ESKAPE pathogens is essential to consolidate current knowledge, identify cross-cutting resistance determinants, and highlight regional variations in genetic profiles. Such insights can inform the development of more effective diagnostics, vaccines, and targeted therapies, while also guiding public health strategies like antimicrobial stewardship and infection control practices. Understanding the genomic diversity of these pathogens globally will not only help to predict future resistance trends but will also promote a more equitable, regionally informed response to AMR.

However, existing systematic reviews often examined AMR in ESKAPE pathogens, but must have focused primarily on phenotypic resistance profiles,[8] single pathogen,[9] or region.[10] A synthesised, cross-pathogen analysis of global genomic data identifying shared and region-specific resistance determinants is lacking. To address this gap, this review aimed to synthesise global genomic data on ESKAPE pathogens and to explore both shared and region-specific resistance genes across the globe.

Methods

Literature search and search strategy

The systematic review was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) (Online Supplementary Document 2).

Articles search was conducted from 09 December 2024 to 16 December 2024 in PubMed, Google Scholar, and Web of Science; only free-text search terms were used, no MeSH (Medical Subject Headings) terms or controlled vocabulary were used for the search. The search strategy employed a standardised Boolean structure to ensure consistent retrieval across databases.

To refine the search, parentheses were used to group related terms. The core search strings applied were: ('genomic insight' AND ('antimicrobial resistance' OR 'microbial pathogenesis') AND ('*Staphylococcus aureus*' OR '*Pseudomonas aeruginosa*' OR '*Klebsiella pneumoniae*' OR '*Acinetobacter baumannii*' OR '*Enterococcus faecium*' OR '*Enterobacter cloacae*').

Full search strings, applied filters, and the number of records retrieved from each database are presented in Online Supplementary Document 1 for transparency and reproducibility. Google Scholar was included for its large indexing of grey literature and non-PubMed-indexed scientific publications. Because Google Scholar yields very large sets, the first 1500 hits were screened.

The search was updated last on 16 December 2024. No additional automated alerts were used; however, backward and forward citation tracking was performed of included articles to identify any additional relevant studies.

The protocol was registered in the Prospective International Registry of Systematic Reviews (PROSPERO) of the National Institute of Health Research (Registration code: CRD420251237490).

Inclusion and exclusion criteria

Our inclusion criteria were defined using the PICO framework to enhance clarity as follows:

Population (P): studies involving any of the ESKAPE Pathogens (*E. faecium*, *S. aureus*, *K. pneumoniae*, *A. baumannii*, *P. aeruginosa*, and *E. cloacae* complex) from human, animal, or environmental sources.

Intervention (I): Genotypic characterisation of AMR, including whole-genome sequencing or polymerase chain reaction (PCR)-based detection of resistance genes.

Comparison (C): Not applicable because the review was descriptive, not comparative.

Outcome (O): Reporting of AMR genes.

We included peer-reviewed primary research articles published in English between 2019 and 2024 that reported genotypic data on AMR for any of the ESKAPE pathogens. In addition, eligible studies reported at least one of the following β-lactam resistance genes: bla_{OXA}, bla_{NDM}, bla_{CTX-M}, bla_{SHV}, bla_{TEM}, and bla_{KPC} or genes associated with resistance to tetracyclines, fluoroquinolones, aminoglycosides (*aac(6′)-Ib*, *ant(2″)-Ia*, and *aph(3′)-III*), or methicillin.

For the exclusion criteria, non-peer-reviewed literature, review papers, genomic databases, conference abstracts, and articles published in languages other than English were excluded. Also, studies presenting information on only one resistance gene were excluded because our review focused on broader genotypic resistance profiles for more meaningful interpretation and comparison across ESKAPE pathogens. Studies containing only phenotypic data were excluded.

Study selection and data extraction

Articles retrieved through the search strategy were first exported into the Rayyan tool for duplicate removal and initial screening. Two authors (Husna F. Ibrahim and Ahmad Abdulhadi) independently screened all titles and abstracts according to predefined eligibility criteria. Although no formal inter-rater reliability statistics (such as Cohen's Kappa) were calculated, disagreements were resolved by a third independent reviewer (Ibrahim Yusuf). Full-text screening was performed in the same manner.

Full texts of the screened publications were obtained from appropriate sources, and the data were extracted into an MS a Microsoft Excel spreadsheet under multiple headings, including country of origin, source, isolate type, number, AMR genes identified, and year of publication, among others. Additional details, such as study type, were also recorded. The full study selection process is presented in the 2020 flow diagram.

Data synthesis

The extracted data were synthesised narratively. Studies were grouped and analysed by geographical region, isolate source, isolate type, and antimicrobial resistance gene (ARG) type. Quantitative summaries are presented as counts (*n*) and proportions (%) of studies reporting specific ARGs or findings

Quality assessment of studies and risk of bias

The methodological quality of the included studies was assessed using the appropriate Joanna Briggs Institute (JBI) Critical Appraisal Checklist for cross-sectional studies.[11] All required checklist questions were extracted and reported individually in the quality assessment sheet without classifying the studies into overall categories of high, moderate, or low risk of bias. For ease of presentation and consistency, Question 1 of each JBI checklist was coded as Q1, Question 2 as Q2, and so forth throughout the quality assessment table. Each item was answered as 'Yes', 'No', 'Unclear', or 'Not Applicable', in accordance with JBI guidance. A comprehensive JBI checklist is presented in Online Supplementary Document 4.

The appraisal was conducted independently by two reviewers (Husna F. Ibrahim and Ahmad Abdulhadi), and

discrepancies were resolved through discussion and, when necessary, by a third reviewer (Ibrahim Yusuf). Quality assessment outcomes were not considered as exclusion criteria but rather informed the interpretation of results.

Results

Study selection

A thorough database search initially identified 2706 records. After removing duplicates, 2501 articles remained for initial screening. Of the 2501 articles, 2362 were excluded after reviewing their titles and abstracts. The remaining 139 articles underwent full-text screening to assess eligibility, resulting in 77 articles meeting the inclusion criteria and being included in the study (Figure 1).

Risk of bias and quality of studies

The methodological quality of the cross-sectional studies included was assessed using the JBI Critical Appraisal Checklist for Analytical Cross-Sectional Studies. Overall, most studies satisfied the majority of the JBI quality criteria, particularly with respect to clearly defined inclusion criteria, valid and reliable measurement of exposure and outcomes, and appropriate statistical analysis. No overall risk categories (high, moderate, or low) were assigned; instead, item-level responses to each checklist question (Q1–Q8) for all studies are provided in Online Supplementary Document 3.

Study characteristics

The study characteristics of the 77 studies included, including region of study, number of studies, isolate source, isolate type, and the ARGs detected, are shown in Table 1. A total of 77 studies were included in the review, which included 15 (19.48%) from Africa, 35 (45.45%) from Asia, 17 (22.08%) from Europe, three (3.90%) from North America, and seven (9.09%) from South America. Based on the source of isolates, about 70% (55/77) were from clinical samples, 18% (13/77) from animals, 6% (5/77) from the environment, 3% (2/77) from food, and about 1% (1/77) from each animal/environment source and environment/clinical source.

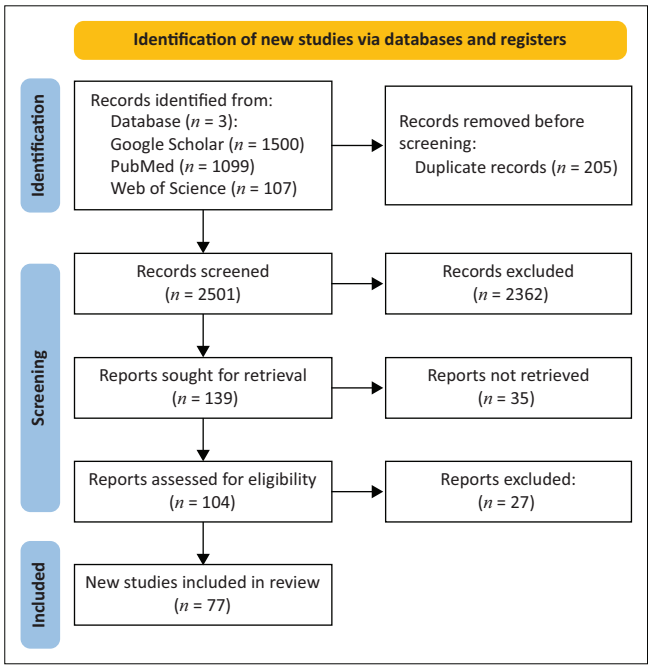


FIGURE 1: Preferred Reporting Items for Systematic Reviews and Meta-analyses flow diagram showing study selection process. The diagram shows the number of records retrieved from the searches conducted between 09 December 2024 and 16 December 2024 across all databases, duplicates removed, and the articles screened, excluded, and included in the final analysis.[12]

Distribution of articles

Overall, 23/77 (29.87%) were published in 2024, 19/77 (24.68%) in 2023, 17/77 (23.38%) in 2022, 8/77 (10.39%) in 2021, 5/77 (6.49%) in 2020 and 4/77 (5.19%) were published in 2019.

