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1 **Epidemiology and prevalence of extended-spectrum β -lactamase- and carbapenemase-**
2 **producing *Enterobacteriaceae* in humans, animals and environments in West and**
3 **Central Africa**

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51 **Abstract**

52 Extended-spectrum β -lactamase-producing *Enterobacteriaceae* (ESBL-PE) and
53 carbapenemase-producing *Enterobacteriaceae* (CPE) are widespread. Here, we used the “One
54 Health” approach to determine the knowledge gaps on ESBL-PE and CPE in West and
55 Central Africa. We searched all articles on ESBL-PE and CPE in these African regions
56 published in PubMed, African Journals Online, and Google Scholar from 2000 onwards.
57 Among the 1201 articles retrieved, we selected 168 studies (West Africa n=121, and Central
58 Africa n=47) with data from 22 of the 26 West and Central Africa countries. Concerning the
59 settings, 137 articles focused only on humans (carriage and/or infection), 6 articles on humans
60 and animals, 12 on animals, 8 on humans and environments, one on environments, and one on
61 humans, animals and environments. ESBL-PE prevalence ranged from 11% to 72% in
62 humans, and from 7% to 79% in aquatic environments (wastewater). In animals, ESBL-PE
63 prevalence hugely varied: 0% in cattle, 11-36% in chickens, 20% in rats, 21-71% in pigs, and
64 32-75% in dogs. The *bla*_{CTX-M-15} gene was the predominant ESBL-encoding gene and was
65 associated with plasmids of the incompatibility groups F, H, K, Y, N, I1 and R. CPE were
66 studied only in humans. Class B metallo- β -lactamases (NDM) and class D oxacillinases
67 (OXA-48 and OXA-181) were the most common carbapenemases. Our results show major
68 knowledge gaps, particularly on ESBL and CPE in animals and environments, that might
69 limit antimicrobial resistance management in these regions. They also emphasize the urgent
70 need to improve active surveillance programs in each country and to support antimicrobial
71 stewardship.

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77 **1. Introduction**

78 Beta-lactams are the most widely used antibiotics for the treatment of bacterial infections.
79 Their misuse and abuse are the main cause of resistance in *Enterobacteriaceae* worldwide [1,
80 2], in community and healthcare settings, and are associated with high mortality and
81 morbidity [3,4].

82 The most prevalent mechanism by which bacteria acquire resistance to β -lactam antibiotics is
83 the production of enzymes (β -lactamases) that hydrolyze these antibiotics [5,6]. Currently,
84 extended spectrum β -lactamases (ESBL), such as the SHV, TEM and CTX-M variants, and
85 carbapenemases (MBL, KPC, and class D oxacillinases) are the most common β -lactamases
86 detected in *Enterobacteriaceae*, particularly in *Escherichia coli* and in *Klebsiella*
87 *pneumoniae*. ESBLs are enzymes that can hydrolyze penicillins, cephalosporins and
88 aztreonam, but neither cephamycins nor carbapenems, and are inhibited by β -lactamase
89 inhibitors, such as clavulanic acid [7,8]. Therefore, carbapenems have been used as the
90 treatment of choice for infections due to ESBL-producing *Enterobacteriaceae* (ESBL-PE),
91 resulting in the emergence of carbapenemase-producing *Enterobacteriaceae* (CPE) [4,9].
92 During the last decade, ESBL-PE and CPE have increasingly been reported worldwide in
93 different ecosystems [7,10–12].

94 Resistance genes can spread by vertical transfer (e.g., transmission of the ESBL-producing *E.*
95 *coli* sequence type 131) or by horizontal transfer of mobile genetic elements, such as plasmids
96 of different incompatibility (Inc) groups (e.g., IncF, IncX3), transposons or integrons, within
97 and between humans, animals and environments [13, 14]. Consequently, only a “one health
98 approach” can help to understand the emergence and transmission of these genes. However,
99 data collection and surveillance of multidrug-resistant Enterobacteria are often limited,
100 particularly in middle and low income countries, such as many African countries.

