



OPEN Non-O157 shiga toxin–producing *Escherichia coli* (STEC) in Africa: a one health systematic review and meta-analysis of prevalence, antimicrobial resistance, and clinical outcomes

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Non-O157 Shiga Toxin–Producing *Escherichia coli* (non-O157 STEC) pathogens cause considerable debilities and were reviewed for its prevalence, antimicrobial Resistance, and clinical outcomes in Africa from a One Health perspective. Following the PRISMA guidelines, studies reporting non-O157 STEC in human, animal, environmental and food samples from African countries were retrieved from African Journals Online, Scopus, Google Scholar, Web of Science, and PubMed. Retrieved data were analyzed using random-effects model by the DerSimonian–Laird method and assessment of risk-of-bias for individual studies, with Egger’s test. Of 22 included studies, most were conducted in Northern Africa (59%, $n = 13$). The most frequently reported STEC serogroups were O26 (26.60%), O45 (8.21%), O111 (7.6%), O121 (3.34%), O145 (4.10%), O78 and O91 (4.10%). Virulence genes including *Stx1* (24.9%), *stx2* (19.7%), and *eae* were commonly detected in isolates from human samples than in isolates from other sources. Pooled prevalence of non-O157 STEC in African countries was 20.7% (95% CI: 11.1–30.2, $I^2 = 97.4\%$, $p = 0.0001$) with combined human-animal sources pooled prevalence of 27.3% (95% CI: 9.2–45.3; $I^2 = 98.6\%$; $p = 0.0001$). More than 10% pooled resistance to commonly used antibiotics and high proportion of strains harboring *blaTEM*, *blaVIM*, *blaNDM*, *blaCTX*, *blaOXA* encoding ESBLs, *tet* variants for tetracycline, *sul1* and *sul2* encoding resistance for sulphamethoxazole, and *qnrS* (for fluoroquinolone resistance) were recorded. Non-O157 STEC associated infections showing clinical presentations characterized by diarrhea, gastroenteritis, and UTI which were mostly observed in children < 5 years. The prevalence rates and antimicrobial resistance pattern of non-O157 STEC serogroups is a growing concern to African populations and clinical outcomes. There is a need for public enlightenment on prevention of non-O157 STEC with One Health approach.

Keywords non-O157 STEC, human, environment, animal, Antibiotic resistance gene, Virulence genes

Escherichia coli are enteric commensals commonly found in human, animal and environment¹. Some *E. coli* strains are pathogenic types and express virulence factors such as shiga toxins (*stx1* and *stx2*) and were referred to shiga toxin-producing *E. coli* (STEC)^{2,3}. STEC are widely known zoonotic food-borne pathogens associated with severe gastrointestinal disease characterized by bloody or non-bloody diarrhea, hemorrhagic colitis (HC) and life-threatening hemolytic uremic syndrome (HUS)^{3,4}. Among the STEC, STEC O157 serogroup is considered a major global health threat, usually implicated as the cause of HC and HUS outbreaks in several countries

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including, Africa^{4–6}. Among other common serogroups of non-O157 STEC; O26, O45, O103, O111, O121, and O145 are known as “big 6” non-O157 STEC; and were frequently associated with global major food-borne illnesses and also prevalent in Africa^{5,7}. The non-O157 STEC infections were implicated in several sporadic food and water-borne infections which were generally caused by the consumption of contaminated water, and animal derived products (mostly cattle, sheep, and pig)^{4,8,9}.

Globally, non-O157 STEC pathogens are responsible for over 169,600 human illnesses in USA, and also reported in pigs and pork in Egypt¹⁰, France¹¹, and Switzerland¹². Considering the One Health perspective of non-O157 STEC prevalence in African sub-regions including Southern, Western, Eastern, Central and Northern Africa, there are reported cases of non-O157 STEC infections in human populations^{13,14}, animal^{15,16} and also found in water sources (environment)^{17–19}. In view of the rising prevalence of non-O157 STEC in Africa, the growing evidence of antimicrobial resistance (AMR) among the emerging serogroups heightens the public health concern^{7,20}, while the use of antimicrobial therapy further threatens shiga toxin pathogenesis and increase risk of HUS¹³. In addition, a few antibiotics such as sulphamethoxazole could exacerbate non-O157 STEC infections through increased shiga toxin production, leading to severe morbidity in the human population^{13,21}. Persistent genetic transfer of antimicrobial resistance genes (ARGs) in non-O157 STEC is poorly estimated in several African countries²⁰. Despite reports of reliable indicators used for the monitoring and surveillance of AMR in livestock, humans and along the food chain, there is a paucity of reports linking ARGs in humans, animal and water.

