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A systematic review and meta-analysis of carbapenem-resistant Enterobacteriaceae in West Africa

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Abstract

Background In Africa, the problem of carbapenem-resistant Enterobacteriaceae (CRE) is aggravated by many factors. This systematic review attempted to describe the current status of the molecular epidemiology of carbapenem resistance in West Africa (WA).

Methods Articles published from 16 West African countries on the molecular epidemiology of carbapenem resistance were reviewed. An extensive literature search was carried out in PubMed, Scopus, Web of Science, and African Journals Online (AJOL) using specific keywords. The meta-analysis and forest plots of major pathogens and carbapenem resistance genes were done using the Open Meta-Analyst, Task Order # 2 software. The data were analysed in binary random model effects by the DerSimonian-Laird method at a 95% confidence interval.

Results Of the 431 articles found in our initial search, 60 (13.92%) were considered suitable for inclusion. Only seven of the 16 West African countries formed part of the analysis, Nigeria (23/60), Ghana (19/60), Burkina Faso (7/60), Senegal (6/60), Benin (2/60), Mali (2/60), and Togo (1/60). Also, 80% (48/60) of the studies used clinical samples, 16.67% (10/60) used environmental samples, and 3.33% (2/60) used animal samples. The average prevalence was highest in *Acinetobacter baumannii* (18.6%; 95% CI = 14.0-24.6, $I^2 = 97.9\%$, $p < 0.001$), followed by *Pseudomonas aeruginosa* (6.5%; 95% CI = 3.1–13.4, $I^2 = 96.52\%$, $p < 0.001$), *Klebsiella pneumoniae* (5.8%; 95% CI = 4.2–7.9, $I^2 = 98.06\%$, $p < 0.001$) and *Escherichia coli* (4.1%; 95% CI = 2.2–7.7, $I^2 = 96.68\%$, $p < 0.001$). The average prevalence of the *blaNDM* gene was 10.6% (95% CI = 7.9–14.3, $I^2 = 98.2\%$, $p < 0.001$), followed by 3.9% (95% CI: 1.8–8.3, $I^2 = 96.73\%$, $p < 0.001$) for *blaVIM* and 3.1% (95% CI: 1.7–5.8, $I^2 = 91.69\%$, $p < 0.001$) for *blaOXA*-48.

Conclusion In West Africa, *K. pneumoniae*, *E. coli*, *A. baumannii*, and *P. aeruginosa* are the main CRE with *blaNDM*, *blaVIM*, and *blaOXA*-48 being the predominant carbapenem resistance genes. In view of these results, ongoing CRE surveillance combined with antimicrobial stewardship improved, laboratory detection methods, and adherence to infection control practices will be needed to control the spread of CRE.

Keywords West Africa, Molecular epidemiology, Carbapenem-resistance, Meta-analysis

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Background

Although a natural phenomenon in bacteria and other microorganisms, antimicrobial resistance (AMR) has become accelerated owing to excessive use, including abuse, of antimicrobials. Bacteria exhibit these traits via a multitude of mechanisms, the nature and effectiveness of which vary per the species involved and their origin [1]. Key among these mechanisms are mutations and acquisition of external genetic material. Especially unsettling is the fact that the magnitude of the AMR problem has become particularly multidimensionally precarious in the 21st Century [2]. According to predictive statistical models, an estimated 4.95 million deaths were associated with bacterial AMR in 2019, 1.27 million of which were directly caused by bacterial AMR [2]. These enormous ramifications of AMR are considerably higher in low-income countries, with the conspicuous data gaps in most parts of these regions suggesting these appraisals to be worse than estimated [2, 3].

In the AMR landscape, carbapenems, which are often used as last-line drugs against bacterial infections, are now a major focus of keen interest, due to emergence and unbridled global spread of bacterial resistance against them [4]. This phenomenon is particularly notable in species of Gram-negative bacteria that naturally have relatively low transmembrane diffusion coefficients to β-lactams, such as *Enterobacter cloacae*, *Pseudomonas aeruginosa*, and *Acinetobacter baumannii*. It also remains much more common in hospital settings than in communities [5]. Most recent studies have shown that the global occurrence of carbapenem resistance (CR) among members of the Enterobacteriaceae (especially, *Klebsiella pneumoniae*, *Escherichia coli*, *Enterobacter* spp., and *Citrobacter freundii*), whether in livestock, the environment, or in hospital- and community-associated infections, impose a huge burden on the healthcare system [6–8]. More concerningly, as carbapenemase genes reside on mobile genetic elements (such as transposons and plasmids), they have a high potential for widespread transmission to a vast spectrum of bacterial genera. In addition, Enterobacteriaceae that harbour carbapenemase-encoding genes can spread from person to person, making CRE a deadly threat within and outside healthcare facilities [9].

In Africa, the problem of carbapenem-resistant Enterobacteriaceae (CRE) is aggravated by factors such as the high rate of infections, poor diagnostic tools, sub-optimal disease surveillance systems, abuse of antibiotics, and dearth of CRE data [10]. In East Africa, high prevalence of carbapenem resistance have been reported in clinical isolates of *A. baumannii*, *P. aeruginosa*, *K. pneumoniae*, *Proteus mirabilis*, and *E. coli* [11]. In West Africa (WA), several studies have reported on the occurrence of CRE, but these rarely conducted detailed analyses on the molecular distribution and epidemiology of CRE in healthcare facilities. Besides, data on CRE differs among countries, are limited, and are not currently available in scientific publications for some countries. To identify data gaps and add to the existing knowledge to inform the scientific community and policymakers, we undertook a systematic review to assess the current status of the molecular epidemiology of carbapenem resistance in West Africa. The main objectives were to (i) evaluate the evolution of carbapenem resistance of bacteria isolated in WA over the years, (ii) determine the pathogens implicated the most, and (iii) identify the predominant carbapenem resistance genes involved.

Methods

Study design

This was a systematic review of the available studies on CRE conducted in 16 countries of West Africa, including Benin, Burkina Faso, Cape Verde, Ivory Coast, Gambia, Ghana, Guinea, Guinea-Bissau, Liberia, Mali, Mauritania, Niger, Nigeria, Senegal, Sierra Leone, and Togo. They included studies that were available in PubMed, Scopus, Web of Science, and African Journals Online (AJOL) databases, following a literature search using the keywords "carbapenem resistance, carbapenemase-producing, Metallo-β-lactamase, West Africa, molecular epidemiology, and the country name". Only articles published in the English and French languages were included.

Study eligibility criteria

The results of the literature search were exported and compiled. Endnote was then used to remove duplicates and to catalogue, collate, and manage the publications. The included studies were available full-text research articles reporting on the prevalence of carbapenemase-producing bacteria isolated in West African countries, those reporting on the study population and the phenotypic and molecular methods used to detect carbapenem resistance. The literature search was conducted from April 8, 2024 to May 20, 2024 and included all publications before May 20, 2024.

Data extraction

Full texts of the screened publications were obtained from appropriate sources and the data were extracted in an MS Excel spreadsheet under multiple headings, such as study period, publication year, country, source of samples, number of isolates, number of carbapenem-resistant isolates, CR prevalence, bacteria, methods for CR gene detection, number of CR genes, and prevalence of CR genes. Articles showing studies with incomplete information related to AMR detection methods, only phenotypic methods, duplicate articles, abstracts, review articles, letters, short communications, posters,

conference proceedings, and studies outside WA countries were excluded.

Data analysis

Initially, a total of 431 potential articles were identified. All titles and abstracts that were related to the study questions were reviewed. Publications were further screened by reviewing their full details, and selected articles were further evaluated. Only those that met the inclusion criteria were included in this review (Fig. 1). The extracted data were used for descriptive statistics. Further analysis was carried out in multiple steps. Excel 2013 was used for data entry and some graphics. The meta-analysis and forest plots of major pathogens and carbapenem resistance genes, as well as estimation of the country effect, were done using the Open Meta-Analyst, Task Order # 2 software (available at https://www.brown.edu/academics/public-health/research/evidence-based-medicine/research-initiatives/software-0). The data were analysed in binary random model effects by the DerSimonian-Laird method at a 95% confidence interval. Individual models were used for analysis of each major pathogen. Inconsistency (or heterogeneity) across the studies estimated in the random-effects model was quantified using inverse variance index (I^2). The I^2 values at 25%, 50%, and 75% were considered as low, moderate, and high heterogeneity, respectively [12]. Significance levels were set at $p<0.001$.

Results

Systematic literature review and study

A total of 60 articles presenting concrete information about the molecular epidemiology of CR in WA were included in the final analysis after screening 431 articles from different electronic databases (Fig. 1). The distribution of the included articles was Benin (2/60), Burkina Faso (7/60), Ghana (19/60), Mali (2/60), Nigeria (23/60), Senegal (6/60), and Togo (1/60) (Table 1), which constitute seven out of 16 West African countries. No article, per the set inclusion criteria, was from Cote d'Ivoire, Cape Verde, Gambia, Guinea, Guinea Bissau, Liberia, Mauritania, Niger, and Sierra Leone.

Distribution of the articles according to publication year

The results showed a high diversity in terms of the publication year. Only five studies were conducted between 2012 and 2017, and one (1.67%) in each of 2012, 2013, 2014, 2016, and 2017. The majority of the studies were done in 2023, followed by 20/60 (33.33%) in 2020. The years 2021 and 2022 recorded 9/60 (15.00%) publications each. Also, 5/60 (8.33%) articles were published in 2019 and 2/60 (3.33%) in 2018 (Table 1).