101 The aim of this review is to provide an overview of the epidemiology of ESBL-PE and CPE
102 circulating in humans, animals and environments in West and Central Africa. For that, we
103 systematically reviewed data published from 2000, on bacterial species, genotypes and
104 genetic mechanisms of resistance of ESBL-PE and CPE isolated in humans, animals and
105 environments.

106 **2. Methods**

107 2.1 Literature search

108 A systematic search was carried out in PubMed, African Journals Online, and Google Scholar
109 using the search terms “ESBL” and/or “carbapenemases”, “*Enterobacteriaceae*”, “West
110 Africa” or “Central Africa”, “antimicrobial” or “antibiotic”, “resistance”, and “susceptibility”
111 or “sensitivity”, combined with the names of the different West and Central African countries.
112 According to the United Nation geographical region definition of the Central and West Africa
113 region [15], this region includes the following countries: Angola, Cameroon, Central African
114 Republic (CAR), Chad, Congo-Brazzaville, Democratic Republic of the Congo (DRC),
115 Equatorial Guinea, Gabon, and São Tomé et Príncipe in Central Africa, and Benin, Burkina
116 Faso, Cape Verde, Côte d’Ivoire, Gambia, Ghana, Guinea, Guinea-Bissau, Liberia, Mali,
117 Mauritania, Niger, Nigeria, Saint Helena, Senegal, Sierra Leone and Togo in West Africa.

118 All studies (in English or French) on ESBL-PE and/or CPE in humans, animals, food, or
119 environment published between January 2000 and January 2019 were included.

120 2.2 Data extraction

121 After excluding irrelevant and duplicate studies, the remaining studies were fully read to
122 extract data for this review: name of the first author, year of publication, country where the
123 study was carried out, study design, and settings (hospital or community, animals, food,
124 environment), *Enterobacteriaceae* species, ESBL-PE and CPE prevalence, method of analysis

125 (phenotypic, molecular, or genomic), resistance genes, genetic support of resistance
126 determinants, and genetic diversity.

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129 **3. Results**

130 3.1 Studies selected for the analysis

131 A total of 1366 articles were identified. After review of titles and abstracts, 1201 articles were
132 excluded (irrelevant and duplicate studies), and 165 articles were fully read. These 165
133 articles (West Africa n=118 and Central Africa n=47) focused on 23 of the 26 countries that
134 constitute the West and Central African region (Figure 1): Nigeria (n=57), Senegal (n=16),
135 Cameroon (n=14), Ghana (n=12), Burkina Faso, Chad, Gabon and Mali (n=7/country), DRC
136 and CAR (n=6/country), Benin (n=5), Angola and Togo (n=4/country), Côte d'Ivoire (n=3),
137 Mauritania, Niger and Sierra Leone (n=2/country), Congo-Brazzaville, Equatorial Guinea,
138 Gambia, Guinea, Guinea-Bissau and São Tomé and Príncipe (n=1/country), and Benin and
139 Togo together (n=1). No article was found on ESBL-PE and/or CPE in Cape Verde, Liberia
140 and Saint Helena (Data and all references in table S1, S2 and S3 in supplemental material).

141 Concerning the settings, 136 articles focused only on humans (carriage and/or infection), 6
142 articles on humans and animals, 13 on animals/foods, one on humans and environment, 8 on
143 the environment, and one on humans, animals and environment. The characteristics of the
144 included studies are presented in Table 1, and Tables S1, S2 and S3 in supplemental material.

145 3.2 Prevalence and genetic determinants of ESBL-PE in humans, animals, animal food and 146 environments

147 Among the 152 studies on ESBL-PE in humans, 54 identified ESBL-PE isolates using
148 phenotypic tests based on synergy between indicators and inhibitors, but without resistance

149 gene identification, 92 used molecular tests for the identification of resistance genes, and 6
150 were based on whole genome sequencing.