Among the studies included, 33/77 (42.86%) were on *K. pneumoniae*, 14/77 (18.18%) on *S. aureus*, 10/77 (12.99%) on *P. aeruginosa*, 10/77 (12.99%) on *A. baumannii*, 5/77 (6.49%) on *E. faecium*, and only 1/77 (1.30%) on *E. cloacae*. However, some articles reported multiple pathogens; for example, one study reported *K. pneumoniae* and *E. cloacae* (2 isolates), another one reported on *K. pneumoniae, A baumannii, P. aeruginosa*, and *E. cloacae* (4 isolates), while only two studies (2.60%) included all ESKAPE pathogens.

Distribution of ARGs

According to the publications, bla_{TEM} was identified in all the ESKAPE pathogens except for *E. faecium*; bla_{CTX-M} and bla_{NDM} were reported in all Gram-negative ESKAPE pathogens (i.e. *K. pneumoniae, A. baumannii, P. aeruginosa*, and *E. cloacae*), while *tetM* was only reported in Gram-positive ESKAPE (*E. faecium* and *S. aureus*). For pathogen-specific ARGs, bla_{OXA} variants, which include 23, 24/40, 51, 58, 66, 67, 69, and 72; and *tet* variants X3 and 39 were only reported in *A. baumannii*. Similarly, *ant(2″)-Ia*, bla_{OXA-50}, $bla_{OXA-395}$, $bla_{OXA-1202}$, $bla_{OXA-1203}$ *and* $bla_{OXA-1204}$, and *qnrVC* were specific to *P. aeruginosa*. In addition, *tet* (K and 38) and *mecA* to *S. aureus*, *aph(3′)-III* were only reported in *E. faecium* and *tetA* in *E. cloacae* (Figure 2a).

The β-lactams resistance encoding genes reviewed were bla_{CTX-M}, bla_{KPC}, bla_{NDM}, bla_{OXA}, bla_{SHV}, and bla_{TEM}. From the publications, bla_{CTX-M} was the most reported (n = 37; 48.05%), followed by bla_{NDM} (n = 35; 45.45%), bla_{SHV} (n = 32; 41.56%), bla_{TEM} (n = 22; 28.57%), and bla_{KPC} (n = 18; 23.38%). Similarly, different variants of bla_{OXA} were reported as follows: bla_{OXA-23} (n = 11; 14.29%), bla_{OXA-1} (n = 9; 11.69%), bla_{OXA-48} (n = 7; 9.09%), $bla_{OXA-1,}$ $bla_{OXA-58,}$ and bla_{OXA-66} (n = 4; 5.19%), bla_{OXA-72} (n = 3; 3.90%); two each of $bla_{OXA-50,}$ $bla_{OXA-232,}$ $bla_{OXA-395,}$ and $bla_{OXA-486}$ (2.60%) and the least reported were $bla_{OXA-24/40,}$ $bla_{OXA-67,}$ $bla_{OXA-69,}$ $bla_{OXA-90,}$ $bla_{OXA-181,}$ $bla_{OXA-1202,}$ $bla_{OXA-1203,}$ and $bla_{OXA-1204}$ (n = 1; 1.30%) (Table 2).

For the aminoglycoside resistance genes, *aac(6′)*-Ib was reported the most (n = 7; 9.09%), followed by *aph(3′)*-III (n = 2; 2.60%), and the least reported were *ant(2″)*-Ia, and *aac(6′)* (n = 1; 1.30%) (Table 2).

TABLE 1: Characteristics of studies included in this global systematic review, comprising articles published between January 2019 and December 2024, including region, number of studies, isolate source, isolate type, and antimicrobial resistance genes (ARGs) detected.

Region	No. of studies	Isolate source	Isolate type	ARGs Detected	References
Africa	4	Animal (4/15)	*K. pneumoniae*	*aac(6')-Ib*	[13,14]
				bla_{NDM}	
				bla_{CTX-M}	
				bla_{SHV}	
				bla_{OXA-1}	
				bla_{TEM}	
				qnrB	
				tetA	
			P. aeruginosa	*ant(2")-Ia*	[15,16]
				bla_{CTX-M}	
				bla_{OXA-1}, bla_{OXA-10}	
				bla_{SHV}	
				bla_{TEM}	
			E. cloacae	bla_{CTX-M}	[14]
				bla_{TEM}	
	10	Clinical (10/15)	*S. aureus*	*aac(6')-Ib*	[17]
			K. pneumoniae	bla_{CTX-M}	[17,18,19,20,21]
				bla_{NDM}	
				bla_{OXA-1}, bla_{OXA-9}, bla_{OXA-48}	
				bla_{SHV}	
				bla_{TEM}	
				mecA	
				qnrB, qnrS	
				tetA, tetB, tetC, tetD	
			A. baumannii	bla_{NDM}	[17,22]
				bla_{OXA-23}, bla_{OXA-58}	
				gyrA	
				parC	
				tetA	
			P. aeruginosa	bla_{NDM}	[17,23,24]
				$bla_{OXA-395}$	
				gyrA	
				qnrVC	
				tetG	
			E. cloacae	bla_{CTX-M}	[17]
				bla_{OXA-48}	
	1	Environmental (1/15)	*A. baumannii*	bla_{NDM}	[22]
				bla_{OXA-28}, bla_{OXA-58}	
				gyrA	
				parC	
				tetX3	
Asia	4	Animal (4/35)	*S. aureus*	bla_{TEM}	[25,26,27]
				gyrA	
				parC	
				mecA	
				tetM	
			K. pneumoniae	bla_{SHV}	[28]
				qnrS	
				tetA	
	28	Clinical (28/35)	*E. faecium*	*aac(6')*	[29]
				aph(3')-III	
			S. aureus	*aph(3')-III*	[30,31,32,33]
				gyrA	
				parC	
				mecA	
				tet38, tetC, tetK, tetM	

Table 1 continues on next page →

TABLE 1 (Continues…): Characteristics of studies included in this global systematic review, comprising articles published between January 2019 and December 2024, including region, number of studies, isolate source, isolate type, and antimicrobial resistance genes (ARGs) detected.