Distribution of the articles according to sample origin

Overall, 48/60 (80%) of the studies reviewed were on clinical samples, 10/60 (16.67%) were on environmental samples (four of which emanated from Nigeria [13–16]

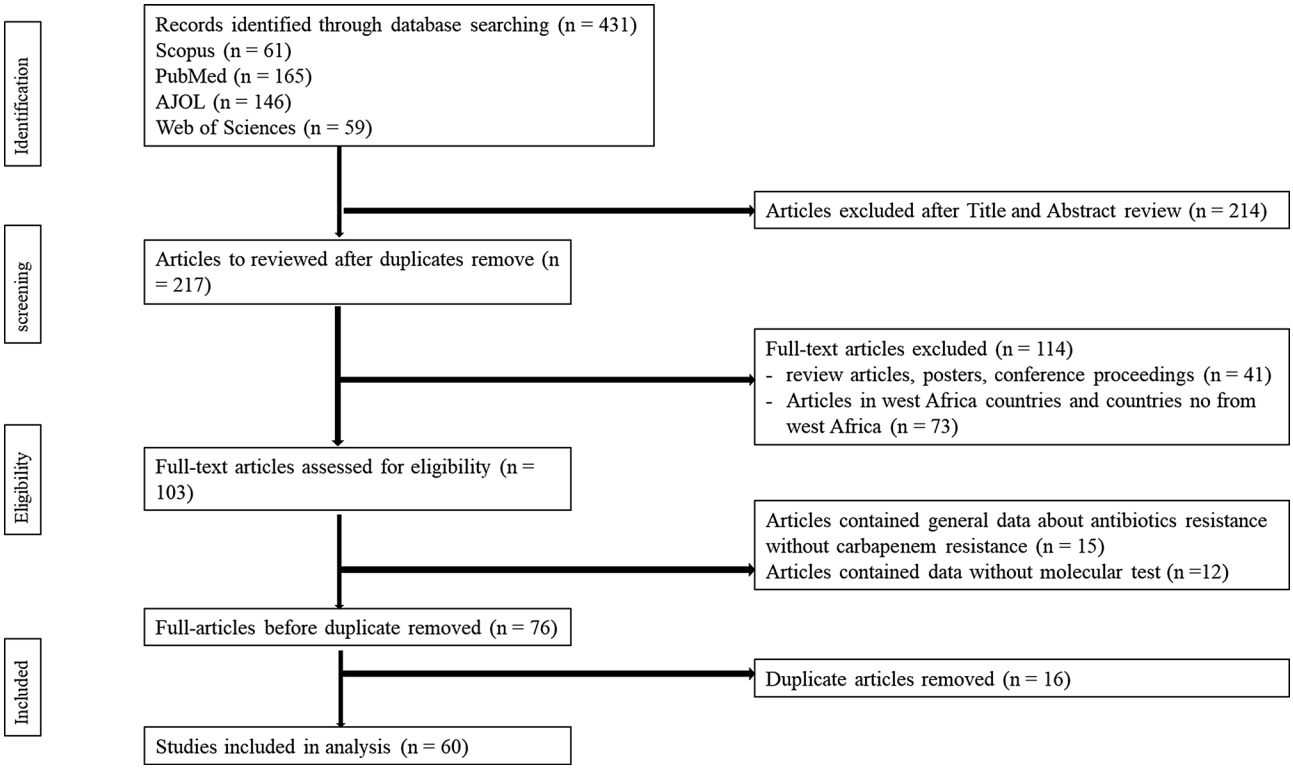


Fig. 1 Flow chart of systematic literature search, identification, screening, and article selection

Table 1 Review of West Africa-based carbapenem resistance studies

Origin	Numb of Isolates	Numb of CR	Methods	Carba genes	Numb carba genes	Prevalence of carba genes (%)	CR organisms	References	Location
Environment	256	45	PCR	*blaNDM-1*	30	66.7	*Klebsiella oxytoca*, *Klebsiella pneumoniae*, *Enterobacter aeroginosa*, *Enterobacter hormaechei*, *Enterobacter asburiae*, *Citrobacter freundii*, *Morganella morganii*, *E. coli*, *Proteus mirabilis*, *Enterobacter gergoviae*, *Klebsiella variicola*	[13]	Nigeria
Clinical	187	41	PCR	*blaNDM*, *blaOXA-48*	3; 12	1.6; 6.4	*E. coli*	[25]	Nigeria
Clinical	180	6	MALDI-TOF, Modified Hodge Test, RESIST-5 O.K.N.V.I, PCR	*blaNDM*, *blaOXA-48*, *blaVIM*	2; 2; 2	1.11 for each gene	*Escherichia coli*, *Pseudomonas aeruginosa*, *Pseudomonas mendocina*, *Enterobacter cloacae*, *Acinetobacter baumannii*	[26]	Benin
Environment	21	10	immu- nochromatographic test O.K.N.V.I. RESIST-5	*blaNDM*, *blaOXA-48*	5; 6	23.81; 28.57	*E. coli*, *Klebsiella pneumonia*	[17]	Burkina Faso
Environment	209	33	MALDI-TOF, PCR	*blaNDM*, *blaVIM*, *blaIMP*, *blaKPC*, *blaOXA-48*	23; 8; 6; 15; 3	11.0; 3.83; 2.87; 7.18; 1.44	*E. coli*, *Klebsiella pneumonia*	[18]	Burkina Faso
Clinical	473	25	immunochromato- graphic test O.K.N.V.I. RESIST-5	*blaNDM*, *blaOXA-48*, *blaVIM*	21; 5; 1	4.44; 1.06; 0.21	*E. coli*, *Klebsiella pneumonia*	[27]	Burkina Faso
Clinical	601	17	PCR, MLST	*blaNDM-1*, *blaOXA-58*, *bla0XA-181*, *blaVIM-2*	9; 1; 6; 1	1.5; 0.17; 1.0; 0.17	*Enterobacterales; A. baumannii; Pseudomonas aeruginosa*	[28]	Burkina Faso
Clinical	71	45	PCR, MALDI-TOF, WGS (using Illumina MiSeq), MLST	*blaNDM*, *blaOXA-48*	27; 1	38.03; 1.41	*Acinetobacter baylyi*, *Acinetobacter indicus*, *Acinetobacter pittii; Escherichia coli*, *Enterobacter bugandensis*, *Enterobacter cloacae*, *Escherichia*, *hermannii*, *Klebsiella pneumoniae*, *Leclercia*, *adecarboxylata*, *Pantoea agglomerans*, *Pseudomonas fluva*, *Pseudomonas stutzeri*, *Mixta calida*	[29]	Ghana
Clinical	62	2	PCR, WGS	*blaOXA-181*	2	3.23	*E. coli*	[30]	Ghana
Clinical	4	4	MALDI-TOF, sequencing (ONT and Illumina	*BlaDIM-1; blaIMP-1*	4; 4	100	*Pseudomonas*	[31]	Ghana
Clinical	3840	26	PCR	*blaNDM-1; blaVIM; blaOXA-48*	16; 8; 2	0.42; 0.21; 0.052	*P. aeruginosa*, *Acinetobacter species*, *E. coli*, *Pseudomonas putida*, *K. pneumoniae*, *Providencia stuartii*, *Shigella sonnei*,	[32]	Ghana
Clinical	36	4	PCR; MALDI-TOF; MLST; WGS (Illumina; ONT)	*blaOXA-23; blaOXA-51*	2; 2	5.56; 5.56	*Acinetobacter baumannii*	[33]	Ghana

Table 1 (continued)

Origin	Numb of Isolates	Numb of CR	Methods	Carba genes	Numb carba genes	Prevalence of carba genes (%)	CR organisms	References	Location
Clinical	45	22	MALDI-TOF; PFGE; MLST	*blaNDM-1*, *blaOXA-23*, *bla0XA-378*, *blaOXA-420*	20; 20; 3; 2	44.44; 44.44; 6.67; 4.44	*Acinetobacter baumannii*	[34]	Ghana
Clinical	230	13	PCR	*blaOXA-48; blaNDM-1*	11; 13	5.65; 4.78	*Proteus vulgaris*, *Proteus mirabilis*, *Citrobacter spp.; Klebsiella pneumoniae*, *E. coli*	[35]	Ghana
Clinical	144	5	PCR	*blaOXA-48; blaNDM*	4; 1	2.78; 0.69	*E. coli*, *Klebsiella pneumonia*	[36]	Ghana
Clinical	110	13	PCR	*blaNDM-1*	3	2.73	*Klebsiella pneumonia*, *Proteus mirabilis*	[37]	Mali
Clinical	240	27	MALDI-TOF; RT-PCR	*blaOXA-48; blaNDM-1*	14; 13	5.83; 5.42	*Escherichia coli*, *Klebsiella*, *pneumoniae*, *Enterobacter cloacae*, *Citrobacter freundii*	[38]	Senegal
Animal clinical	55	9	MALDI-TOF; PCR; RT-PCR; PFGE	*blaOXA-23*	9	16.36	*Acinetobacter baumannii*	[23]	Senegal
Clinical	53	15	PCR	*BlaNDM; blaVIM*	13; 5	24.53; 9.43	*E. coli*	[39]	Burkina Faso
Environment	183	18	PCR	*blaNDM-1*, *blaOXA-48*	14; 4	7.65; 2.19	*Escherichia coli*, *Klebsiella pneumoniae*, *Klebsiella oxytoca*	[19]	Burkina Faso
Clinical	29	29	WGS	*blaOXA-181*	29	100	*Klebsiella pneumoniae*	[40]	Ghana
Clinical	87	21	PCR	*blaNDM*	21	24.14	*Acinetobacter baumannii*	[41]	Ghana
Clinical	21	2	MALDI-TOF, MLST, PCR, WGS	*blaOXA – 181; blaNDM – 1*	1; 1	4.76; 4.76	*E. coli*	[42]	Ghana
Clinical	29	3	WGS, MLST, PCR	*blaOXA-48*, *blaOXA-181*	1; 2	3.45; 6.90	*K. quasipneumoniae*, *Klebsiella pneumonia*, *Enterobacter cloacae*	[43]	Ghana
Environment	36	25	WGS	*blaVIM*, *blaNDM-1*,	25	69.44	*Pseudomonas putida*, *Citrobacter werkmanii*,	[20]	Ghana
Clinical	382	2	PCR	*blaNDM-1*	2	0.52	*Klebsiella pneumoniae*	[44]	Ghana
Environment	174	61	PCR	*blaNDM-1*	61	35.06	*Acinetobacter spp.*, *Citrobacter freundii*, *Enterobacter spp.*, *Escherichia coli*, *Bacillus spp.*, *Klebsiella spp.*, *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Streptococcus agalactiae*, *Providencia spp.*, *Pseudomonas aeruginosa*, *Vibrio spp.*	[21]	Ghana
Clinical	181	5	PCR	*blaOXA-48*, *blaNDM-1*, *blaKPC*	3; 2; 2	1.66; 1.10; 1.10	*E. coli*, *K. pneumoniae*, *Acinetobacter baumannii*, *Providencia vermicola*	[45]	Ghana
Clinical	111	26	PCR, ERIC-PCR	*blaNDM-1*, *blaVIM-1; blaOXA-48*	16; 8; 2	14.4; 7.2; 1.8	*Acinetobacter baumannii*, *Pseudomonas aeruginosa*, *Escherichia coli; Klebsiella pneumonia*, *Pseudomonas putida; Providencia stuartii*, *Shigella sonnei*	[46]	Ghana
Clinical	50	1	PCR, MLST	*blaNDM-5*	1	2.0	*E. coli*	[47]	Mali
Animal	101	13	PCR, sequencing (ONT)	*blaOXA-48; blaKPC-2*	7; 6	6.93; 5,94	*Klebsiella pneumoniae*	[48]	Senegal