151 ESBL-PE prevalence varied among countries, and within countries it increased over time. For
152 example, in Nigeria, ESBL-PE prevalence increased from 11% in 2009 to 72% in 2011.
153 Overall, the highest ESBL-PE rate (79%) was detected in *Enterobacteriaceae* isolates from
154 children in Senegal in 2017, and the lowest rate (4%), in clinical *Enterobacteriaceae* isolates
155 from CAR in 2006 (Table 1).

156 The *bla*_{CTX-M-15} gene (60 to 100%) was the most frequent ESBL-encoding gene detected in
157 ESBL-PE isolates from 17 countries (n=67 studies), followed by *bla*_{SHV-12} (2 to 30%) in 10
158 countries (n=13 studies), *bla*_{CTX-M-1} (3 to 6%) in 4 countries (n=13 studies), *bla*_{CTX-M-3} (3 to
159 4%) in 3 countries (2 studies), and *bla*_{CTX-M-14} in 4 (2 studies) (Table 1). These ESBL-
160 encoding genes were detected in different *Enterobacteriaceae* species, especially in *E. coli*, *K.*
161 *pneumoniae*, *Salmonella*, *Enterobacter*, *Morganella*, *Proteus* and *Providencia*.

162 The few studies on the resistance genetic support identified several genetic elements (e.g.
163 transposons and/or plasmids) involved in the mobility of resistance genes. In humans, the
164 frequency of plasmids associated with ESBL was as follows: IncF (30 to 100%), IncNT (0 to
165 100%), IncY (0 to 100%), IncH and IncK (Table 1). The *bla*_{CTX-M-15} gene was commonly
166 detected in insertion sequences (ISEcp1), and was associated mainly with IncF (90%)
167 plasmids, but also with IncN, IncY, IncH, and IncK plasmids (Table 1).

168 Among the 165 reviewed articles, only 29 concerned ESBL-PE in animals and/or
169 environments in Angola, Benin, Burkina Faso, Cameroon, Côte d'Ivoire, CAR, DRC, Gabon,
170 Ghana, Guinea, Mali, Nigeria, and Senegal. In animals, ESBL-PE prevalence was
171 investigated in pigs [16–20], dogs [21–23], rats [24,25], chickens or chicken food [19,21,26–
172 31], gorillas [32], cattle [19,20,33], goats and sheep [21]. Only one article described ESBL-
173 PE isolated from drinking water [34] (Table S2 in supplemental material).

174 ESBL-PE prevalence in animals varied widely: 0% in cattle, 11-36% in chickens, 20% in rats,
175 21-71% in pigs, and 32-75% in *E. coli* isolated from dogs (Table S2 in supplemental
176 material).

177 The *bla*_{CTX-M-15} gene (67 to 100%) was the predominant ESBL-encoding gene and was mainly
178 associated with IncF, IncY, IncN, IncI1, and IncR plasmids. The genes encoding CTX-M-1,
179 CTX-M-2, CTX-M-14, CTX-M-27, CTX-M-55, CTX-M-61 and SHV-12 were also detected
180 (Table 1).

181 In the environment, ESBL-PE were isolated from wastewater [35–41], hospital surfaces [42],
182 and a poultry farm [43]. ESBL-PE prevalence in wastewater varied from 7 to 79%. Like in
183 humans and animals, CTX-M-15 was the predominant ESBL type (Table S3 in supplemental
184 material).