Region	No. of studies	Isolate source	Isolate type	ARGs Detected	References
			K. pneumoniae	*aac(6')-Ib*	34,35,36,37,38,39,40,41,42,43,44,45,46
				bla_{CTX-M}	
				bla_{KPC}	
				bla_{NDM}	
				bla_{OXA-1}, bla_{OXA-48}, $bla_{OXA-181}$, $bla_{OXA-232}$	
				bla_{SHV}	
				bla_{TEM}	
				tetA, tetB	
				qnrA, qnrB, qnrS	
			A. baumannii	bla_{CTX-M}	47,48
				bla_{OXA-23}, bla_{OXA-66}, bla_{OXA-69}, bla_{OXA-72}, bla_{OXA-90}	
				tetA, tetB, tetR	
				qnr	
			P. aeruginosa	bla_{OXA-50}, $bla_{OXA-1202}$, $bla_{OXA-1203}$, $bla_{OXA-1204}$	49,50,51,52
				bla_{SHV}	
				qnrB, qnrVC	
			E. cloacae	bla_{CTX-M}	53
				bla_{NDM}	
				bla_{SHV}	
				qnrS	
				tetA	
	1	Environmental (1/35)	*S. aureus*	*mecA*	54
	1	Environmental/Clinical (1/35)	*A. baumannii*	bla_{OXA-23}, bla_{OXA-66}, bla_{OXA-67}	55
				tet39, tetB	
	1	Food (1/35)	*S. aureus*	*mecA*	56
				tetK	
Europe	2	Animal (2/17)	*K. pneumoniae*	*blaSHV*	57
			A. baumannii	bla_{OXA-23}, bla_{OXA-51}	57
			P. aeruginosa	bla_{OXA-50}, $bla_{OXA-486}$	57,58
			E. cloacae	bla_{SHV}	57
				bla_{TEM}	
		Animal or Environmental (1/17)	*E. faecium*	*tetM, tetL*	59
	10	Clinical (10/17)	*S. aureus*	*mecA*	60,61
				tetM	
			K. pneumoniae	*aac(6')-Ib*	61,62,63,64,65
				bla_{CTX-M}	
				bla_{KPC}	
				bla_{NDM}	
				bla_{OXA-1}, bla_{OXA-48}	
				bla_{SHV}	
				bla_{TEM}	
				gyrA	
				parC	
				qnrB, qnrS	
			A. baumannii	*aac(6')-Ib*	61,66,67
				bla_{NDM}	
				bla_{OXA-23}, $bla_{OXA-24/40}$, bla_{OXA-51}, bla_{OXA-58}, bla_{OXA-66}, bla_{OXA-72}	
				gyrA	
				parC	
				tetB	
			P. aeruginosa	bla_{NDM}	61,68,69
				$bla_{OXA-395}$	
				gyrA	
				qnrVC	
				tetG	

Table 1 continues on next page →

TABLE 1 (Continues...): Characteristics of studies included in this global systematic review, comprising articles published between January 2019 and December 2024, including region, number of studies, isolate source, isolate type, and antimicrobial resistance genes (ARGs) detected.

Region	No. of studies	Isolate source	Isolate type	ARGs Detected	References
	3	Environmental (3/17)	*E. faecium*	*tetM*	70
			K. pneumoniae	*aac(6')-Ib*	71,72
				bla_{CTX-M}	
				bla_{KPC}	
				bla_{NDM}	
				bla_{SHV}	
				qnrB	
				tetA, tetB, tetD, tetR	
	1	Food (1/17)	*E. faecium*	*aph(3')-III*	73
				tetL, tetM	
North America	1	Animal (1/3)	*S. aureus*	*mecA*	74
				tet38, tetM	
	2	Clinical (2/3)	*K. pneumoniae*	bla_{CTX-M}	75
				bla_{KPC}	
				bla_{SHV}	
				gyrA	
				parC	
				qnrB	
			A. baumannii	$bla_{OXA-23-like}$, bla_{OXA-66}	76
				blaTEM	
				tetB	
South America	3	Animal (3/7)	*S. aureus*	*mecA*	77,78
				tet38	
			K. pneumoniae	bla_{CTX-M}	79
				bla_{SHV}	
				bla_{TEM}	
				qnrE	
				tetA	
	4	Clinical (4/7)	*S. aureus*	*mecA*	80
				tet38	
			K. pneumoniae	bla_{CTX-M}	81,82
				bla_{KPC}	
				bla_{NDM}	
				bla_{OXA-1}	
				bla_{SHV}	
				bla_{TEM}	
				tetA, tetD	
				qnrS	
			A. baumannii	bla_{OXA-23}, $bla_{OXA-51-like}$, bla_{OXA-72}	83
				bla_{TEM}	

Note: Please see the full reference list of Ibrahim HF, Abdullahi N, Yusuf I, et al. Systematic review on genomic insights into antimicrobial resistance in ESKAPE pathogens. Afr J Lab Med. 2026;15(1), a2893. https://doi.org/10.4102/ajlm.v15i1.2893.

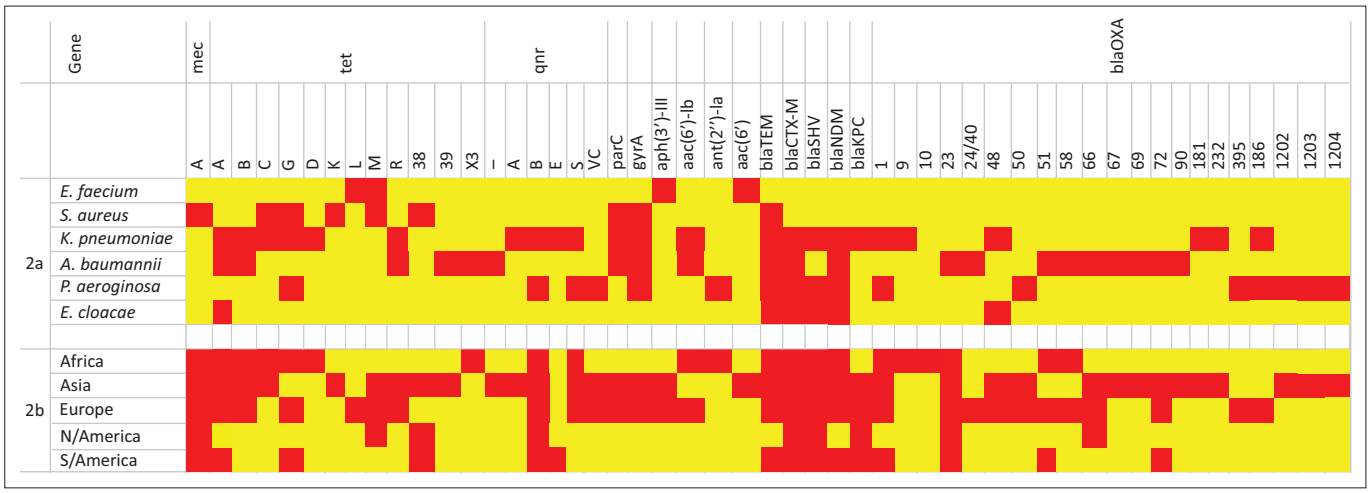
ARG, antimicrobial resistance gene.

The *mec*A gene was reported in all 16 articles published on *S. aureus* (*n* = 16; 20.78%), other ARGs such as *tet38* and *tetM* were also reported in *S. aureus* (Table 2).

Different variants of quinolone ARGs were reported in many articles, which included *qnr, qnrA, qnrB, qnrE, qnrS, qnrVC, gyrA,* and *parC*. The most commonly reported out of these genes was *qnrB* (*n* = 12; 15.58%), followed by mutation in *gyrA* (*n* = 9; 11.69%), *qnrS* (*n* = 8; 10.39%), mutation in *parC* (*n* = 7; 9.09%), then *qnrVC* (*n* = 2; 2.60%) while the least reported genes were *qnr, qnrA,* and *qnrE* (*n* = 1; 1.30%) (Table 2).

For tetracycline, the most reported ARG was *tetA* (*n* = 14; 18.18%), followed by *tetB* (*n* = 7; 9.09%), *tetM* (*n* = 5; 6.49%), *tet38* and *tetD* (*n* = 3; 3.90%), the *tetC, tetK, tetL, tetR*, and *tetG* (*n* = 2; 2.60%), and only one study reported *tet39*, and *tetX3* in (environment, *n* = 1; 1.30%) (Table 2).

According to the studies included, the ARGs distribution across all the regions is presented in Figure 2b. However, some ARGs from the included studies were reported more often in a particular region, while some were cross-regional. *tetG* and *tetX3*, *ant(2'')-IIa*, $bla_{OXA-1,}$ and bla_{OXA-10} were reported in studies conducted in Africa in this review. In contrast, *tetk, qnrA* and bla_{OXA} variants 67, 69, 90, 181, 232, 1202, 1203, and 1204 are reported in studies conducted in Asia. In addition, *tetL* and bla_{OXA} *variants* (24/40, 395, and 486) were found in studies conducted in Europe, while *qnrE* was only reported in South America. However, *mecA, qnrB,* bla_{CTX-M}, bla_{SHV}, and bla_{OXA-23} were reported in all the regions of the globe; hence, cross-regional ARGs (Figure 2b).



Note: Please see the full reference list of Ibrahim HF, Abdullahi N, Yusuf I, et al. Systematic review on genomic insights into antimicrobial resistance in ESKAPE pathogens. Afr J Lab Med. 2026;15(1), a2893. https://doi.org/10.4102/ajlm.v15i1.2893.