Prevalence of non-O157 STEC infection impact clinical outcome of several infected individuals presenting mild, watery bloody or non-bloody diarrhea leading to HC, HUS, thrombocytopenia and deaths in some cases^{3,22}. Despite a high numbers of studies on non-O157 STEC in Africa, information on the infection sequelae is limited to guide clinical management mostly in paediatric cases with renal complications and damage to the colon with complications^{13,23}. For clinical evaluations, investigations reporting the complex interplay of non-O157 STEC virulence and host factors are limited in several African countries.

Despite several reports of the epidemiology of non-O157 STEC with or without the One Health approach in Africa^{12–15}, there are limited data on the prevalence of non-O157 STEC antimicrobial resistance with clinical outcomes, making epidemiological estimation difficult. Therefore, this systematic review aimed to evaluate the pooled prevalence of non-O157 STEC across Africa sub-regions, estimate the pooled antimicrobial resistance rates and prevalent ARG driving AMR carriage, and estimate the complex interplay of virulence and host factors impacting the clinical outcome and infection burden in Africa.

Methods

Study design

This study was conducted from 17 October 2025 to 21 January 2026 following the use of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines³⁴. Searches for published studies in African countries were conducted in African Journals Online, Scopus, Google Scholar, Web of Science, and PubMed using Boolean operators to combine specific keywords such as non-shiga toxin-producing *Escherichia coli* O157 (non-O157 STEC), “Verotoxin-Producing *Escherichia coli*”, One Health, human, clinical, animal and environmental samples, water sources, food products, and, names of African countries as shown (Supplementary File (SF) -Table 1).

Study eligibility criteria

All available studies retrieved from different databases were selected based on the following inclusion criteria; (a) Full-text of published articles reporting non-O157 STEC in African countries with reported investigations in human, animal, food products, and environment, or animal and human, or human and environment, or animal and environment (b). Articles should provide details of the study population, sources of sample(s) and sample size, number of isolates, and methods used to detect non-O157 STEC strains (c). All articles were published in English.

Exclusion criteria: Articles with incomplete information, focused solely on clinical, animal/livestock, or environmental samples without a broad context were excluded. In addition, conference proceedings, review articles, abstracts, short communications, letters, posters, duplicate publications, and studies from non-African countries were also excluded.

Screening and data extraction

The titles and abstracts of relevant articles were reviewed in detail by the authors. To determine the eligibility of the reported articles, full-text of the selected paper was carefully reviewed, and only studies that met the criteria were included for the review. Data extraction was done separately, and systematically collected and organized in an MS Excel spreadsheet. Obtained data included the author names, publication year, country, study period, country, sampling population (specifically the animal, human, or environmental source/host), reservoirs studied (human, environment and food products; human, animal, food products and environment, animal and human, animal environment), methods for non-O157 STEC detection, number of isolates from each source (animal, human, environment, food products), prevalence of isolates, antibiotic resistance pattern and clinical presentations.

Study quality

Quality of the selected studies was assessed by using the Joanna Briggs Institute checklist (SF-Table 2) followed with the scoring of nine questions; 1 for a YES response or 0 for a NO response (SF-Table 3).

Data analysis

Following the entry of the extracted data on Excel 2013, all data were analysed for descriptive, meta-analysis, forest plots, and funnel plots using R software (version 4.4.2) with the meta package (version 4.20-2). The random-effects model was applied for the estimation of pooled prevalence based on the DerSimonian-Laird (DL) method taking the confidence interval at 95%. To determine the heterogeneity across the studies, the inverse variance index (I²) and Cochran's q were used and I² value of > 75% suggested a considerable heterogeneity taking significance at p-value < 0.05²². Egger's test was used to validate the asymmetry of the funnel plot.

Result

Characteristics of the included studies and distribution of samples, Sources, and methods of isolates identification

With our search strategy, a total of 1,503 articles on non-O157 STEC that reported human, animal, environment and food products from African countries were generated from various databases. Following screening of the title and abstract, 64 articles were assessed for eligibility and 22 articles were selected for data synthesis after exclusion of 42 studies that have duplicated data and unavailable full text (Fig. 1). Of all the included studies, 59% were from Northern Africa ($n = 13$), Southern Africa (27%, $n = 6$), Western Africa (9%, $n = 2$), and Eastern Africa