Table 1 (continued)

Origin	Numb of Isolates	Numb of CR	Methods	Carba genes	Numb carba genes	Prevalence of carba genes (%)	CR organisms	References	Location
Clinical	152	4	WGS, MLST, PCR	*blaNDM-5; blaOXA-181*	1; 3	0.66; 1.97	*Enterobacter cloacae, E. coli*	[49]	Togo
Clinical	67	12	PCR	*blaNDM, blaVIM, blaGES*	4; 5; 2	5.97; 7.46; 2.98	*Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa and Proteus spp.*	[50]	Nigeria
Clinical	66	57	MALDI-TOF, WGS(Illumina)	*blaNDM, blaOXA-23, blaOXA-66, blaOXA-98, blaOXA-58, blaVIM-5, blacphA, blaOXA- 66, blaPOM-1*	26; 6; 6; 3; 3; 4; 2; 1; 1	39.39; 9.1; 9.1; 4.55; 4.55; 6.1; 3.03; 1.52; 1.52	*Acinetobacter baumannii, Aeromonas hydrophila, Pseudomonas otitidis, Providencia rettgeri, Enterobacter cloacae, Escherichia coli, P. aeruginosa*	[51]	Nigeria
Clinical	306	21	PCR	*blaVIM, blaGES, blaNDM*	9; 10; 2	2.94; 3.27; 0.65	*E. coli, Pseudomonas spp., Proteus spp., Klebsiella spp.*	[52]	Nigeria
Clinical	119	30	Xpert1 Carba-R, RT-PCR	*blaNDM, blaVIM*	26; 4	28.85; 3.36	*Pseudomonas aeruginosa, Acinetobacter baumannii, Providencia rettgeri, Escherichia coli, Klebsiella pneumoniae, Enterobacter spp*	[53]	Nigeria
Environment	259	125	PCR, MALDI-TOF, WGS (using Illumina MiSeq)	*blaOXA-23; blaOXA-40; blaNDM; blaPON, blaVIM*	1; 2; 6; 2; 1	0.39; 0.72; 2.32; 0.72; 0.39	*Klebsiella pneumoniae, Enterobacter cloacae, Pseudomonas otitidis, Acinetobacter baumannii, Aeromonas caviae, Citrobacter freundii*	[14]	Nigeria
Animal	141	43	PCR	*blaIMP*	43	30.50	*E. coli, Pseudomonas aeruginosa, Klebsiella pneumonia*	[24]	Nigeria
Environment	65	61	PCR, WGS	*blaVIM-5*	61	94.85	*Pseudomonas, Stenotrophomonas, Cupriavidus, Burkholderia, Pandoraea, Ralstonia*	[16]	Nigeria
Clinical	45	33	PCR,	*blaNDM-1, blaNDM-5, blaOXA-48, blaOXA-181*	17; 12; 3; 1	37.78; 26.67; 6.67; 2.22	*E. coli*	[54]	Nigeria
	306	161	PCR	*blaVIM, blaNDM, blaGES*	9; 2; 10	2.94; 0.65; 3.27	*Klebsiella spp., E. coli; Pseudomonas spp. Proteus spp.*	[55]	Nigeria
Clinical	123	32	WGS (Illumina), MALDI-TOF	*blaNDM-1, blaVIM-2, blaVIM-5-like*	32; 4; 11	26.02; 3.25; 8.94	*Pseudomonas aeruginosa*	[56]	Nigeria
	34	34	WGS, PFGE	*blaOXA-51, blaOXA-23, blaNDM, blaOXA-58, blaOXA-420*	34; 17; 21; 15; 2; 2	100; 50; 61.76; 44.12; 5.88; 5.88	*Acinetobacter baumannii*	[57]	Nigeria
Clinical	128	62	PCR; MLST	*blaVIM, blaOXA-48, blaIMP, blaNDM, blaKPC*	55; 37; 29; 22; 17	43.0; 28.9; 22.7; 17.2; 13.3	*Klebsiella pneumonia*	[58]	Nigeria
Clinical	100	86	WGS	*balOXA-23; blaNDM-1; blaOXA-58*	30; 24; 10	34.9; 27.9; 11.6	*Acinetobacter baumannii*	[15]	Nigeria
Clinical	93	5	WGS; MLST	*blaNDM*	5	5.38	*Klebsiella quasipneumoniae subsp. similipneumoniae*	[59]	Nigeria

Table 1 (continued)

Origin	Numb of Isolates	Numb of CR	Methods	Carba genes	Numb carba genes	Prevalence of carba genes (%)	CR organisms	References	Location
Environment	77	36	WGS	*blaNDM-1*, *blaOXA-23*, *bla0XA-58*	24; 22; 7	31.2; 28.6; 9.1	*Acinetaubacter baumannii*	[60]	Nigeria
Clinical	52	12	MALDI-TOF; WGS	*blaNDM; blaOXA395'*	9; 3	17.31; 5.77	*E. coli*, *K. pneumoniae, and P. aeruginosa*	[61]	Nigeria
Clinical	200	5	PCR	*blaVIM*	5	2.5	*Pseudomonas aeruginosa*	[62]	Nigeria
Clinical	44	5	RT-PCR	*blaVIM*	5	11.36	*Pseudomonas spp.*	[63]	Nigeria
Clinical	8	3	PCR	*blaKPC*, *blaVIM*, *blaNDM*	3; 3; 3	37.5; 37.5; 37.5	*Klebsiella pneumonia*	[64]	Nigeria
Clinical	213	199	PCR	*blaNDM*	199	93.4	*Klebsiella pneumonia*	[65]	Nigeria
Clinical	29	26	PCR, MALDI	*blaOXA-51*, *blaOXA-23*, *blaNDM*	26; 26; 1	89.69; 89.69; 3.45	*Acinetobacter baumannii*	[66]	Senegal
Clinical	28	6	PCR, MALDI-TOF	*blaOXA-48*	6	21.43	*Klebsiella pneumonia*	[67]	Senegal
Clinical	4	4	PCR, Sequencing	*blaOXA-181*	4	100	*Escherichia coli*	[68]	Burkina Faso
Clinical	218	9	WGS, MLST	*blaOXA-181*, *blaNDM-1; blaOXA-48*	1; 2; 6	0.36; 0.71; 2.16	*Klebsiella pneumoniae*, *Escherichia coli*, *Enterobacter cloacae*,	[69]	Nigeria
Clinical	3	3	MALDI-TOF; MLST	*blaOXA-23*	3	100	*Acinetobacter baumannii*	[70]	Senegal
Clinical	2	2	Sequencing (ONT)	*blaNDM-1*	2	100	*Klebsiella pneumonia*,	[71]	Ghana
Clinical	1	1	MALDI-TOF, WGS(Illumina)	*blaNDM-1*, *blaOXA-58*, *blaOXA-558*	1; 1; 1	100	*Acinetobacter baumannii*	[72]	Benin
Environment	121	23	WGS (Illumina); MLST	*blaOXA-181*, *blaNDM*, *blaOXA-48*	16; 5; 2	13.22; 4.13; 1.65	*Escherichia coli*, *Klebsiella pneumonia*, *Enterobacter cloacae*	[22]	Ghana

Numb = number, CR = carbapenem resistance, carba = carbapenem, % = percent

and three each from Burkina Faso [17–19] and Ghana [20–22]), and 2/60 (3.33%) from animal samples (one each from Senegal [23] and Nigeria [24]) (Table 1). The environmental samples included wastewater, soil, sediment, effluent, and water sources. The animal samples were from cattle, poultry, termites, and chimpanzees. However, none of the studies was on food products. Regarding the studies involving clinical samples, 22/60 (36.67%) were on urine, 18/60 (30%) were on swabs (vaginal, wound, ear, eye, neonatal, and throat), 17/60 (28.33%) were on blood, 13/60 (21.67%) were on stool, and 11/60 (18.33%) were on pus. However, some clinical samples did not specify the type (Table 1). Among the genotypic methods used in detecting the carbapenem resistance genes, PCR (standard PCR and RT-PCR) was the predominant (42/60; 70%), followed by whole-genome sequencing (20/60; 33.33%), MALDI-TOF (17/60; 28.33%), MLST (13/60; 21.67%), PFGE (3/60; 5%), immunochromatographic test O.K.N.V.I. RESIST-5 (3/60; 5%), and Xpert1 Carba-R (1/60; 1.67%) (Table 1).

Prevalence of pathogens included

Five types of bacteria were mainly prevalent in the 60 different articles studied. *Klebsiella* spp. was the most reported across the publications ($n=34$; 56.67%) followed by *E. coli* ($n=27$; 45%), *Acinetobacter* spp. ($n=20$; 33.33%), *Pseudomonas* spp. ($n=17$; 28.33%), and *Enterobacter* spp. ($n=11$; 18.33%). Some pathogens, such as *Providencia* spp. ($n=8$; 13.33%), *Citrobacter* spp. ($n=10$; 16.67%), *Proteus* spp. ($n=9$; 15%), *Aeromonas* spp. ($n=3$; 5%), *Morganella* spp. ($n=3$; 5%), *Shigella* spp. ($n=2$: 3.33%), and *Vibrio* spp. ($n=1$; 1.67%), were less represented. The pooled prevalence of *Acinetobacter* spp. in the samples was 42.17% ($n=20$), followed by *E. coli* at 27.60% ($n=27$), *Klebsiella* spp. at 25.20% ($n=34$), *Pseudomonas* spp. at 16.55% ($n=17$), *Proteus* spp. at 7.94% ($n=4$), *Citrobacter* spp. at 6.8% ($n=5$), *Providencia* spp. at 6.36% ($n=7$), *Vibrio* spp. at 4.02% ($n=7$), *Enterobacter* spp. at 3.81% ($n=11$), *Morganella* spp. at 2.34% ($n=1$), *Aeromonas* spp. at 1.71% ($n=2$), and *Shigella* spp. at 0.9% ($n=2$).