FIGURE 2: (a) Distribution of antimicrobial resistance genes among ESKAPE pathogens across global studies published between January 2019 and December 2024, showing pathogen-specific and shared genes; (b) Regional distribution of antimicrobial resistance genes among ESKAPE pathogens across global studies published between January 2019 and December 2024. Red color indicates the presence of a gene, while yellow indicates its absence.

Discussion

The systematic review aimed to synthesise global genomic ARG data for ESKAPE pathogens. Our analysis confirmed the widespread presence of ARGs in ESKAPE globally. It also showed that Asia has the highest number of publications on ARGs in ESKAPE pathogen. This is not surprising because Southeast Asia is regarded as a global hub for AMR emergence. After all, it has the highest risk of AMR emergence among all WHO regions in Asia, according to Shrestha, He and Legido-Quigley.[84] The publication pattern may also be as a result of regional differences in research capacity and surveillance investment. Several Asian countries have established large AMR research networks and publish actively in journals indexed in major international databases, which may contribute to their strong representation. Following Asia, the European region has the next highest publications on ESKAPE-related ARGs, which most likely shows both the widespread distribution of the ESKAPE pathogens in the region and the emphasis of the region on AMR surveillance and research funding. Differences in indexing coverage across regions may also influence publication distribution. The African region, though predominantly low- and middle-income countries, came third as far as the number of publications on ARGs of ESKAPE pathogens is concerned. Africa has been characterised by low surveillance data on AMR owing to some challenges, which include weak laboratory infrastructure, limited capacity and training, funding and communication issues,[85] and inability to pay for high-cost open access charges (which leads to most of the articles to be published in low impact journals).

The review indicates a notable increase in publications on AMR in ESKAPE pathogens from 2019 to 2024, with 30% of the studies published in 2024. This surge reflects heightened global attention to AMR, particularly concerning ESKAPE pathogens. The escalation in publications may be linked to the increased use of antibiotics during the coronavirus disease 2019 pandemic, which has raised concerns about the amplification of AMR.[86] The WHO reported widespread overuse of antibiotics in hospitalised coronavirus disease 2019 patients, with approximately 75% receiving antibiotics despite only 8% having bacterial co-infections. This excessive use most likely contributed to the acceleration of AMR during and after the pandemic; hence, also in publications.[87] In addition, some studies have indicated an increase in AMR trends during and after the coronavirus disease 2019 period compared to pre-pandemic times. For instance, a rise in resistance among pathogens such as *K. pneumoniae* and *A. baumannii* to certain antibiotics post-coronavirus disease 2019 has been reported.[88]

The systematic review revealed further that ARGs are more frequently reported in clinical samples than in non-clinical samples. The lack of adequate non-clinical data (environmental, animal, food) represents a critical knowledge gap. Antimicrobial resistance genes in clinical settings are probably part of a larger cycle involving agricultural runoff, wastewater, and wildlife. Integrating One Health surveillance using metagenomics is essential to understanding global reservoir dynamics and transmission routes. A similar trend was observed in a systematic review by Somda et al.[89] on carbapenem-resistant Enterobacteriaceae in West Africa, which also reported a higher prevalence of resistant strains in hospital-based sources compared to environmental or community settings.

In addition, *K. pneumoniae* emerged as the most frequently reported ESKAPE pathogen globally, reinforcing its recognised role as a major driver of the global AMR burden. This aligns with previous findings by Navon-Venezia et al.,[90] which emphasise the ability of *K. pneumoniae* to develop multi and extensive drug-resistant phenotypes, making it a critical public health threat.

TABLE 2: Distribution of antimicrobial resistance genes based on studies included in this global systematic review (articles published between January 2019 and December 2024).

Antibiotics	ARGs	No. of studies included	Distribution (%)
β-lactams	bla_{CTX-M}	37	48.05
	bla_{NDM}	35	45.45
	bla_{SHV}	32	41.56
	bla_{TEM}	22	28.57
	bla_{KPC}	18	23.38
	bla_{OXA-23}	11	14.29
	bla_{OXA-1}	9	11.69
	bla_{OXA-48}	7	9.09
	bla_{OXA-51}	4	5.19
	bla_{OXA-58}	4	5.19
	bla_{OXA-66}	4	5.19
	bla_{OXA-72}	3	3.90
	$bla_{OXA-232}$	2	2.60
	$bla_{OXA-395}$	2	2.60
	$bla_{OXA-486}$	2	2.60
	bla_{OXA-50}	2	2.60
	bla_{OXA-9}	1	1.30
	bla_{OXA-10}	1	1.30
	$bla_{OXA-24/40}$	1	1.30
	bla_{OXA-67}	1	1.30
	bla_{OXA-69}	1	1.30
	bla_{OXA-90}	1	1.30
	$bla_{OXA-181}$	1	1.30
	$bla_{OXA-1202}$	1	1.30
	$bla_{OXA-1203}$	1	1.30
	$bla_{OXA-1204}$	1	1.30
Aminoglycoside	*aac(6′)-Ib*	7	9.09
	aph(3′)-III	2	2.60
	ant(2″)-Ia	1	1.30
	aac(6′)	1	1.30
Methicillin	*mecA*	16	20.78
Quinolone	*qnrB*	12	15.58
	gyrA	9	11.69
	qnrS	8	10.39
	parC	7	9.09
	qnrVC	2	2.60
	qnrE	1	1.30
	qnr	1	1.30
	qnrA	1	1.30
Tetracycline	*tetA*	14	18.18
	tetB	7	9.07
	tetM	5	6.49
	tet38	3	3.90
	tetD	3	3.90
	tetK	2	2.60
	tetL	2	2.60
	tetR	2	2.60
	tetG	2	2.60
	tetC	2	2.60
	tet39	1	1.30
	tetX3	1	1.30

Note: Please see the full reference list of Ibrahim HF, Abdullahi N, Yusuf I, et al. Systematic review on genomic insights into antimicrobial resistance in ESKAPE pathogens. Afr J Lab Med. 2026;15(1), a2893. https://doi.org/10.4102/ajlm.v15i1.2893.

ARG, antimicrobial resistance gene.

Among the ARGs identified in this review, bla_{CTX-M}, bla_{NDM}, and bla_{SHV} (Table 2) were the most frequently reported. Their high prevalence is most likely because of not only their association with mobile genetic elements such as plasmids and transposons that enable rapid horizontal transfer, but also to ecological and clinical factors that promote their global dominance. For instance, bla_{CTX-M} variants have been linked frequently to highly successful epidemic plasmids and dominant *E. coli* and *K. pneumoniae* lineages circulating in both community and hospital environments. Also, the widespread occurrence of bla_{NDM} is reinforced by its repeated emergence in high-risk ESKAPE clones, which aids in its rapid spread in healthcare networks. In addition, the habit of antibiotic consumption, especially the widespread use of third-generation cephalosporins and carbapenems, leads to strong selective pressures that favour the bacteria carrying these genes. The combination of gene mobility, successful host lineages, and sustained antibiotic selection most likely explains their global dissemination and persistence.

Carbapenem resistance genes, such as bla_{KPC}, were found exclusively in *K. pneumoniae* in this review, which aligns with findings from Teixeira et al.,[91] who reported that bla_{KPC} was only detected in *K. pneumoniae*. However, this contrasts with the study by Hazen et al.,[92] which also identified bla_{KPC} in *E. coli* and other bacterial species. Similarly, in this review, the $bla_{OXA-181}$ gene was detected solely in *K. pneumoniae* strains, consistent with the findings of Somda et al.,[89] who reported its presence in *K. pneumoniae* among Gram-negative ESKAPE pathogens studied.

The systematic review identified cross-regional ARGs as *mec*A, *qnr*B, bla_{CTX-M}, bla_{SHV} and bla_{OXA-23} (Figure 2b), which shows their global spread of resistance genes and the potential for horizontal gene transfer. These ARGs are often found on mobile genetic elements, such as plasmids and integrons, which facilitate their rapid transfer between bacteria across different regions. This horizontal gene transfer, coupled with the widespread use of antibiotics globally, creates a strong selective pressure that promotes their persistence and dissemination worldwide.