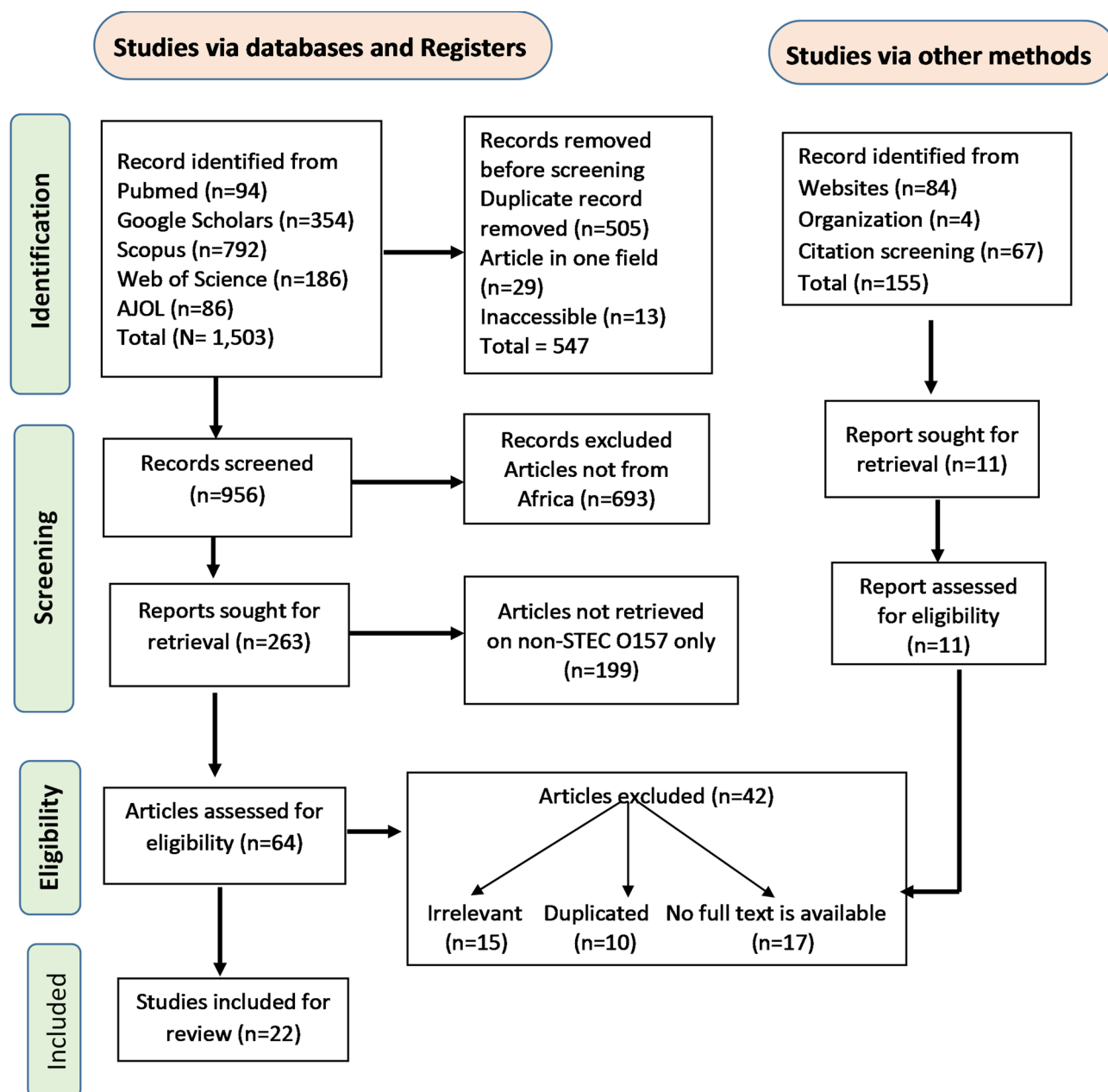


Fig. 1. PRISMA flowchart showing the selection processes of articles on non-O157 STEC in Africa.

(4.5%, $n = 1$), while no study met the set criteria from Central Africa (SF-Fig. 1A and B). Of 22 detailed reports on non-O157 STEC, Southern and Northern Africa sub-regions had higher rates of reports from human, animal and environment compared to others (SF-Fig. 1C). Samples from human-animal-environment ($n = 2$), human-animal ($n = 8$), animal-environment-food ($n = 2$), animal-food ($n = 4$), human-food ($n = 1$), human-animal-food ($n = 2$), animal-environment ($n = 2$) and human-animal-environment-food ($n = 1$) were recorded (Table 1). The methods utilized for the identification of isolates included culture-based techniques and standard biochemical tests (100%, $n = 22$), agglutination test (68.2%, $n = 15$), serotyping using *E. coli* O157 anti-sera (40.9%, $n = 9$), PCR (100%, $n = 22$), whole genome sequencing (9.1%, $n = 2$), pulse field gel electrophoresis (PFGE) (9.1%, $n = 2$), and metagenomics (4.6%, $n = 1$). For antimicrobial susceptibility, 9 studies utilized disc diffusion assay and two study investigated antimicrobial susceptibility testing (AST) using Whole genome sequencing (WGS) for antibiotic resistance gene detection while eleven studies did not report the use of any AST method (Table 1).