Prevalence of CR bacteria in West Africa

There was a high heterogeneity in the prevalence of carbapenem-resistant bacteria isolated in the various studies. The average prevalence was highest in *A. baumannii* (18.6%; 95% CI=14.0–24.6, I^2=97.9%, p<0.001) (Fig. 2), followed by *P. aeruginosa* (6.5%; 95% CI=3.1–13.4, I^2=96.52%, p<0.001) (Fig. 3), *K. pneumoniae* (5.8%; 95% CI=4.2–7.9, I^2=98.06%, p<0.001) (Fig. 4), and *E. coli* (4.1%; 95% CI=2.2–7.7, I^2=96.68%, p<0.001) (Fig. 5). However, CR was lowest in *Providencia* spp. (1.6%; 95% CI=0.4–6.2, I^2=93.23%, p<0.001), *P. mirabilis* (3.8%), *Vibrio* spp. (3.45%), *E. cloacae* (2.4%; 95% CI=1.5–3.8, I^2=45.68%, p=0.048), *Morganella morganii* (2.34%), *Aeromonas* spp. (1.71%), *Citrobacter freundii* (1.47%), and *Shigella sonnei* (0.45%). The highest pooled prevalence of CR bacteria from the environment were in *A. baumannii* (16.92%), *E. coli* (10.15%), *K. pneumoniae* (7.67%), and *P. aeruginosa* (7.11%). *Vibrio* spp., *M. morganii*, and *Aeromonas* spp. were only found in environmental samples, with average prevalence of 3.45%, 2.34%, and 1.71%, respectively. The pooled prevalence of *E. cloacae*, *C. freundii*, *Providencia* spp., and *P. mirabilis* were least in environmental and clinical samples, at 2.09%, 1.73%, 1.15%, and 0.78%, respectively. *Shigella sonnei* was not isolated in environmental samples. The pooled prevalence of CR in the two studies on animal samples was highest in *P. aeruginosa* (11.35%), followed by *K. pneumoniae* (8.79%), and then *E. coli* (8.51%).

Prevalence of CR genetic determinants in West Africa

The *blaNDM* gene is the most widespread CR gene in West Africa, evidenced by its detection in most of the studies (n=46; 76.67%). The second most dominant genes were *blaOXA*-48 and *blaVIM* (n=20; 33.33% each), followed by *blaOXA*-23 (n=10; 16.67%). On the other hand, other genes like *blaOXA*-181 (n=8; 13.33%), *bla0XA*-58 (n=5; 8.33%), *blaKPC* (n=5; 8.33%), *blaIMP* (n=4; 6.67%), *blaGES* (n=4; 6.67%), *blaOXA*-51 (n=3; 5%) were least detected.

The pooled prevalence of the *blaNDM* gene in West Africa, as calculated from the reviewed publications (n=46), was 10.6% (95% CI=7.9–14.3, I^2=98.2%, p<0.001) (Fig. 6), comprising *blaNDM*-1 (30.39%) and *blaNDM*-5 (13.48%). The pooled prevalence of *blaVIM* and *blaOXA*-48, calculated from the reviewed publications (n=20), were 3.9% (95% CI=1.8–8.3, I^2=96.73%, p<0.001) and 3.1% (95% CI=1.7–5.8, I^2=91.69%, p<0.001) (Figs. 7 and 8), respectively. Also, the pooled prevalence of *blaOXA*-23 (n=10) was 26.6% (95% CI=16.1–44.0, I^2=94.57%, p<0.001); those of other genes were *blaOXA*-181 (9.4%; n=8; 95% CI=3.8–23.4, I^2=96.52%, p<0.001), *blaOXA*-58 (6.5%; n=5; 95%

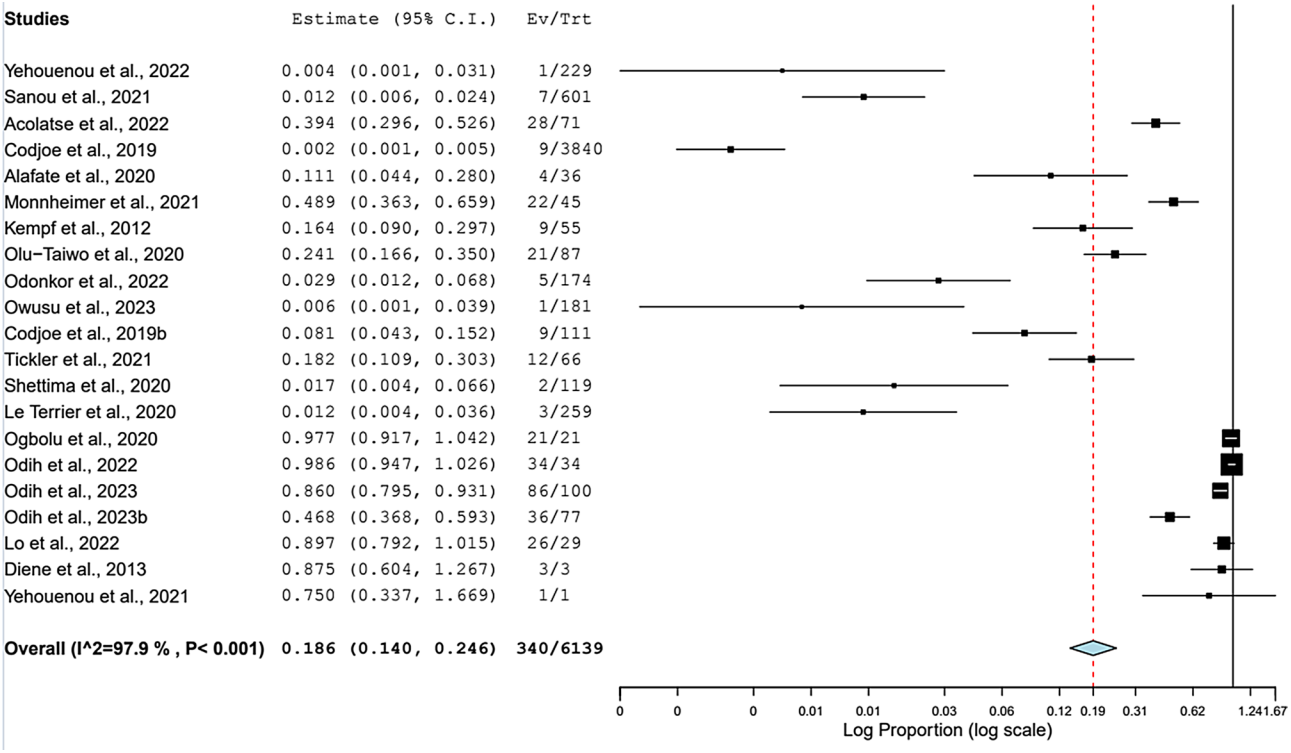


Fig. 2 Forest plot with adjusted average prevalence of carbapenem-resistance of *Acinetobacter baumannii* in West Africa. **Legend**: Random Effects Mode (95% CI = 14.0–24.6, I^2 = 97.9%, p < 0.001). X-axis is the proportion of the organism reported in individual studies as listed along the Y-axis, with the range of proportion in 95% confidence interval (CI)

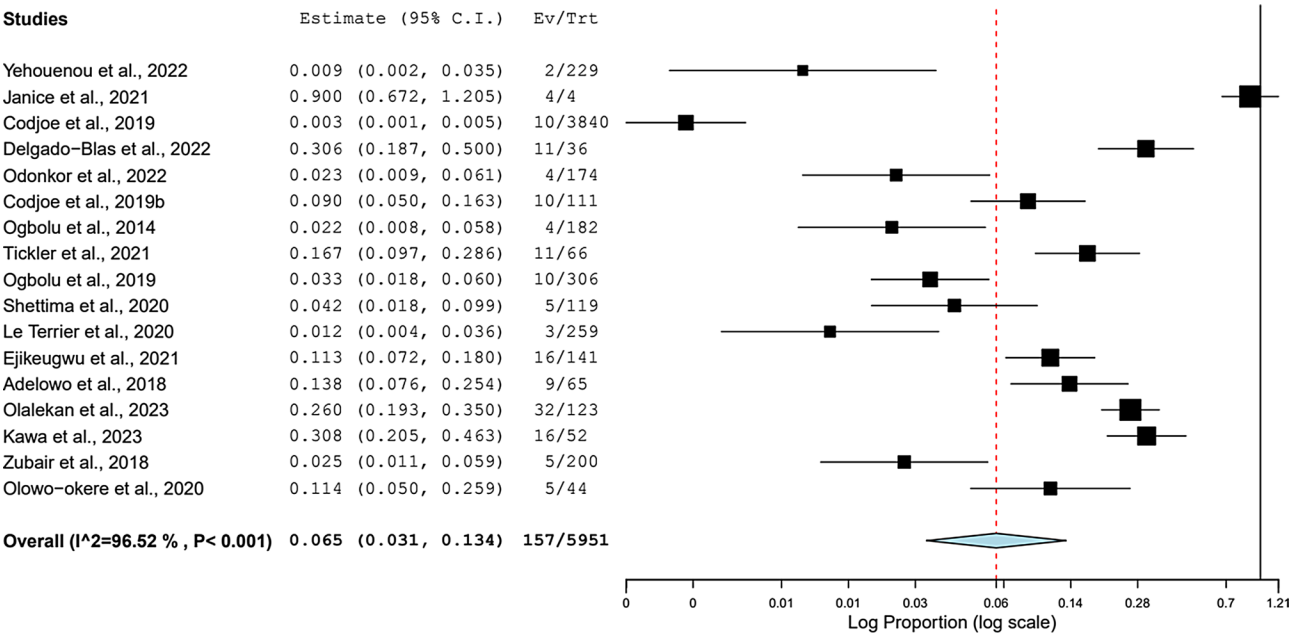


Fig. 3 Forest plot with adjusted average prevalence of carbapenem-resistance of *Pseudomonas aeruginosa* in West Africa. **Legend**: Random Effects Mode (95% CI = 3.1–13.4, $I^2 = 96.52\%$, $p < 0.001$). X-axis is the proportion of the organism reported in individual studies as listed along the Y-axis, with the range of proportion in 95% confidence interval (CI)