In addition, the review identified some region-specific ARGs, such as *tetG* and *tetX3* for Africa, *tetk, qnrA*, and bla_{OXA} variants (67, 69, 90, 181, 232, 1202, 1203, and 1204) for Asia, *tetL*, and bla_{OXA} variants (24/40, 395, and 486) for Europe, and *qnrE* for South America. The reason might be that different regions often have distinct antibiotic usage patterns both in clinical and agricultural settings, which create unique selective environments. These pressures favour the acquisition and persistence of certain resistance genes over others in local bacterial populations. Moreover, regional differences in infection control practices, healthcare infrastructure, and environmental factors can influence the types of bacteria and mobile genetic elements specific to that area, thereby promoting localised genetic adaptations. This underlines the importance of regional surveillance and tailored antimicrobial stewardship strategies.

Compared with earlier systematic reviews on ESKAPE pathogens, which largely summarised phenotypic resistance[8] or focused on specific regions[10] or organisms,[9] our findings highlight several novel insights. First, by incorporating only

genomic studies published between 2019 and 2024, this review captures emerging ARGs that were under-represented in previous analyses. Second, integration of regional comparison demonstrates distinct ARG distributions between Africa, Asia, Europe and the Americas, a dimension not examined in earlier reviews. Finally, this synthesis integrates cross-ARGs, revealing shared ARG reservoirs and high-risk clones across ESKAPE pathogens. Together, these emphasise the added value and novel contribution of our study to the current genomic AMR landscape.

The strengths of the study include a comprehensive systematic review which incorporates a large dataset (77 studies) and provides a robust and diverse analysis of ESKAPE pathogens. It also categorises ARGs based on geographical regions, which provides insights into global and region-specific resistance trends. Finally, the review also presents detailed tables and figures that summarise ARGs distribution across different pathogens and regions, which may help in reinforcing the importance of global AMR surveillance.

These findings have direct implications for Infection Prevention and Control (IPC) and stewardship. The prevalence of bla_{NDM} in Asia suggests that travel screening and targeted stewardship may be warranted. The detection of identical ARG-carrying plasmids across continents emphasises the need for enhanced genomic surveillance to track transmission networks.

Limitations

Despite its strength, the study has some limitations. It only includes studies from 2019 to 2024, which may result in excluding older but still relevant studies. The restriction to English-language studies also introduces bias, potentially overlooking non-English research from various regions. The imbalance in studies from all regions may under-represent the true AMR burden in underreported regions. In addition, the limited availability of articles from non-clinical sources may result in potential underestimation of ARG diversity in environmental and animal reservoirs.

To address these limitations, future research should focus on expanding surveillance studies to include regional or multilingual databases and non-English publications to reduce geographic and language bias. Also, incorporating metagenomics surveillance data and environmental monitoring programmes would help to capture ARGs beyond clinical settings.

Conclusion

This systematic review provides valuable insights into the genomic basis of AMR in ESKAPE pathogens, with a strong focus on regional variations and global trends in ARGs specific to these pathogens. The study reveals important gaps in current genomic surveillance, especially in low- and middle-income countries and in non-clinical reservoirs such as the environment and clinical sectors. The findings from this review emphasise the need for continuous genomic surveillance to monitor the evolution of resistance genes, particularly in regions with a high prevalence of AMR genes.

Strengthening global data sharing, harmonising sequencing and reporting standards, and expanding surveillance capacity in underrepresented regions will be essential for improving early-warning systems for AMR threats. Also, the synthesis presented here provides an evidence base to support policymakers, researchers, and public health bodies in prioritising interventions and investing in the genomic infrastructure needed to curb the continued expansion of resistance in ESKAPE pathogens.

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Competing interests

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CRediT authorship contribution

Husna F. Ibrahim: Conceptualisation, Methodology, Resources, Visualisation, Writing – original draft, Writing – review & editing. Nasiru Abdullahi: Conceptualisation, Supervision, Writing – review & editing. Ibrahim Yusuf: Conceptualisation, Supervision, Writing – review & editing. Bashir I. Waziri: Conceptualisation, Supervision, Writing – review & editing. Ahmad Abdulhadi: Conceptualisation, Writing – review & editing. Khadija Muhammad: Conceptualisation, Writing – review & editing. Maryam M. Ibrahim: Conceptualisation, Writing – review & editing. Aisha A. Abdullahi: Methodology, Writing – review & editing. Muhammad A. Abbas: Supervision, Writing – review & editing. Hamisu Salihu: Supervision, Writing – review & editing. All authors reviewed the article, contributed to the discussion of results, approved the final version for submission and publication, and take responsibility for the integrity of its findings.

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Data availability

Data sharing is not applicable to this article as no new data were created or analysed in this study.