185 3.3 CPE in humans, animals and environments

186 CPE were reported by 25 studies in 12 countries, mainly in *E. coli* and *K. pneumoniae*
187 isolates. Ambler class A carbapenemases (KPC and GES), class B metallo- β -lactamases
188 (NDM, VIM and DIM), and class D oxacillinases (OXA-48, OXA-51-Like, OXA-58 and
189 OXA-181) were detected in CPE from different West and Central Africa countries (Figure 2).
190 Specifically, production of NDM-1, -4, -5 and -7 was reported by studies in Angola,
191 Cameroon, Chad, Gabon, Ghana, Nigeria, Senegal, and Sierra Leone. VIM-1 and -2 were
192 detected in Nigeria and Sierra Leone, and DIM-1 only in Nigeria. KPC and GES were
193 identified only in one study in Nigeria. OXA-48 was detected in Nigeria and Senegal, OXA-
194 181 in Angola, Chad, Burkina-Faso, Mali, Nigeria, São Tomé and Príncipe, and Togo, and
195 OXA-51- like and OXA-58 in Sierra Leone (Table 2).

196 The *bla*_{NDM-1} gene was associated with many broad-host-range plasmid types, including
197 IncA/C, IncH1 and IncF. The *bla*_{OXA-181} and *bla*_{NDM-5} genes were detected on IncX3 plasmids,
198 and *bla*_{OXA-48} and *bla*_{NDM-4} on IncL/M and IncF plasmids, respectively. The plasmid

199 frequencies and the frequencies of plasmid-enzyme associations are summarized in Table 2.
200 No article was identified on CPE in food, animals, and/or environments.

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204 **4. Discussion**

205 In this systematic review, the “one health” approach was used to analyze literature data on
206 ESBL-PE and CPE prevalence in different ecosystems (humans, animals, food and
207 environment) in Central and West Africa regions. Our results revealed major knowledge gaps
208 that may hinder the management of antimicrobial resistance in these regions.

209 First, most articles focused mainly on humans (n=136), and only a limited number of
210 published studies investigated ESBL-PE and CPE isolated from animals and environments
211 (n=29). Only one study carried out in Angola [44] analyzed the distribution and molecular
212 epidemiology of ESBL-PE and CPE in different non-clinical ecosystems (healthy individuals,
213 healthy animals, and aquatic environments). Six other studies focused on ESBL-PE isolated
214 from humans and animals: three in Cameroon (ESBL-PE in pigs and humans) [16–18], one in
215 CAR (ESBL-PE isolates in wild mammals and humans) [32], one in Cote d’Ivoire (ESBL-
216 producing *E. coli* in humans, dogs, cats, and wildlife) [22], and one in Nigeria (ESBL-
217 producing *E. coli* isolates in humans and cattle) [33].

218 Second, no molecular characterization was performed in some countries (Congo, Equatorial
219 Guinea and Gambia), leading to a serious lack of information on the different resistance genes
220 involved in ESBL-PE and CPE.

221 Third, no data was available on CPE in animals and environments in Central and West Africa
222 regions, although they were detected in animals and environment in other African countries
223 [12,45,46].

224 Nevertheless, most of the ESBL-PE prevalence rates in humans in Central and West Africa
225 regions were much higher than in North America [47] and Europe [48], in agreement with
226 studies from other African regions [7] and Asian countries [49]. ESBL-PE prevalence in
227 animals (*E. coli* isolated from dogs, pigs and cattle) and environment (hospital wastewater)
228 also was high, when investigated.

229 Several factors could explain this alarming situation in West and Central Africa. Indeed,
230 major gaps in the knowledge on antimicrobial resistance in Africa undermine the
231 development and implementation of evidence-based public health policies and medical
232 practice. Moreover, the lack of standardized antimicrobial resistance detection methods and
233 interpretation guidelines do not allow comparing the results of different studies and optimal
234 resistance tracking. In addition, access to reference laboratories and to external quality
235 assessment schemes must be improved by strongly promoting and scaling up the Global Anti-
236 Microbial Surveillance System (GLASS) launched by the World Health Organization in 2015
237 and based on each country's specific priority pathogens [4,10,11].