Prevalence serogroups

Of all the serogroups reported, the “big six” non-O157 STEC were more prevalent, accounting for O26 (26.60%), O45 (8.21%), O111 7.6%), O121 (3.34%), O145 (4.10%) and O78 and O91 (4.10%) compared to other serogroups (SF-Fig. 2A). Among the serogroups, O78 (20.4%), O26 (15.3%), O55 (11.5%) and O117 (10.0%) were most prevalent in human samples, while O26 (43.34%), O78 (31.6%), O111 (14.4%), and O128 (11.3%) were most common isolates in animal samples. O8 (7.0%), O20 (3.3%), and O178 (3.5%) were abundant in environmental samples and O26 (24.4%), O111 (15.3%), and O86 (9.5%) were common in food samples (SF-Fig. 2B). Only 7 studies reported virulence genes expressed among the serogroups. Shiga toxins (*stx1*, 24.9%; *stx2*, 19.7%), intimin (*eae*, 2.1%), hemolysin (*sheA*, 8.1%) and enterohemo-lysin (*ehx*, 7.2%) were predominant among the recovered serogroups from human samples. Among serogroups from animal samples, *stx1* (31.8%), *stx2* (24.4%), *eae* (11.2%), hemolysinA (*hlyA*, 11.6%) and *ehx* (4.0%) were most common while *stx1* (8.3%), *stx2* (20.8%), *eae* (16.7%), and *sheA* (37.5%) were dominant among the serogroups from environmental samples. Of the serogroups from food products, *stx1* (21.4%), *stx2* (22.5%), *eae* (34.7%), and *hlyA* were identified (SF-Fig. 2C).

Meta-analysis

Distribution of non-O157 STEC Prevalence by Country

The pooled prevalence of non-O157 STEC in African countries was 20.7% (95% CI: 11.1–30.2,) with high heterogeneity ($I^2 = 97.4\%$, $p < 0.0001$) across the studies indicating substantial variability. This suggests that the included studies differ considerably (Fig. 2A). High pooled prevalence in South Africa at 30.3% ($n = 6$; 95% CI: 3.4–57.2, $I^2 = 93.9\%$, $p < 0.001$), and Egypt at 19.8% ($n = 6$; 95% CI: 7.5–32.1, $I^2 = 98\%$, $p < 0.001$). A high heterogeneity was observed ($I^2 = 97.4\%$, $\tau^2 = 0.0502$, $p < 0.0001$) and further confirmed by an asymmetrical funnel plot (Fig. 2B). Based on sub-regional pooled prevalence; South Africa at 30.3% (95% CI: 3.4–57.2, $I^2 = 93.9\%$, $p < 0.0001$) was estimated in South Africa (SF-Fig. 3) and 19.1% (95% CI: 7.7–30.4, $I^2 = 97.8\%$, $p < 0.0001$) in Northern Africa sub-region (SF-Fig. 4).

Distribution of non-O157 STEC prevalence by the combination of sample Sources

Pooled prevalence of non-O157 STEC based on human-animal-environment sources was estimated as 13.7% (95% CI: 0.0–34.7; $I^2 = 98.4\%$; $p = 0.0001$) (SF-Fig. 5). Combination of the human-animal sources gave an estimated pooled prevalence of 27.3% (95% CI: 9.2–45.3; $I^2 = 98.6\%$; $p = 0.0001$) while the sample sources from animal-environment pooled prevalence was 19.1% (95% CI: 3.4–34.8; $I^2 = 91.1\%$; $p = 0.0001$). For animal-environment-food products sources, only one study provided prevalence of 14.9% (95% CI: 10.8–19.9) and animal-food sources gave pooled prevalence of 12.0% (95% CI: 8.5–15.6; $I^2 = 49.6\%$; $p = 0.1376$). Sample sources from human-animal-food sources gave a pooled prevalence of 20.6% (95% CI: 0.0–44.3) with high heterogeneity ($I^2 = 97.9\%$; $p = 0.0001$), suggesting potential variability in the diagnostic methods, study quality, sampling strategies, and etc. The funnel plot showed the distribution bias in effect estimates and significant meta-regression analysis among studies (SF-Fig. 6; Table 4).