CI = 1.6–26.7, $I^2 = 90.46\%$, $p < 0.001$), *blaKPC* (8.4%; $n = 5$; 95% CI = 4.0–17.8, $I^2 = 82.8\%$, $p < 0.001$) (Fig. 9A), and *blaIMP* (22.1%; $n = 4$; 95% CI = 8.8–55.3, $I^2 = 96.55\%$, $p < 0.001$) (Fig. 9B). In the environmental samples, the average prevalence were *blaNDM* (28.45%; $n = 9$), *blaVIM* (21.88%; $n = 4$), *blaOXA*-48 (4.89%; $n = 4$), *bla0XA*-181 (13.22%, $n = 1$), *blaKPC* (7.18%; $n = 1$), and *blaIMP* (3.83%; $n = 1$). The two studies on the animal samples had a pooled prevalence of 6.93% for *blaOXA*-48, 5.93% for *blaKPC*, and 30.5% for *blaIMP* [48] (Table 1).

Distribution of CR genetic determinants by countries in West Africa

The information on the prevalence of carbapenem resistance genes in West Africa varies considerably among countries and depends on the genes detected (Table 2). The highest pooled prevalence of *blaNDM* was in Nigeria (17.42%; 95% CI = 12.6–23.9, $I^2 = 97.87\%$, $p < 0.001$) and the lowest was in Togo (0.66%). In the case of *blaVIM*, the highest pooled prevalence was in Nigeria (5.8%) and the lowest was in Benin (0.87%). The prevalence of *blaOXA*-48 ranged from 0.87% in Benin to 9.3% in Senegal. That of *blaOXA*-23 was 17.5% in Nigeria, 53.5% in Senegal, and 19.2% in Ghana. On the other hand, the prevalence of *blaOXA*-181 was 100% in Burkina Faso, 14.3% in Ghana, 1.97% in Togo, and 1.29% in Nigeria. The *blaOXA*-58 gene recorded a prevalence of 100% in Benin, 8.3% in Nigeria, and 0.17% in Burkina Faso. Those of *blaIMP* were 100%, 26.2%, and 3.83% in Ghana, Nigeria, and Burkina Faso, respectively. The prevalence of *blaKPC* in Nigeria, Burkina Faso, and Senegal were 20.7%, 7.18%, and 5.94%, respectively. The *blaGES* gene was only found in Nigeria (6.3%; 95% CI = 0.005–0.760, $I^2 = 98.33\%$, $p < 0.001$) (Table 2).

Average prevalence of CR genetic determinants detected in the pathogens

Nigeria

The prevalence of *blaNDM* was highest in *A. baumannii*, followed by *K. pneumoniae* at 14.05%, *P. aeruginosa* at 8.48%, *E. coli* at 8.09%, and *E. cloacae* at 1.32%. The highest prevalence of *blaVIM* were seen in 10.28% of *K. pneumoniae* cases, 5.01% of *P. aeruginosa* cases, and 0.76% of *E. coli* cases. For *K. pneumoniae*, *blaOXA*-48 was detected in 14.68% of the samples, 4.5% in *E. coli*, and 1.83% in *E. cloacae*; *blaOXA*-23 and *blaOXA*-58 were only detected in 24.6% and 7.86% of *A. baumannii*, respectively. In *A. baumannii*, *P. aeruginosa*, *K. pneumoniae*, and *E. coli*, *blaGES* was prevalent at 4.76%, 2.91%, 0.76%, and 0.44%, respectively. On the other hand, the prevalence of *blaIMP* was 16.67% for *K. pneumoniae*, 11.35% for *P. aeruginosa*, and 9.93% for *E. coli*. The prevalence of *blaOXA*-181 was 2.22% for *E. coli* and 0.36% for *K. pneumoniae*. However, *blaKPC* was only detected in 25.4% of *K. pneumoniae*.

Ghana

The highest pooled prevalence of *blaNDM* was 18.92% in *K. pneumoniae*, followed by 16.91% in *A. baumannii*, 8.69% in *P. aeruginosa*, 5.17% in *E. cloacae*, and 3.30% in

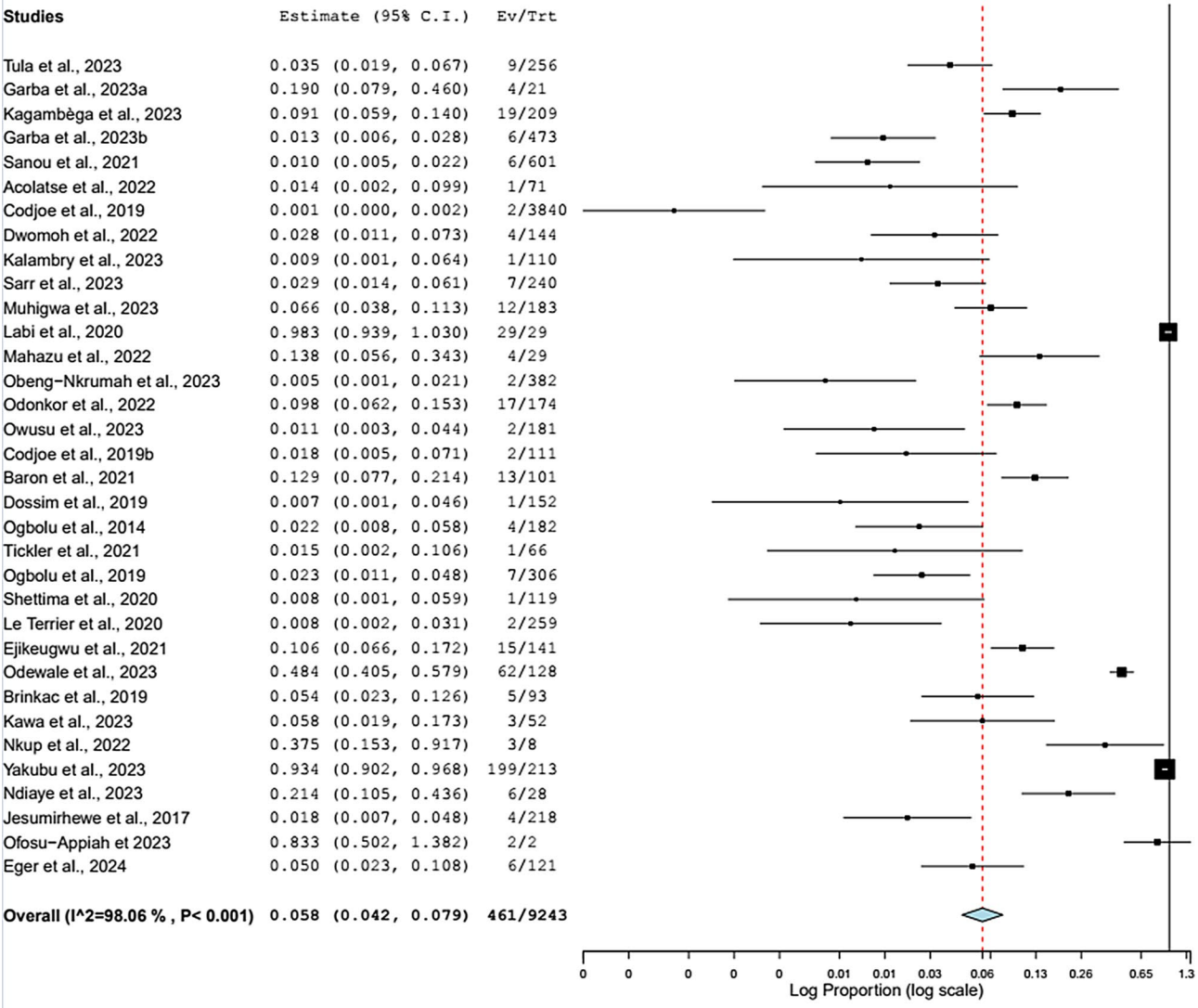


Fig. 4 Forest plot with adjusted average prevalence of carbapenem-resistance of *Klebsiella pneumoniae* in West Africa. **Legend**: Random Effects Mode (95% CI = 4.2–7.9, $I^2 = 98.06\%$, $p < 0.001$). X-axis is the proportion of the organism reported in individual studies as listed along the Y-axis, with the range of proportion in 95% confidence interval (CI)

E. coli. The highest pooled prevalence of *blaOXA*-48 was 4.78% in *P. mirabilis*, followed by 1.48% in *E. coli*, 1.41% in *K. pneumoniae*, and 0.98% in *A. baumannii.* In *A. baumanni*, *blaVIM* and *blaIMP* were 12.67% and 100% prevalent, respectively. Similarly, in *K. pneumoniae* and *E. coli*, *blaOXA*-181 was prevalent at 36.14% and 5.75%, respectively.

Burkina Faso

The pooled prevalence of the blaNDM gene was 9.42% in *E. coli*, 3.38% in *K. pneumoniae*, and 0.67% in *A. baumannii*. The *blaVIM* gene prevalence was 3.84% in *E. coli* and 0.22% in *K. pneumoniae*. In the case of *blaOXA*-48, the pooled prevalence was 6.86% in *E. coli* and 6.4% in *K. pneumoniae*. *BlaKPC* and *blaIMP* were prevalent at 0.48% and 1.19% in *E. coli*, and further at 2.87% and 0.48% in *K. pneumoniae*. The least prevalence of *blaOXA*-58 were in *K. pneumoniae* (1%) and *A. baumannii* (0.17%). However, in a study by Ouédraogo et al. [68], all the four *E. coli* isolated had the *blaOXA*-181 gene present.