238 Some studies (REF) showed that the degree of household antimicrobial usage, as reported by
239 farmers, was higher in humans than in animals, probably because people have more access to
240 human drugs than veterinary drugs. This might increase the risk of ESBL-PE circulation in
241 humans and also between humans and animals. Then, free-roaming animals contribute to the
242 dispersal of multidrug-resistant ESBL-producing bacteria in the community through fecal
243 shedding and environmental contamination. Close contacts between humans and animals,
244 poor household waste disposal methods, poor environmental sanitation and other related
245 factors could facilitate the exchange of antimicrobial resistant bacteria between humans and
246 animals within households. Therefore, a national antimicrobial monitoring program should be
247 put in place to track the marketing and consumption of antimicrobial agents, including the
248 quantity and reasons of antimicrobial drug usage in humans and animals [7,17,21].

249 The *bla*_{CTX-M-15} gene was the most prevalent ESBL-encoding gene in humans, animals and
250 environment, and was carried by different plasmids, mainly of the IncF group, but also of the
251 IncN, IncY, IncH, and IncK groups. This suggests that *bla*_{CTX-M-15}-carrying plasmids are
252 widely exchanged among humans, animals and environments, and may be responsible for the
253 increasing ESBL-PE rate in this region. This hypothesis should be confirmed by detailed
254 genetic studies.

255 Despite the data paucity, CPE prevalence among human isolates varied between 4% and 27%
256 in Central and West Africa. CPE were mainly detected in *E. coli* and *K. pneumoniae* isolates,
257 and less frequently in other *Enterobacteriaceae*, such as *Enterobacter cloacae*, *Klebsiella*
258 *aerogenes*, *Citrobacter freundii* and *Morganella morganii*. Class D carbapenemases
259 (especially OXA-181) were the most reported enzymes in Central and West Africa. All the
260 available publications found that *bla*_{OXA-181} was associated with the IncX3 plasmid. OXA-
261 181, a variant of OXA-48, was first identified in different *Enterobacteriaceae* isolates from
262 Indian patients [50], and has spread in different countries, including Africa [4,51].

263 Our results also indicate that many variants of the metallo- β -lactamase NDM are produced by
264 different *Enterobacteriaceae* species. NDM-1 was identified in *E. coli* and *K. pneumoniae*
265 isolates from Angola, Nigeria and Ghana [45,46,52–57], NDM-4 in *E. coli* isolates from
266 Cameroon [58], NDM-5 in *E. coli*, *K. pneumoniae* and *E. cloacae* from Angola, Chad and
267 Togo [46,59,60], and NDM-7 in *K. pneumoniae* and *E. cloacae* from Gabon [61]. Hence,
268 NDM-producing *Enterobacteriaceae* are considered to be endemic CPE in North African
269 countries [62]. In addition, other metallo- β -lactamase-encoding genes, such as *bla*_{VIM} and
270 *bla*_{DIM}, were detected only in *E. coli* and *K. pneumoniae* isolates from Nigeria and Sierra
271 Leone [52,53,63]. Unlike North America and Europe where *bla*_{KPC} is the most common β -
272 lactamase gene in CPE isolates, KPC-producing *E. coli* was detected only in Nigeria [52].

273

274 **5. Conclusions**

275 This review on ESBL-PE and CPE in humans, animals and environments in Central and West
276 Africa confirms the dissemination of β -lactamase-producing *Enterobacteriaceae* in this
277 region of the world.

278 Our findings highlight the paucity of data on ESBL-PE and CPE in animals and the
279 environment. They also emphasize the urgent need to improve active surveillance programs in
280 each country and to support antimicrobial stewardship.