Disclaimer

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References

1. WHO. Antimicrobial resistance [homepage on the Internet]. World Health Organization; 2023 [cited 2025 Mar 24]. Available from: https://www.who.int/news-room/fact-sheets/detail/antimicrobial-resistance
2. Founou RC, Founou LL, Allam M, Ismail A, Essack SY. Whole genome sequencing of extended spectrum β-lactamase (ESBL)-producing Klebsiella pneumoniae isolated from hospitalized patients in KwaZulu-Natal, South Africa. Sci Rep. 2019;9(1):6266. https://doi.org/10.1038/s41598-019-42672-2
3. World Health Organization. Global antimicrobial resistance and use surveillance system (GLASS) [homepage on the Internet]. 2026 [cited 2025 Mar 24]. Available from: https://www.who.int/initiatives/glass
4. GBD 2021 Antimicrobial Resistance Collaborators. Global burden of bacterial antimicrobial resistance 1990–2021: A systematic analysis with forecasts to 2050. Lancet. 2024;404(10459):1199–1226. https://doi.org/10.1016/S0140-6736(24)01867-1
5. Mulani MS, Kamble EE, Kumkar SN, Tawre MS, Pardesi KR. Emerging strategies to combat ESKAPE pathogens in the era of antimicrobial resistance: A review. Front Microbiol. 2019;10:539. https://doi.org/10.3389/fmicb.2019.00539
6. WHO bacterial priority pathogens list, 2024: Bacterial pathogens of public health importance to guide research, development and strategies to prevent and control antimicrobial resistance [homepage on the Internet]. 2024 [cited 2025 Mar 24]. Available from: https://www.who.int/publications/i/item/9789240093461
7. Santajit S, Indrawattana N. Mechanisms of antimicrobial resistance in ESKAPE pathogens. BioMed Res Int. 2016;2016:1–8. https://doi.org/10.1155/2016/2475067
8. De Oliveira DMP, Forde BM, Kidd TJ, et al. Antimicrobial resistance in ESKAPE pathogens. Clin Microbiol Rev. 2020;33(3):e00181-19. https://doi.org/10.1128/CMR.00181-19
9. Forero-Hurtado D, Corredor-Rozo ZL, Ruiz-Castellanos JS, et al. Worldwide dissemination of blaKPC gene by novel mobilization platforms in Pseudomonas aeruginosa: A systematic review. Antibiotics. 2023;12(4):658. https://doi.org/10.3390/antibiotics12040658
10. Tansarli GS, Poulikakos P, Kapaskelis A, Falagas ME. Proportion of extended-spectrum β-lactamase (ESBL)-producing isolates among Enterobacteriaceae in Africa: Evaluation of the evidence – Systematic review. J Antimicrob Chemother. 2014;69(5):1177–1184. https://doi.org/10.1093/jac/dkt500
11. Martin J. Critical appraisal checklist for analytical cross sectional studies [homepage on the Internet]. Adelaide: Joanna Briggs Institute; 2017 [cited 2025 Apr 4]. Available from: https://jbi.global/sites/default/files/2019-05/JBI_Critical_Appraisal-Checklist_for_Analytical_Cross_Sectional_Studies2017_0.pdf
12. Haddaway NR, Page MJ, Pritchard CC, McGuinness LA. PRISMA 2020: An R package and Shiny app for producing PRISMA 2020-compliant flow diagrams, with interactivity for optimised digital transparency and open synthesis. Campbell Syst. Rev. 2022;18(2):e1230. https://doi.org/10.1002/cl2.1230
13. Mairi A, Barraud O, Muggeo A, De Champs C, Touati A. Genomic analysis of a multidrug-resistant Klebsiella pneumoniae ST11 strain recovered from Barbary deer (Cervus elaphus barbarus) in Akfadou Forest, Algeria. J Glob Antimicrob Resist. 2020;22:515–518. https://doi.org/10.1016/j.jgar.2020.04.027
14. Olorunleke SO, Kirchner M, Duggett N, et al. Rapid detection and molecular epidemiology of β-lactamase producing Enterobacteriaceae isolated from food animals and in-contact humans in Nigeria. PLoS One. 2024;19(4):e0289190. https://doi.org/10.1371/journal.pone.0289190
15. Rizk AM, Elsayed MM, Abd El Tawab AA, et al. Phenotypic and genotypic characterization of resistance and virulence in Pseudomonas aeruginosa isolated from poultry farms in Egypt using whole genome sequencing. Vet Microbiol. 2024;292:110063. https://doi.org/10.1016/j.vetmic.2024.110063
16. Salem M, Younis G, Sadat A, et al. Dissemination of mcr-1 and β-lactamase genes among Pseudomonas aeruginosa: Molecular characterization of MDR strains in broiler chicks and dead-in-shell chicks infections. Ann Clin Microbiol Antimicrob. 2024;23(1):9. https://doi.org/10.1186/s12941-024-00669-4
17. Zalegh I, Bourhia M, Zerouali K, et al. Molecular characterization of gene-mediated resistance and susceptibility of ESKAPE clinical isolates to Cistus monspeliensis L. and Cistus salviifolius L. Extracts. Ramanathan S, editor. Evid Based Complement Alternat Med. 2022;2022:1–16. https://doi.org/10.1155/2022/7467279
18. Altayb HN, Elbadawi HS, Baothman O, et al. Genomic analysis of multidrug-resistant hypervirulent (hypermucoviscous) Klebsiella pneumoniae strain lacking the hypermucoviscous regulators (rmpA/rmpA2). Antibiotics. 2022;11(5):596. https://doi.org/10.3390/antibiotics11050596
19. Elgayar FA, Gouda MK, Badran AA, El Halfawy NM. Pathogenomics analysis of high-risk clone ST147 multidrug-resistant Klebsiella pneumoniae isolated from a patient in Egypt. BMC Microbiol. 2024;24(1):256. https://doi.org/10.1186/s12866-024-03389-z
20. Lewis JM, Mphasa M, Banda R, et al. Genomic and antigenic diversity of colonizing Klebsiella pneumoniae isolates mirrors that of invasive isolates in Blantyre, Malawi. Microb Genomics. 2022;8(3):000778. https://doi.org/10.1099/mgen.0.000778
21. Odewale G, Jibola-Shittu MY, Ojurongbe O, Olowe RA, Olowe OA. Genotypic determination of extended spectrum β-lactamases and carbapenemase production in clinical isolates of Klebsiella pneumoniae in Southwest Nigeria. Infect Dis Rep. 2023;15(3):339–353. https://doi.org/10.3390/idr15030034
22. Odih EE, Oaikhena AO, Underwood A, et al. High genetic diversity of carbapenem-resistant Acinetobacter baumannii isolates recovered in Nigerian Hospitals in 2016 to 2020. mSphere. 2023;8(3):e00098-23. https://doi.org/10.1128/msphere.00098-23
23. Eladawy M, Thomas JC, Hoyles L. Phenotypic and genomic characterization of Pseudomonas aeruginosa isolates recovered from catheter-associated urinary tract infections in an Egyptian hospital. Microb Genomics. 2023;9(10):001125. https://doi.org/10.1099/mgen.0.001125
24. Olalekan A, Bader BK, Iwalokun B, et al. High incidence of carbapenemase-producing Pseudomonas aeruginosa clinical isolates from Lagos, Nigeria. JAC-Antimicrob Resist. 2023;5(2):dlad038. https://doi.org/10.1093/jacamr/dlad038
25. Rahman MH, El Zowalaty ME, Falgenhauer L, et al. Molecular identification and whole genome sequence analyses of methicillin-resistant and mastitis-associated Staphylococcus aureus sequence types 6 and 2454 isolated from dairy cows. J Genomics. 2024;12:19–25. https://doi.org/10.7150/jgen.90833
26. Sivakumar R, Pranav PS, Annamanedi M, et al. Genome sequencing and comparative genomic analysis of bovine mastitis-associated Staphylococcus aureus strains from India. BMC Genomics. 2023;24(1):44. https://doi.org/10.1186/s12864-022-09090-7
27. Sivaraman GK, Sivam V, Ganesh B, Elangovan R, Vijayan A, Mothadaka MP. Whole genome sequence analysis of multi drug resistant community associated methicillin resistant Staphylococcus aureus from food fish: Detection of clonal lineage ST 28 and its antimicrobial resistance and virulence genes. PeerJ. 2021;9:e11224. https://doi.org/10.7717/peerj.11224
28. Khan MM, Mushtaq MA, Abbas N, et al. Occurrence, antimicrobial resistance and genomic features of Klebsiella pneumoniae from broiler chicken in Faisalabad, Pakistan. Front Vet Sci. 2024;11:1433124. https://doi.org/10.3389/fvets.2024.1433124
29. Shen J, Long X, Jiang Q, et al. Genomic characterization of a vancomycin-resistant strain of Enterococcus faecium harboring a rep2 Plasmid. Infect Drug Resist. 2023;16:1153–1158. https://doi.org/10.2147/IDR.S398913
30. Ballah FM, Hoque MN, Islam MdS, et al. Genomic insights of a methicillin-resistant biofilm-producing Staphylococcus aureus strain isolated from food handlers. BioMed Res Int. 2024;2024(1):5516117. https://doi.org/10.1155/2024/5516117
31. Batool N, Shamim A, Chaurasia AK, Kim KK. Genome-wide analysis of Staphylococcus aureus sequence Type 72 isolates provides insights into resistance against antimicrobial agents and virulence potential. Front Microbiol. 2021;11:613800. https://doi.org/10.3389/fmicb.2020.613800
32. Chopjitt P, Wongsurawat T, Jenjaroenpun P, et al. Draft Genome Sequence of Methicillin-Resistant Staphylococcus aureus harboring staphylococcal cassette chromosome mec Type IX, Isolated from a fatal bacteremic pneumonia case. Dunning Hotopp JC, editor. Microbiol Resour Announc. 2021;10(30):e00616-21. https://doi.org/10.1128/MRA.00616-21
33. Islam S, Nasrin N, Tithi NS, et al. Antimicrobial Susceptibility and Genomic profiles of multidrug-resistant Staphylococcus aureus from nasopharynx of asymptomatic children in Dhaka, Bangladesh. Life. 2024;14(8):971. https://doi.org/10.3390/life14080971
34. Balushi MA, Kumar R, Al-Rashdi A, et al. Genomic analysis of the emerging carbapenem-resistant Klebsiella pneumoniae sequence type 11 harbouring Klebsiella pneumoniae carbapenemase (KPC) in Oman. J Infect Public Health. 2022;15(10):1089–1096. https://doi.org/10.1016/j.jiph.2022.08.014
35. Chen L, Zhou Y, Wang S, et al. Genomic analysis of carbapenem-resistant hypervirulent Klebsiella pneumoniae in a Chinese tertiary hospital. Infect Drug Resist. 2023;16:6385–6394. https://doi.org/10.2147/IDR.S425949
36. Chukamnerd A, Pomwised R, Jeenkeawpiam K, Sakunrang C, Chusri S, Surachat K. Genomic insights into blaNDM-carrying carbapenem-resistant Klebsiella pneumoniae clinical isolates from a university hospital in Thailand. Microbiol Res. 2022;263:127136. https://doi.org/10.1016/j.micres.2022.127136
37. Cienfuegos-Gallet AV, Zhou Y, Ai W, Kreiswirth BN, Yu F, Chen L. Multicenter genomic analysis of carbapenem-resistant Klebsiella pneumoniae from Bacteremia in China. Microbiol Spectr. 2022;10(2):e02290-21. https://doi.org/10.1128/spectrum.02290-21
38. Liu Z, Chen R, Xu P, Wang Z, Li R. Characterization of a blaNDM-1-bearing IncHI5-like plasmid from Klebsiella pneumoniae of infant origin. Front Cell Infect Microbiol. 2021;11:738053. https://doi.org/10.3389/fcimb.2021.738053
39. Pei N, Liu X, Jian Z, et al. Genome sequence and genomic analysis of liver abscess caused by hypervirulent Klebsiella pneumoniae. 3 Biotech. 2023;13(3):76. https://doi.org/10.1007/s13205-023-03458-6
40. Sheng J, Cave R, Ter-Stepanyan MM, et al. Emergence of mcr-8.1 -bearing MDR-hypervirulent Klebsiella pneumoniae ST307. Microbiol Spectr. 2024;13(1):e01910-24. https://doi.org/10.1128/spectrum.01910-24
41. Shi Q, Han X, Huang Q, et al. The genetic characteristics and carbapenem resistance mechanism of ST307 Klebsiella pneumoniae coharbouring blaCMY-6, blaOXA-48, and a Truncated blaNDM-1. Antibiotics. 2022;11(11):1616. https://doi.org/10.3390/antibiotics11111616
42. Tian X, Zhang L, Li C, Xia D, Ying J. Genome sequence of a sequence type 1 NDM-5-producing carbapenem-resistant Klebsiella pneumoniae in China. J Glob Antimicrob Resist. 2024;38:271–274. https://doi.org/10.1016/j.jgar.2024.05.001
43. Wang L, Zhu M, Yan C, et al. Class 1 integrons and multiple mobile genetic elements in clinical isolates of the Klebsiella pneumoniae complex from a tertiary hospital in eastern China. Front Microbiol. 2023;14:985102. https://doi.org/10.3389/fmicb.2023.985102
44. Xu J, Zhao Z, Ge Y, He F. Unravelling the genome sequence of NDM-1 and KPC-2 co-producing Klebsiella pneumoniae ST11 isolated from a bloodstream infection. J Glob Antimicrob Resist. 2020;20:339–341. https://doi.org/10.1016/j.jgar.2020.01.021