Senegal

The pooled prevalence of *blaNDM* were 7.7%, 3.45%, and 3.4%, respectively, in *K. pneumoniae*, *A. baumannii*, and *E. coli*. For *blaOXA*-48, the highest pooled prevalence was in *K. pneumoniae* (11.34%) and the lowest was in *E. coli* (3.4%). *BlaKPC* and *blaOXA*-23 were prevalent at 5.94% and 68.66%, respectively, in *K. pneumoniae* and *A. baumannii*.

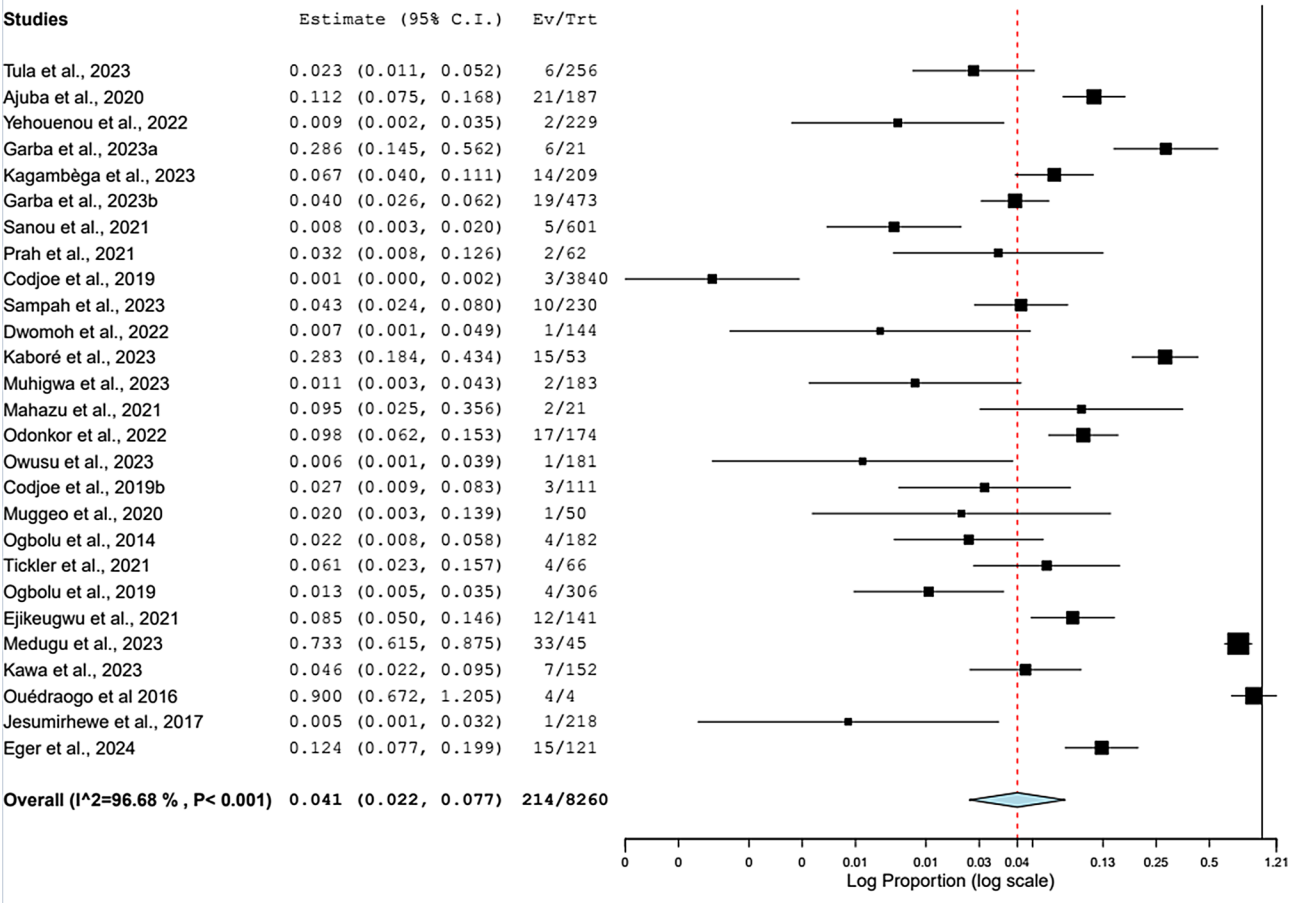


Fig. 5 Forest plot with adjusted average prevalence of carbapenem-resistance of *Escherichia coli* in West Africa. **Legend**: Random Effects Mode (95% CI = 2.2–7.7, $I^2 = 96.68\%$, $p < 0.001$). X-axis is the proportion of the organism reported in individual studies as listed along the Y-axis, with the range of proportion in 95% confidence interval (CI)

Discussion

The aim of this systematic and meta-analysis, which focused on 16 West African countries, was to assess the evolution and molecular epidemiology of carbapenem resistance in West Africa. The findings showed widespread carbapenem-resistant bacteria and CR genetic determinants in West African countries. It also revealed Nigeria and Ghana as the countries with publication contributions on carbapenem-resistant bacteria in the region. A study in 2023 by Somda et al. on AMR among foodborne pathogenic bacteria in West Africa between 2010 and 2020 also made a similar observation regarding research efforts. Nigeria and Ghana were the predominant countries publishing in the field. This was explained by the fact that those countries have more well-equipped universities and research centres than do the other WA countries, and their Governments prioritise dissemination of scientific knowledge [73]. Most laboratories and health care centres in West Africa are not equipped with the necessary equipment and/or are unfamiliar with the significance of screening for carbapenem resistance genes and traits. According to Uthman and Uthman, researchers in African countries tend to publish their research articles (65%) in local journals that are not listed or indexed in international databases [74].

Out of the 60 publications reviewed for this study, nearly 78.33% covered the period between 2020 and 2023 and the majority were found in Nigeria and Ghana in 2023. This indicates, in part, an increased attention to the issues of carbapenem resistance of bacteria in this region in recent years, particularly their molecular epidemiology. The high number of studies from Nigeria and Ghana, could be primarily due to the availability of state-of-the-art laboratory equipment provided by international donor agencies and collaborative research.

This systematic review revealed that carbapenem-resistant bacteria (CRB) are highly detected in clinical samples than in non-clinical ones. This is due to the observation that most of the published articles were on hospital-based studies (48/60), while just a few were environment (10/60) and animal-based (2/60). In recent years, the prevalence of CRB transmission around the world has risen, and further exacerbated by the increased

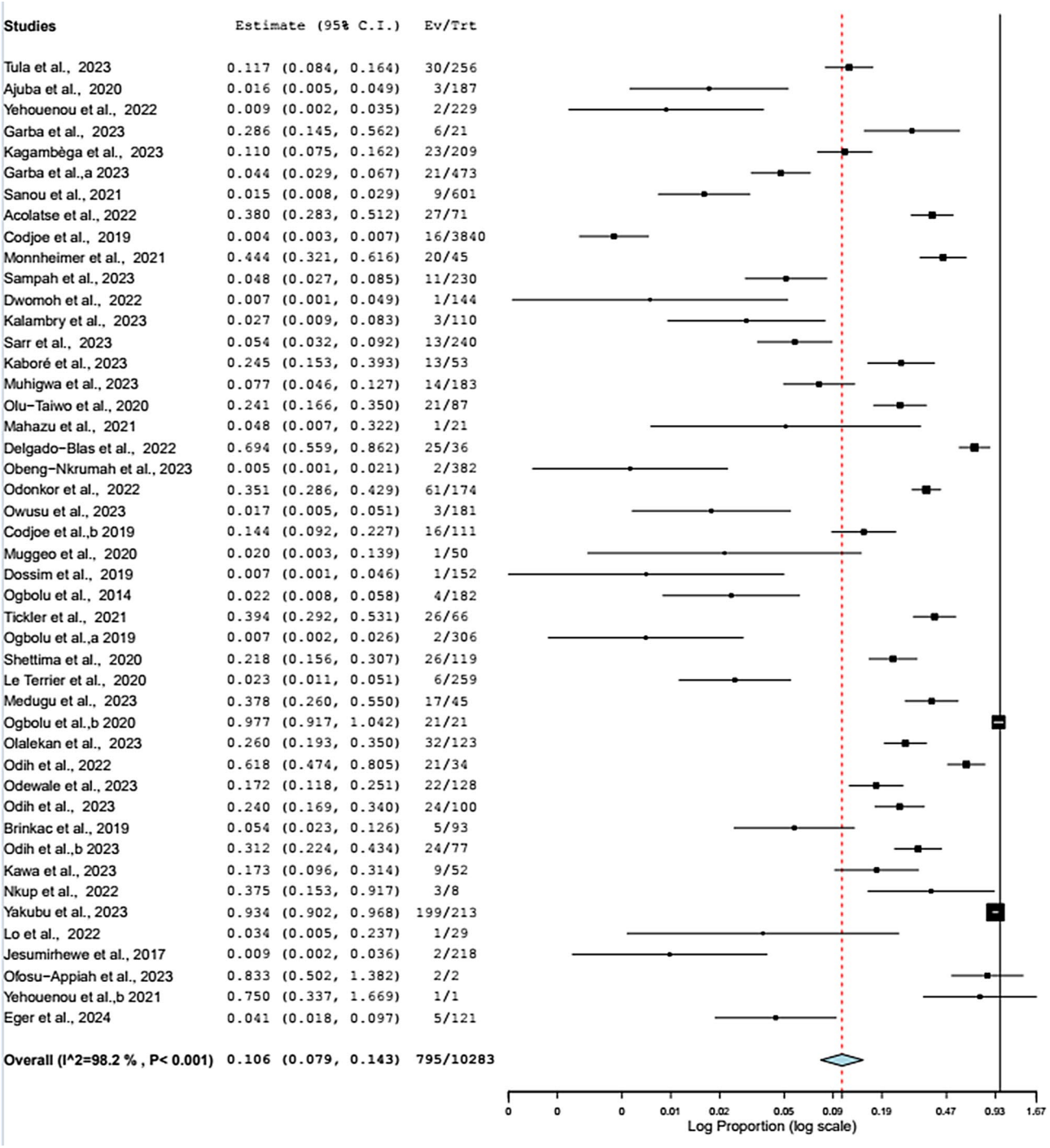


Fig. 6 Forest plot with adjusted average prevalence of *blaNDM* gene in West Africa. **Legend**: Random Effects Mode (95% CI = 7.9–14.3, $I^2 = 98.2\%$, $p < 0.001$). X-axis is the proportion of the *blaNDM* gene reported in individual studies as listed along the Y-axis, with the range of proportion in 95% confidence interval (CI)

antibiotic pressure associated with the COVID-19 pandemic [8]. Overall, the most prevalent CR bacteria across the West African region, based on our comprehensive analysis, were *A. baumannii* (18.6%; 95% CI = 14.0–24.6; $I^2 = 97.9\%$, $p < 0.001$), *P. aeruginosa* (6.5%; 95% CI = 3.1–13.4; $I^2 = 96.52\%$, $p < 0.001$), *K. pneumoniae* (5.8%; 95% CI = 4.2–7.9; $I^2 = 98.06\%$, $p < 0.001$), and *E. coli* (4.1%; 95% CI = 2.2–7.7; $I^2 = 96.68\%$, $p < 0.001$). In East Africa, carbapenem resistance was more exhibited in *A. baumannii* (23%), *P. aeruginosa* (17%), *K. pneumoniae* (15%), *P. mirabilis* (14%), and *E. coli* (12%). Our results are consistent with the observation from East Africa by