281

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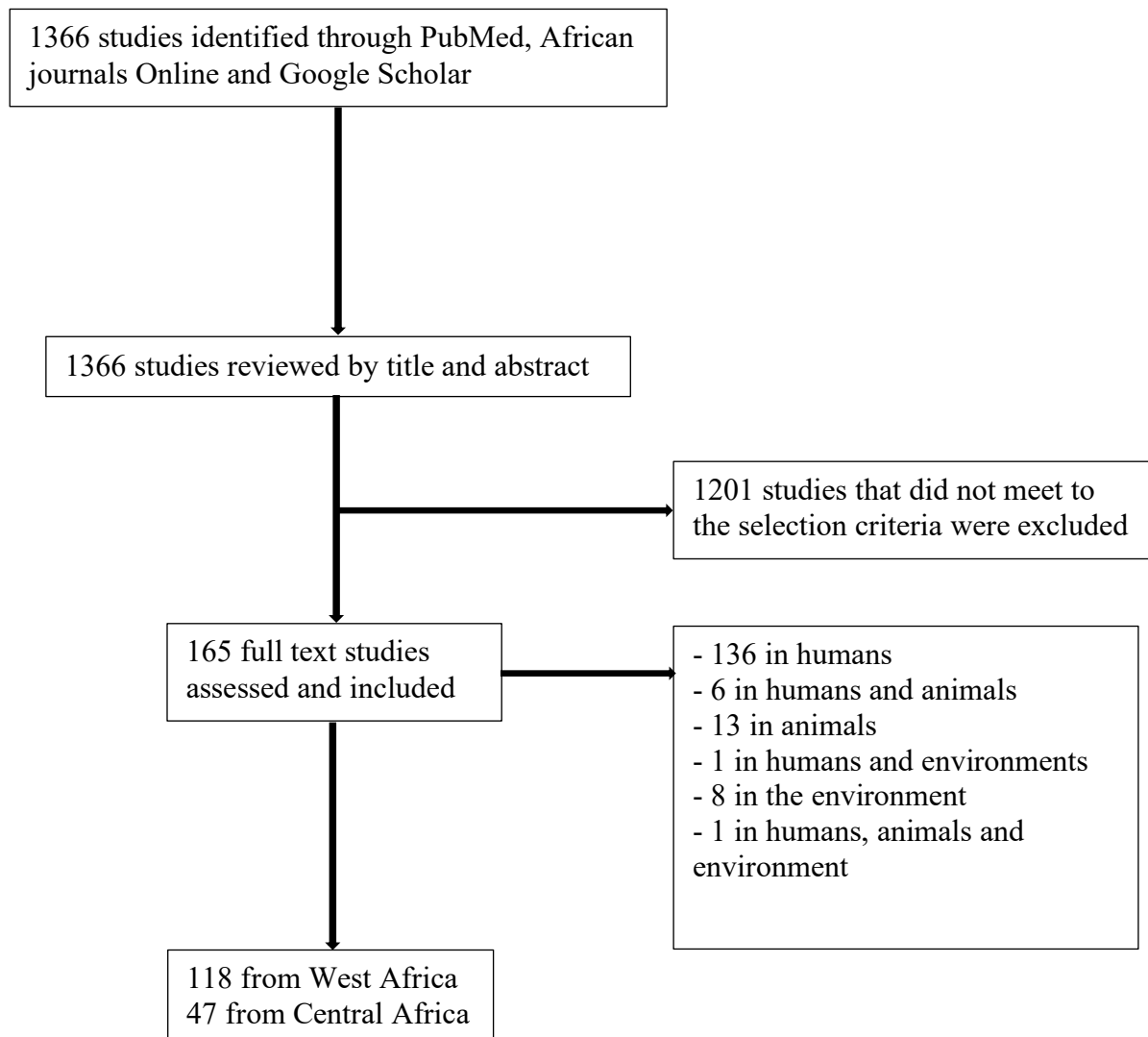