45. Yang Q, Jia X, Zhou M, et al. Emergence of ST11-K47 and ST11-K64 hypervirulent carbapenem-resistant Klebsiella pneumoniae in bacterial liver abscesses from China: A molecular, biological, and epidemiological study. Emerg Microbes Infect. 2020;9(1):320–331. https://doi.org/10.1080/22221751.2020.1721334

46. Zhang W, Wang Y, Wang K, et al. Hybrid sequencing-based genomic analysis of Klebsiella pneumoniae from urinary tract infections among inpatients at a tertiary hospital in Beijing. Infect Drug Resist. 2024;17:1447–1457. https://doi.org/10.2147/IDR.S448253

47. Farlow J, Nozadze M, Mitaishvili N, et al. Comparative genomic analysis of four multidrug-resistant isolates of Acinetobacter baumannii from Georgia. J Glob Antimicrob Resist. 2020;21:363–368. https://doi.org/10.1016/j.jgar.2019.11.002

48. Khan MAS, Chaity SC, Hosen MA, Rahman SR. Genomic epidemiology of multidrug-resistant clinical Acinetobacter baumannii in Bangladesh. Infect Genet Evol. 2024;123:105656. https://doi.org/10.1016/j.meegid.2024.105656

49. He J, Lu X, Yuan C, et al. Genetic characteristics of novel IncpSE5381-aadB plasmids, integrative and mobilizable elements, and integrative and conjugative elements in Pseudomonas aeruginosa. Infect Drug Resist. 2024;17:2053–2068. https://doi.org/10.2147/IDR.S462670

50. Amoudizaj FF, Aghayi E, Matin MG, Soltani N, Mala P. Antibiotic resistance pattern and frequency of PER-1, SHV-1 and AMPC Type B-lactamase genes in Pseudomonas aeruginosa isolated from clinical samples. Open Microbiol J. 2019;13(1):308–312. https://doi.org/10.2174/1874285801913010308

51. Chukamnerd A, Pomwised R, Chusri S, et al. Antimicrobial susceptibility and molecular features of colonizing isolates of Pseudomonas aeruginosa and the report of a novel sequence type (ST) 3910 from Thailand. Antibiotics. 2023;12(1):165. https://doi.org/10.3390/antibiotics12010165

52. Hu Z, Zhou L, Tao X, et al. Antimicrobial resistance survey and whole-genome analysis of nosocomial P. Aeruginosa isolated from eastern Province of China in 2016–2021. Ann Clin Microbiol Antimicrob. 2024;23(1):12. https://doi.org/10.1186/s12941-023-00656-1

53. Dong X, Zhu M, Li Y, et al. Whole-genome sequencing-based species classification, multilocus sequence typing, and antimicrobial resistance mechanism analysis of the Enterobacter cloacae complex in Southern China. Microbiol Spectr. 2022;10(6):e02160-22. https://doi.org/10.1128/spectrum.02160-22

54. Gaber Y, TumAllah HM, AbdelAllah NH, et al. Emergence and genomic characterization of a spa Type t4407 ST6-SCCmec Type IVa methicillin-resistant Staphylococcus aureus strain isolated from Al-Karak Hospital, Jordan. Medicina (Mex). 2024;60(2):295. https://doi.org/10.3390/medicina60020295

55. Yasir M, Subahi AM, Shukri HA, et al. Bacterial community and genomic analysis of carbapenem-resistant Acinetobacter baumannii isolates from the environment of a health care facility in the western region of Saudi Arabia. Pharmaceuticals. 2022;15(5):611. https://doi.org/10.3390/ph15050611

56. Chang J, Zhang Y, Zhang Z, et al. Prevalence, antimicrobial resistance, and genetic characteristics of Staphylococcus aureus isolates in frozen flour and rice products in Shanghai, China. Curr Res Food Sci. 2023;7:100631. https://doi.org/10.1016/j.crfs.2023.100631

57. Zendri F, Isgren CM, Devaney J, Schmidt V, Rankin R, Timofte D. Resistome-based surveillance identifies ESKAPE pathogens as the predominant gram-negative organisms circulating in veterinary hospitals. Front Microbiol. 2023;14:1252216. https://doi.org/10.3389/fmicb.2023.1252216

58. Torres RT, Cunha MV, Ferreira H, Fonseca C, Palmeira JD. A high-risk carbapenem-resistant Pseudomonas aeruginosa clone detected in red deer (Cervus elaphus) from Portugal. Sci Total Environ. 2022;829:154699. https://doi.org/10.1016/j.scitotenv.2022.154699

59. Lopes J, De Lencastre H, Conceição T. Genomic analysis of Enterococcus faecium from non-clinical settings: Antimicrobial resistance, virulence, and clonal population in livestock and the urban environment. Front Microbiol. 2024;15:1466990. https://doi.org/10.3389/fmicb.2024.1466990

60. Coppens J, Xavier BB, Vlaeminck J, et al. Genomic analysis of methicillin-resistant Staphylococcus aureus clonal complex 239 isolated from Danish patients with and without an international travel history. Front Microbiol. 2022;13:1016829. https://doi.org/10.3389/fmicb.2022.1016829

61. Wolff N, Hendling M, Schroeder F, et al. Full pathogen characterisation: Species identification including the detection of virulence factors and antibiotic resistance genes via multiplex DNA-assays. Sci Rep. 2021;11(1):6001. https://doi.org/10.1038/s41598-021-85438-5

62. Fox V, Mangioni D, Renica S, et al. Genomic characterization of Klebsiella pneumoniae carbapenemase-producing Klebsiella pneumoniae (KPC-Kp) strains circulating in three university hospitals in Northern Italy over three years. Antimicrob Resist Infect Control. 2024;13(1):70. https://doi.org/10.1186/s13756-024-01429-x

63. Garcia-Gonzalez N, Fuster B, Tormo N, Salvador C, Gimeno C, Gonzalez-Candelas F. Genomic analysis of the initial dissemination of carbapenem-resistant Klebsiella pneumoniae clones in a tertiary hospital. Microb Genom. 2023;9(6):mgen001032. https://doi.org/10.1099/mgen.0.001032

64. Kuzina ES, Kislichkina AA, Sizova AA, et al. High-molecular-weight plasmids carrying carbapenemase genes blaNDM-1, blaKPC-2, and blaOXA-48 coexisting in clinical Klebsiella pneumoniae strains of ST39. Microorganisms. 2023;11(2):459. https://doi.org/10.3390/microorganisms11020459

65. Martin MJ, Corey BW, Sannio F, et al. Anatomy of an extensively drug-resistant Klebsiella pneumoniae outbreak in Tuscany, Italy. Proc Natl Acad Sci USA. 2021;118(48):e2110227118. https://doi.org/10.1073/pnas.2110227118