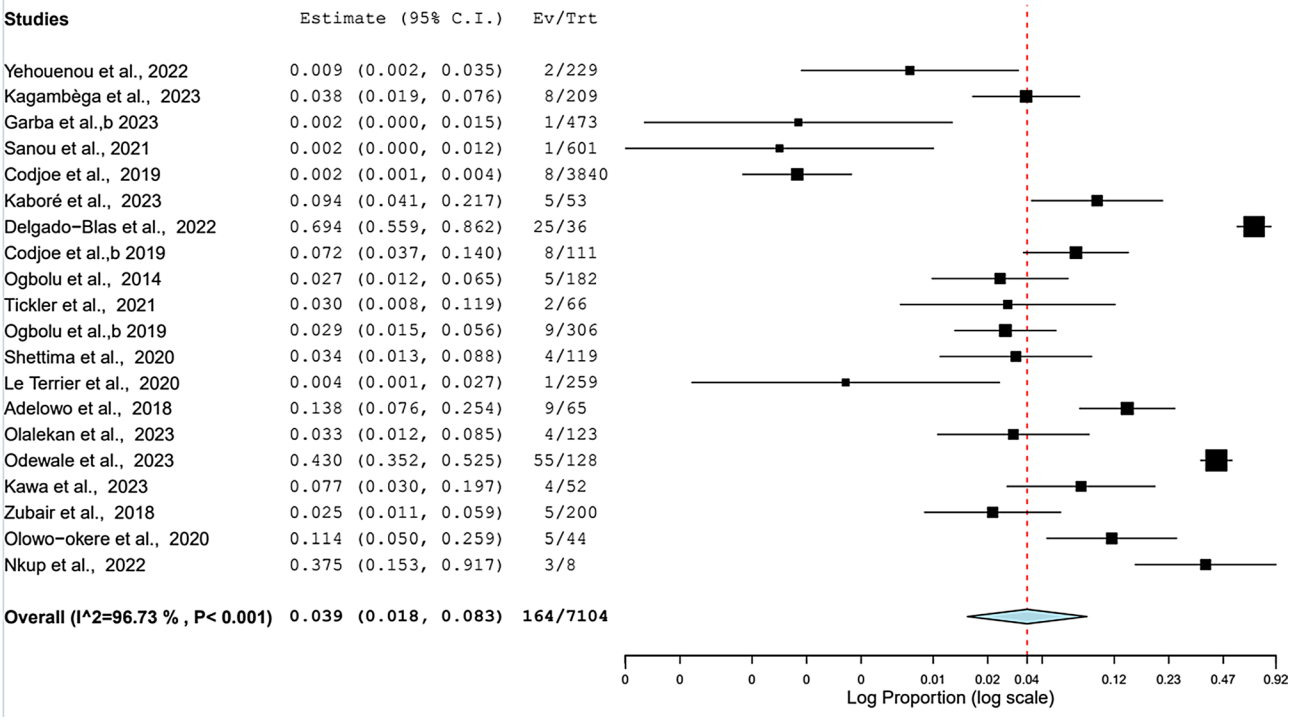


Fig. 7 Forest plot with adjusted average prevalence of the *blaVIM* gene in West Africa. **Legend**: Random Effects Mode (95% CI = 1.8–8.3, $I^2 = 96.73\%$, $p < 0.001$). X-axis is the proportion of the *blaVIM* gene reported in individual studies as listed along the Y-axis, with the range of proportion in 95% confidence interval (CI)

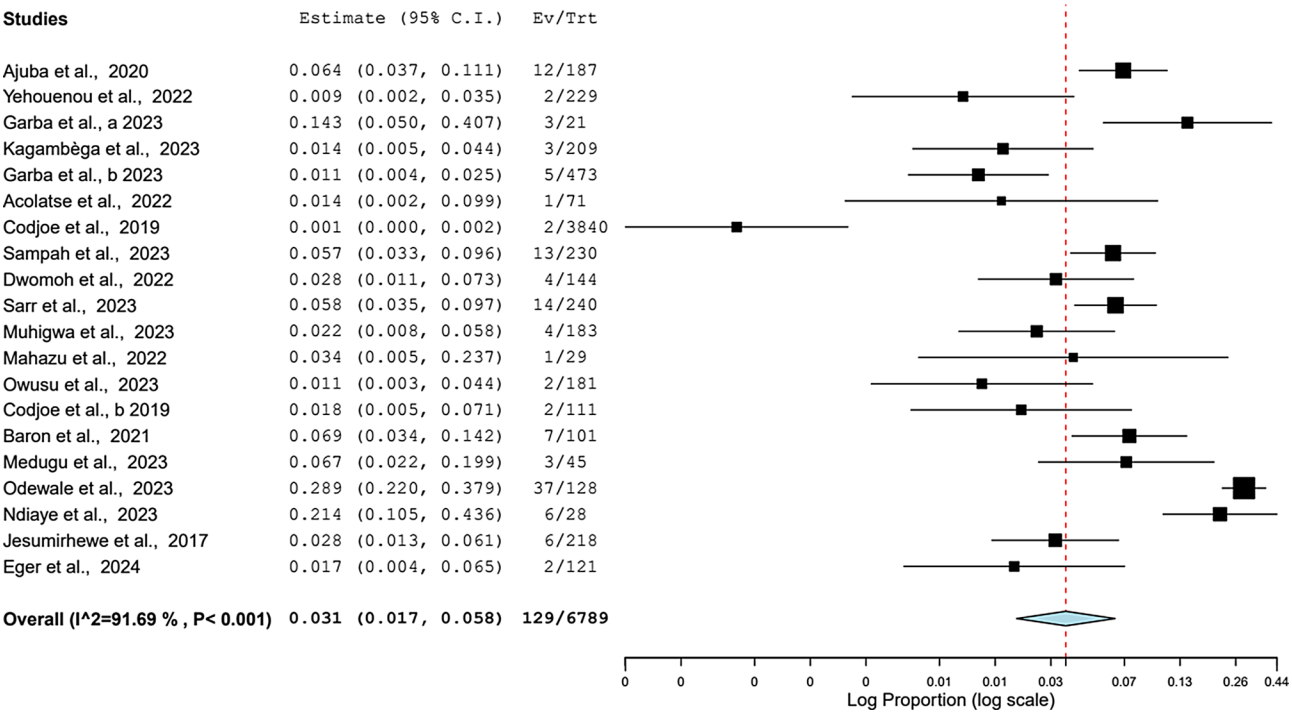
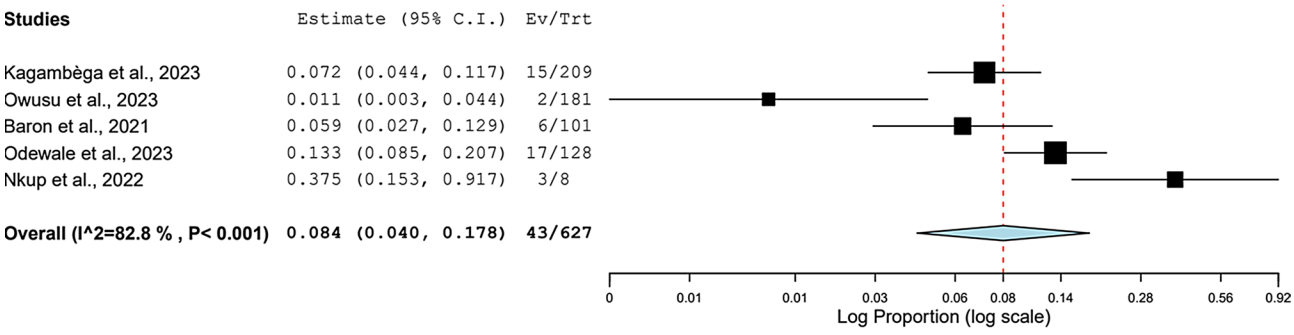
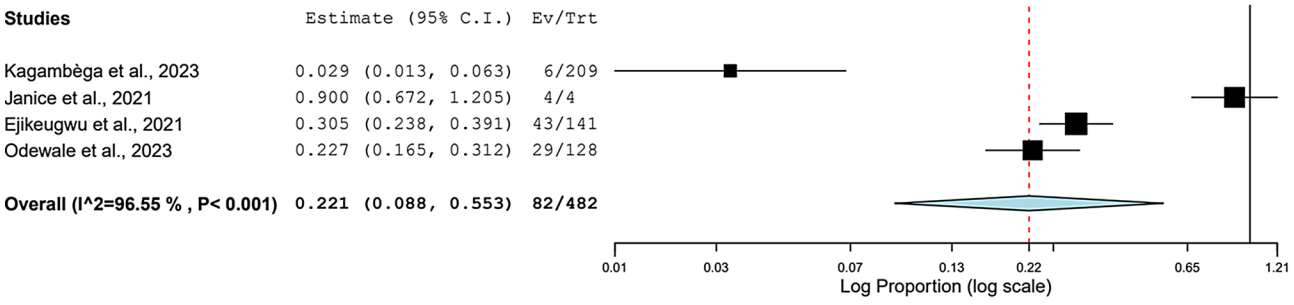