Figure 1: Flow diagram summarizing article identification and selection

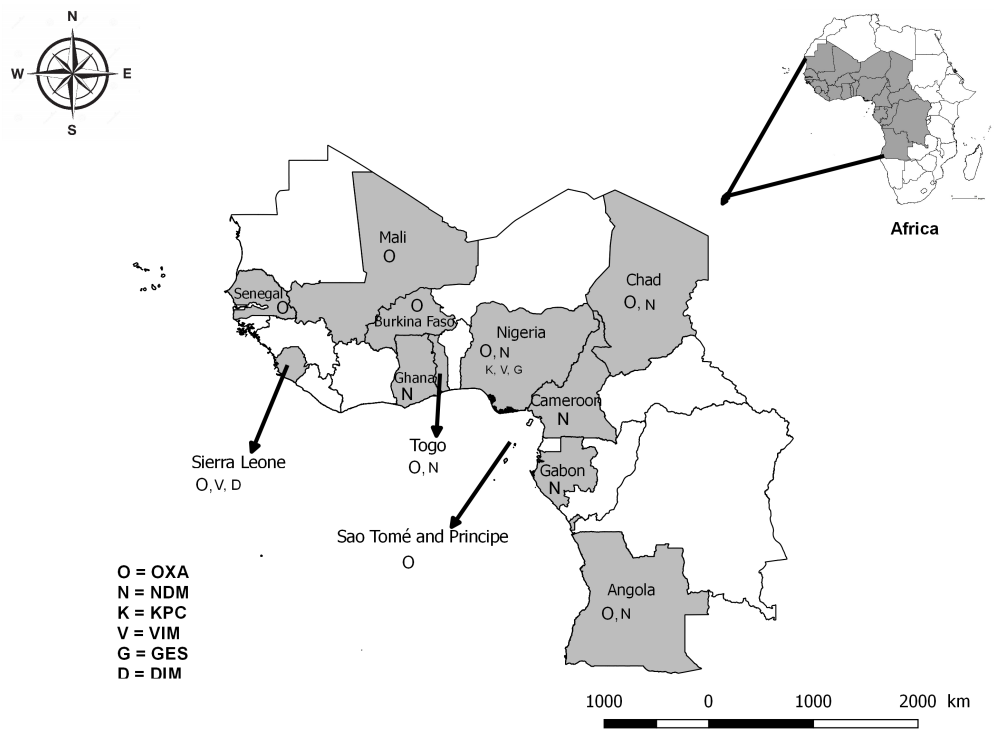


Figure 2: Geographic distribution of carbapenemases produced by *Enterobacteriaceae* isolates in the West and Central Africa region.

Table 1: Prevalence and genetic determinants of extended-spectrum β -lactamase-producing *Enterobacteriaceae* isolated in humans, animals and environments in West and Central Africa.

ESBL type (%)	Origin	Incompatibility group	ESBL-PE prevalence	Country	References
CTX-M-15 (98%) SHV-12 (2%)	Humans	IncF [30% (100% associated with CTX-M-15)] IncHI2 [15% (100% associated with CTX-M-15)] IncY [20% (100% associated with CTX-M-15)] NT [35% (100% associated with CTX-M-15)] NA	22%	Angola	[44]
CTX-M-15 (100%)	Animals	IncF [45.5% (100% associated with CTX-M-15)] IncY [36.4% (100% associated with CTX-M-15)] NT [18.2% (100% associated with CTX-M-15)]	75%		[23,44]
CTX-M-15 (100%)	Humans	NA	35-65%	Benin	[42,47,49]
CTX-M	Environments	NA	15%		[36,42]
CTX-M-15 (94%) SHV-12 (6%)	Humans	NA	32-58%	Burkina Faso	[64,65]
SHV-100	Environments	NA	NA		[37]
CTX-M-15 (97%) SHV-12 (3%)	Humans	IncF [100% (100% associated with CTX-M-15)] NA	12-55%	Cameroon	[66–70]
CTX-M-15 (100%)	Animals	IncF [100% (100% associated with CTX-M-15)]	21%		[16,18]
CTX-M-15 (65%) CTX-M-3 (5%) SHV-12 (20%) SHV-2a (10%)	Humans	IncF [89% (100% associated with CTX-M-15)] IncHI2 [4% (100% associated with CTX-M-15)] NT [7% (100% associated with CTX-M-15)] NA NA NA	4-63%	CAR	[71–74]
CTX-M-15 (100%)	Animals	NA			[32]
CTX-M-15 (96%) CTX-M-9 (4%)	Humans	IncF [100% (100% associated with CTX-M-15)]	48%	Chad	[59,75,76]
CTX-M-15 (98%)	Humans	IncF [24% (100% associated with CTX-M-15)]	27-56%	Côte d'Ivoire	[22,77]