66. Chapartegui-González I, Lázaro-Díez M, Redondo-Salvo S, Navas J, Ramos-Vivas J. Antimicrobial resistance determinants in genomes and plasmids from Acinetobacter baumannii clinical isolates. Antibiotics. 2021;10(7):753. https://doi.org/10.3390/antibiotics10070753

67. Valcek A, Nesporova K, Whiteway C, et al. Genomic analysis of a strain collection containing Multidrug-, extensively drug-, pandrug-, and carbapenem-resistant modern clinical isolates of Acinetobacter baumannii. Antimicrob Agents Chemother. 2022;66(9):e00892-22. https://doi.org/10.1128/aac.00892-22

68. Hernández-García M, González De Aledo M, Ponce-Alonso M, et al. Simultaneous clonal spread of NDM-1–producing Pseudomonas aeruginosa ST773 from Ukrainian patients in the Netherlands and Spain. IJID Reg. 2024;12:100415. https://doi.org/10.1016/j.ijregi.2024.100415

69. Torrens G, Van Der Schalk TE, Cortes-Lara S, et al. Susceptibility profiles and resistance genomics of Pseudomonas aeruginosa isolates from European ICUs participating in the ASPIRE-ICU trial. J Antimicrob Chemother. 2022;77(7):1862–1872. https://doi.org/10.1093/jac/dkac122

70. Valenza G, Eisenberger D, Esse J, et al. High prevalence of the recently identified clonal lineage ST1299/CT3109 vanA among vancomycin-resistant Enterococcus faecium strains isolated from municipal wastewater. mSphere. 2024;9(9):e00396-24. https://doi.org/10.1128/msphere.00396-24

71. Di Cesare A, Cornacchia A, Sbaffi T, et al. Treated wastewater: A hotspot for multidrug- and colistin-resistant Klebsiella pneumoniae. Environ Pollut. 2024;359:124598. https://doi.org/10.1016/j.envpol.2024.124598

72. Sakkas H, Bozidis P, Ilia A, Mpekoulis G, Papadopoulou C. Antimicrobial resistance in bacterial pathogens and detection of carbapenemases in Klebsiella pneumoniae isolates from hospital wastewater. Antibiotics. 2019;8(3):85. https://doi.org/10.3390/antibiotics8030085

73. Belloso Daza MV, Milani G, Cortimiglia C, Pietta E, Bassi D, Cocconcelli PS. Genomic insights of Enterococcus faecium UC7251, a multi-drug resistant strain from ready-to-eat food, highlight the risk of antimicrobial resistance in the food chain. Front Microbiol. 2022;13:894241. https://doi.org/10.3389/fmicb.2022.894241

74. Naushad S, Nobrega DB, Naqvi SA, Barkema HW, De Buck J. Genomic analysis of bovine Staphylococcus aureus isolates from milk to elucidate diversity and determine the distributions of antimicrobial and virulence genes and their association with mastitis. mSystems. 2020;5(4):e00063-20. https://doi.org/10.1128/mSystems.00063-20

75. Selvaraj Anand S, Wu CT, Bremer J, et al. Identification of a novel CG307 sub-clade in third-generation-cephalosporin-resistant Klebsiella pneumoniae causing invasive infections in the USA. Microb Genomics. 2024;10(2):001201. https://doi.org/10.1099/mgen.0.001201

76. VanNatta M, Grier L, Khan MH, et al. In vivo emergence of pandrug-resistant Acinetobacter baumannii strain: Comprehensive resistance characterization and compassionate use of sulbactam-durlobactam. Open Forum Infect Dis. 2023;10(10):ofad504. https://doi.org/10.1093/ofid/ofad504

77. Souza GÁAD, Almeida ACD, Xavier MADS, et al. Characterization and molecular epidemiology of Staphylococcus aureus strains resistant to beta-lactams isolated from the milk of cows diagnosed with subclinical mastitis. Vet World. 2019;12(12):1931–1939. https://doi.org/10.14202/vetworld.2019.1931-1939

78. Vasconcelos PC, Leite EL, Saraiva MMS, et al. Genomic analysis of a community-acquired methicillin-resistant Staphylococcus aureus sequence Type 1 associated with caprine mastitis. Pathogens. 2023;13(1):23. https://doi.org/10.14202/vetworld.2019.1931-1939

79. Davies YM, Cunha MPV, Dropa M, et al. Pandemic clones of CTX-M-15 producing Klebsiella pneumoniae ST15, ST147, and ST307 in companion parrots. Microorganisms. 2022;10(7):1412. https://doi.org/10.3390/microorganisms10071412

80. Guillén R, Salinas C, Mendoza-Álvarez A, et al. Genomic epidemiology of the primary methicillin-resistant Staphylococcus aureus clones causing invasive infections in Paraguayan children. Microbiol Spectr. 2024;12(4):e03012-23. https://doi.org/10.1128/spectrum.03012-23

81. Jure MA, Albarracin L, Vargas JM, et al. Draft genome sequences of two hypermucoviscous carbapenem-resistant ST25 Klebsiella pneumoniae strains causing respiratory and systemic infections. J Glob Antimicrob Resist. 2021;26:174–176. https://doi.org/10.1016/j.jgar.2021.05.018

82. Veloso M, Arros P, Acosta J, et al. Antimicrobial resistance, pathogenic potential, and genomic features of carbapenem-resistant Klebsiella pneumoniae isolated in Chile: High-risk ST25 clones and novel mobile elements. Microbiol Spectr. 2023;11(5):e00399-23. https://doi.org/10.1128/spectrum.00399-23

83. Nodari CS, Cayô R, Streling AP, et al. Genomic analysis of carbapenem-resistant Acinetobacter baumannii isolates belonging to major endemic clones in South America. Front Microbiol. 2020;11:584603. https://doi.org/10.3389/fmicb.2020.584603

84. Shrestha P, He S, Legido-Quigley H. Antimicrobial resistance research collaborations in Asia: Challenges and opportunities to equitable partnerships. Antibiotics. 2022;11(6):755. https://doi.org/10.3390/antibiotics11060755

85. Iskandar K, Molinier L, Hallit S, et al. Surveillance of antimicrobial resistance in low- and middle-income countries: A scattered picture. Antimicrob Resist Infect Control. 2021;10(1):63. https://doi.org/10.1186/s13756-021-00931-w

86. Malik SS, Mundra S. Increasing consumption of antibiotics during the COVID-19 pandemic: Implications for patient health and emerging anti-microbial resistance. Antibiotics. 2022;12(1):45. https://doi.org/10.3390/antibiotics12010045

87. World Health Organization. WHO reports widespread overuse of antibiotics in patients hospitalized with COVID-19 [homepage on the Internet]. 2024 [cited 2025 Apr 4]. Available from: https://www.who.int/news/item/26-04-2024-who-reports-widespread-overuse-of-antibiotics-in-patients--hospitalized-with-covid-19

88. Abdelaziz Abdelmoneim S, Mohamed Ghazy R, Anwar Sultan E, Hassaan MA, Anwar Mahgoub M. Antimicrobial resistance burden pre and post-COVID-19 pandemic with mapping the multidrug resistance in Egypt: A comparative cross-sectional study. Sci Rep. 2024;14(1):7176. https://doi.org/10.1038/s41598-024-56254-4

89. Somda NS, Nyarkoh R, Kotey FCN, Tetteh-Quarcoo PB, Donkor ES. A systematic review and meta-analysis of carbapenem-resistant Enterobacteriaceae in West Africa. BMC Med Genomics. 2024;17(1):267. https://doi.org/10.1186/s12920-024-02043-x

90. Navon-Venezia S, Kondratyeva K, Carattoli A. Klebsiella pneumoniae: A major worldwide source and shuttle for antibiotic resistance. FEMS Microbiol Rev. 2017;41(3):252–275. https://doi.org/10.1093/femsre/fux013

91. Teixeira P, Tacão M, Pureza L, et al. Occurrence of carbapenemase-producing Enterobacteriaceae in a Portuguese river: blaNDM, blaKPC and blaGES among the detected genes. Environ Pollut. 2020;260:113913. https://doi.org/10.1016/j.envpol.2020.113913

92. Hazen TH, Mettus R, McElheny CL, et al. Diversity among blaKPC-containing plasmids in Escherichia coli and other bacterial species isolated from the same patients. Sci Rep. 2018;8(1):10291. https://doi.org/10.1038/s41598-018-28085-7