Fig. 8 Forest plot with adjusted average prevalence of *blaOXA*-48 gene in West Africa. **Legend**: Random Effects Mode (95% CI = 1.7–5.8, $I^2 = 91.69\%$, $p < 0.001$). X-axis is the proportion of the *blaOXA*-48 gene reported in individual studies as listed along the Y-axis, with the range of proportion in 95% confidence interval (CI)



A : Forest_plot blaKPC



B : Forest_plot blaIMP

Fig. 9 Forest plot with adjusted average prevalence of *blaKPC* and *blaIMP* genes in West Africa. **Legend**: (**A**): Random Effects Mode (95% CI = 4.0–17.8, $I^2 = 82.8\%$, $p < 0.001$). X-axis is the proportion of the blaKPC gene reported in individual studies as listed along the Y-axis, with the range of proportion in 95% confidence interval (CI). (**B**): Random Effects Mode (95% CI = 8.8–55.3, $I^2 = 96.55\%$, $p < 0.001$). X-axis is the proportion of the *blaIMP* gene reported in individual studies as listed along the Y-axis, with the range of proportion in 95% confidence interval (CI)

Table 2 Average prevalence of CR genetic determinant by country in West Africa

Countries	blaNDM % (*N*)	blaVIM % (*N*)	bla-OXA-48% (*N*)	bla-OXA-23% (*N*)	bla-OXA-181% (*N*)	bla-OXA-58% (*N*)	blaIMP % (*N*)	blaGES % (*N*)	blaKPC % (*N*)	bla-OXA-51% (*N*)
Nigeria	17.42 (20) ($I^2 = 97.87\%$, $p < 0.001$)	5.8 (12) ($I^2 = 94.26\%$, $p < 0.001$)	7.9 (4) ($I^2 = 94.01\%$, $p < 0.001$)	19.2 (5) ($I^2 = 89.26\%$, $p < 0.001$)	1.29 (2) ($I^2 = 20.74\%$, $p = 0.261$)	8.3 (3) ($I^2 = 0\%$, $p = 0.589$)	26.6 (2) ($I^2 = 51.5\%$, $p = 0.151$)	6.3 (4) ($I^2 = 98.33\%$, $p < 0.001$)	20.7 (2) ($I^2 = 75.93\%$, $p = 0.042$)	100 (1)
Ghana	9.0 (13) ($I^2 = 97.52\%$, $p < 0.001$)	4.8 (3) ($I^2 = 99.26\%$, $p < 0.001$)	1.4 (8) ($I^2 = 83.15\%$, $p < 0.001$)	17.5 (2) ($I^2 = 88.44\%$, $p = 0.003$)	14.3 (4) ($I^2 = 97.32\%$, $p < 0.001$)	0	100 (1)	0	0	5.56 (1)
Burkina Faso	8.6 (6) ($I^2 = 92.89\%$, $p < 0.001$)	1.4 (4) ($I^2 = 86.77\%$, $p < 0.001$)	2.6 (4) ($I^2 = 80.48\%$, $p = 0.002$)	0	100 (1)	0.17 (1)	3.83 (1)	0	7.18 (1)	0
Senegal	5.2 (2) ($I^2 = 0\%$, $p = 0.658$)	0	9.3 (3) ($I^2 = 77.65\%$, $p = 0.011$)	53.5 (3) ($I^2 = 93.31\%$, $p < 0.001$)	0	0	0	0	5.94 (1)	89.69 (1)
Benin	8.4 (2) ($I^2 = 96.66\%$, $p < 0.001$)	0.87 (1)	0.87 (1)	0	0	100 (1)	0	0	0	0
Mali	2.5 (2) ($I^2 = 0\%$, $p = 0.786$)	0	0	0	0	0	0	0	0	0
Togo	0.66 (1)	0	0	0	1.97 (1)	0	0	0	0	0

Legend: % = percent, (N) = Number of publications concerned, I^2 = Inconsistency (or heterogeneity), *p* = *p-value*

Ssekatawa et al. [11] and those from other parts of the world [5, 75]. Indeed, our reports conform with worldwide reports acknowledging that the magnitude of CRB is similar to that of carbapenem-resistant Enterobacteriaceae [76]. Contrary to this, Dossouvi et al., in their systematic review, identified that the most reported CRB in West Africa were *Escherichia* spp. (26.1%), *Klebsiella* spp. (20.8%), *Pseudomonas* spp. (20%), and *Acinetobacter* spp. (19.2%) [77]. Additionally, in Nigeria, Tula et al. reported *E. coli* and *Klebsiella* as the most prevalent CRB [13]. An explanation for the observation could be the concurrent use of phenotypic and molecular carbapenem resistance detection techniques in their study. The distribution of CRB, such as *E. coli*, *K. pneumoniae*, *P. aeruginosa*, and *A. baumannii*, is general all over the world according to several studies [78–81]. Comparing results with those of other independent studies may be challenging due to differences in study design and population [82]. In addition, protective measures taken to reduce the risk of new virus transmission may simultaneously facilitate the spread of other drug-resistant bacteria [8].

In the healthcare setting, carbapenems are considered the last resort for the treatment of patients, but an increasing trend of bacteria resistance is posing a big challenge. In this review, the prevalence of carbapenem resistance genetic determinants in WA was estimated as *blaNDM* (10.6%) *blaVIM* (3.9%), and *balOXA*-48 (3.1%). These prevalence are consistent with those reported in other studies in West Africa, Nigeria, and the USA [77, 83, 84]. They are, however, lower compared to those reported in other studies in East Africa (35.0%), India (30% and 43%), and South Africa (68%) [11, 85, 86]. Even though the prevalence differed from country to country, the most prevalent carbapenem resistance genes across WA were *blaNDM*, *blaVIM*, and *blaOXA*-48, similar to observations in other studies in West Africa [11, 13] and worldwide reports [5, 87–90]. Several studies show that the enzymes responsible for the hydrolysis of carbapenems frequently found and spread in the last decade are *blaKPC*, *blaNDM*, *blaVIM*, and *blaOXA*-48, which is in agreement with our results [91–93]. These studies also showed that the Enterobacterales harbouring these genes are mostly *Klebsiella* spp., *E. coli*, *Pseudomonas* spp., and *Acinetobacter* spp., which is consistent with our results. These bacteria, being responsible for most infections, are thus more prevalent in hospital settings and the environment. Carbapenems are widely used to combat these bacteria, which could explain the high resistance of these bacteria against these antibiotics.

In this review, 10 studies conducted with environment-based samples were from Nigeria, Ghana, Burkina Faso, and Senegal, and two studies involved animal samples. In all these studies, *A. baumannii*, *E. coli*, *K. pneumoniae*, and *P. aeruginosa* were mostly found. According to several studies, in Africa, the actual occurrence of environmental contamination by carbapenem-resistant bacteria is not well researched, although hospital environments tainted with CRB by infected patients are implicated as the main routes of transmission [11]. Nevertheless, the few studies that have been reported in other parts of the world have shown that *blaNDM*, *blaKPC*, *blaVIM*, and *blaOXA*-48 genes were mostly spreading [94, 95]. These obervations agree with our results indicating a prevalence of 28.45% for *blaNDM*, 21.88% for *blaVIM*, 4.89% for *blaOXA*-48, 13.22% for *blaOXA*-181, and 7.18% for *blaKPC*. The environment is considered to be the fastest route for the transmission and dissemination of AMR genes. Antibiotics are employed in intensive livestock farms, for disease treatment of animals and animal growth promotion, which selects for resistant bacteria and results in presence of antibiotic residues in farming effluents. Therefore, the environment impacted by livestock farming has been regarded as a reservoir for resistant bacteria [94]. Likewise, some studies have reported on the presence of CRB in foods and aquatic products [96]. However, from the articles reviewed, no publication has reported on CRB in food or aquatic origin in West Africa.

In this systematic review, PCR and whole genome sequencing (WGS) constituted most methods used to identify carbapenem resistance genes.

Our study had some limitations. First, the databases accessed for the publications are more international-oriented, which limits the scope of access since most African publications are in local databases. Another limitation is the non-existence of appropriate studies across some of the different countries in WA. In addition to this, there was a disproportion in the number of publications among the included countries, which may have introduced some bias. Also, the use of modern methods for the detection of CRBs was limited in some countries because they are scarce and expensive.

Conclusion

This review highlighted that in West Africa, Nigeria and Ghana are the countries which have the most publications related to carbapenem-resistant bacteria. Further, the most prevalent CRBs were *K. pneumoniae*, *E. coli*, *A. baumannii*, and *P. aeruginosa*. Also, among the several types of carbapenem resistance genes detected, *blaNDM*, *blaVIM*, and *blaOXA*-48 were the most prevalent. It can be deduced that by employing the use of a robust molecular platform such as WGS, MALDI-TOF, multilocus sequence typing (MLST), and phylogenetic analyses, all genetic determinants of carbapenem resistance in humans, the environment, and livestock could be identified and documented. These methods could further deepen our understanding of CRB strains circulating in

West Africa. The findings of this study highlight the need to implement suitable and appropriate control strategies to reduce complications and prevent the dissemination of resistant bacteria in West Africa.

Abbreviations

CRE	Carbapenem-resistant Enterobacteriaceae
CRB	Carbapenem Resistance Bacteria
CR	Carbapenem resistance
AJOL	African Journals Online
WA	West Africa

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Conception and design: SNS and ESD; development of data screening form: SNS; data screening: SNS and RN; data analysis and interpretation: SNS, RN, PBT-Q and FCNK; draft preparation and revisions: SNS, RN, FCNK, PBT-Q and ESD; funding acquisition: ESD. All the authors approved of the final version of the manuscript.

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Data availability
All data generated or analysed are included in this review.

Declarations

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