SHV-12 (2%)		IncN [6% (100% associated with CTX-M-15)] IncR [6% (100% associated with CTX-M-15)] NT [64% (100% associated with CTX-M-15)] NA			
CTX-M-15 (100%)	Animals	IncF, IncR	32%		[22]
CTX-M-15 (100%)	Humans	IncY [100% (100% associated with CTX-M-15)]	50%	DRC	
CTX-M-1(100%)	Environments	NA	7%		
CTX-M-15 (90%) CTX-M-1 (3%) CTX-M-8 (7%)	Humans	NA	45-51%	Gabon	[78,79]
CTX-M-1 (35%) CTX-M-14 (55%) CTX-M-32 (10%)	Animals	NA	23%		[80]
CTX-M-15 (98%) CTX-M-27 (2%)	Humans	IncF [100% (98% associated with CTX-M-15 and 2% with CTX-M-27)]	49-62%	Ghana	[81]
CTX-M-15 (77%) CTX-M-1 (6%) CTX-M-2 (6%) CTX-M-14 (8%) SHV-12 (3%)	Animals	NA	36%		[27,28]
CTX-M	Humans	NA	32%	Guinea Bissau	[82]
CTX-M-15 (67%) CTX-M-14 (17%) CTX-M-9(16%)	Animals	IncF [100% (98% and 16% associated with CTX-M-15, CTX-M-14 and CTX-M-9, respectively)]	20%	Guinea	[24]
CTX-M-15 (82%) CTX-M-14 (9%) SHV-12 (8%) SHV-27 (1%)	Humans	NA	58%	Mali	[83–86]
CTX-M-15 (82%) CTX-M-27 (15%) SHV-12 (3%)	Environments	NA	NA		[38]
CTX-M-15 (100%)	Humans	NA	6%	Mauritania	[87]
CTX-M-15 (91%)	Humans	IncF [57% (100% associated with CTX-M-15)]	31%	Niger	[88]

SHV-12 (3%)		NT [43% (100% associated with CTX-M-15)]			
SHV-2a (3%)		NA			
SHV-44 (3%)		NA			
CTX-M-15 (90%) CTX-M-1 (6%) CTX-M-3 (4%)	Humans	IncF [54.5% (100% associated with CTX-M-15)] IncH [27.3% (100% associated with CTX-M-15)] IncK [18.2% (100% associated with CTX-M-15)]	11-72%	Nigeria	[33,89–99]
CTX-M-14 (4%) CTX-M-15 (92%) CTX-M-27 (2%) CTX-M-55 (2%)	Animals	IncF [100% (100% associated with CTX-M-15)]	71%		[20,21,30,31]
CTX-M-15 (72%) SHV-12 (14%) SHV-2 (14%)	Environments	IncF [100% (100% associated with CTX-M-15)]	79%		[40,41]
CTX-M-15 (100%)	Humans	NA	NA	São Tomé and Príncipe	[100]
CTX-M-15 (60%) SHV-12 (30%) SHV-2 (10%)	Humans	IncHI2 (50%), IncN (33%), IncF (17%) IncHI2 (100%) IncN (100%)	26-79%	Senegal	[101–106]
CTX-M-15 (100%)	Animals	NA	NA		
CTX-M	Humans	NA	64%	Sierra Leone	[107]
CTX-M	Humans	NA	22-55%	Togo	[108]

ESBL-PE: Extended-spectrum β -lactamase- producing *Enterobacteriaceae*; NA: not available; NT: not typeable; CAR: Central Africa Republic ; DRC: Democratic Republic of Congo.