Pooled antimicrobial resistance (AMR) prevalence

The pooled antimicrobial resistance was estimated based on the combination of all the resistant isolates reported from 9 out of 22 included studies (SF-Figs. 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26 and 27), to provide a holistic perspective of AMR prevalence based on the One Health context. Phenotypic antibiotic resistance subgroup showed more than 10% pooled non-O157 STEC strain resistance to chloramphenicol, tetracycline, norfloxacin, rifampicin, ampicillin, streptomycin and erythromycin with 95%CI ranging between 0.00 and 58.2 and the highest I^2 at 98% as shown in Table 2.

Genotypic resistance

Very few studies ($n = 6$) analyzed antibiotic resistance genes (ARGs) revealing non-O157 STEC serogroups isolated from urine, ear, poultry and cattle fecal samples in Egypt to harbour ESBIs (*blaCTX-M-1*, *blaTEM*), tetracycline (*tetA*, *tetB*, *tetC*, *tetE*, and *tetG*)². In other studies in South Africa⁸ and Egypt^{9,18,19}, serogroups from animal manure, soil, water, raw cow milk, ice cream, raw beef, animal stool and human stool samples encoded *sul1*, *sul2*, *blaCTXM*, *blaOXA*, *blaTEM qnrS*, *blaVIM*, *blaNDM*, *blaTEM blaCTX*, *blaOXA*, *blaTEM* and *tetA* genes as shown in Table 3.

Clinical outcome of non-O157 STEC infections

A total of 14 studies reported clinical presentations of subjects with non-O157 STEC infections in various African countries (Table 4). Of these studies, 4/14 studies reported populations of children and adults hospitalized with

African Regions	Authors	Year of publication	Country	Sources	Sample number	Samples				Methods	Antimicrobial assay
					Human	Animal	Environment	Food			
					N(n)						
Southern Africa	[23]	2024	South Africa	Cattle (Feedlots), abattoirs (walls and floor, faeces, rinsates and effluents), retail raw beef and ready to-eat beef products.	73(5)	NA	0(0)	12(4)	61(1)	Culture, biochemical tests, serotyping, PCR, WGS	WGS
	[22]	2022	South Africa	Feedlot, abattoir floor/wall, animal (the retail red meat)	261(39)	NA	76(13)	147(11)	38(15)	Culture, biochemical tests, serotyping, PCR PFGE	Disk diffusion
	[14]	2022	South Africa	Water and animal	25(11)	NA	18(10)	7(1)	NA	Culture, biochemical tests, PCR	NA
	[8]	2022	South Africa	Animal manure, soil and water	192(40)	NA	48(28)	144(12)	NA	Culture, biochemical tests, PCR	NA
	[4]	2025	South Africa	Animal (Sheep, goat, manure), water	207(12)	NA	199(12)	8(0)	NA	Culture, biochemical tests, PCR	Disk diffusion
	[3]	2017	South Africa	Human stool and animal	4(4)	4(4)	0(0)	0(0)	0	Culture, biochemical tests, PCR, metagenomics	NA
Northern Africa	[21]	2020	Egypt	Animal (fecal, beef) human (urine),	75(60)	15(15)	60(45)	NA	NA	Culture, biochemical tests, serotyping, PCR	Disk diffusion
	[19]	2017	Egypt	Food (meat samples) Human (stool)	310(18)	50(4)	NA	NA	260(14)	Culture, biochemical tests, serotyping, PCR	Disk diffusion
	[18]	2021	Egypt	Food (raw beef, milk), animal (fecal), human (stool)	400(35)	100(10)	100(12)	NA	200(13)	Culture, biochemical tests, serotyping, PCR	Disk diffusion
	[16]	2021	Egypt	Animal (cattle feces, milk samples), Human stool	260(18)	80(13)	180(5)	NA	0(0)	Culture, biochemical tests, serotyping, PCR	Disk diffusion
Northern Africa (contd)	[13]	2013	Egypt	Animal products (meat products, poultry products, seafood and dairy products.). Water, Human (stool and urine) and food	384(12)	183(4)	156(7)	31(1)	14(0)	Culture, biochemical tests, serotyping, PCR	Disk diffusion
	[12]	2023	Egypt	Human (stool) and animal (raw milk, karish cheese, fresh meat, and minced meat)	250(70)	50(24)	200(46)	NA	0(0)	Culture, biochemical tests, serotyping, PCR	NA
	[11]	2014	Egypt	Human and animals	461(56)	93(5)	368(51)	NA	NA	Culture, biochemical tests, serotyping, PCR	NA
	[10]	2015	Egypt	raw beef and chicken Food (kofte, beef burger, fresh sausage and beef luncheon)	300(46)	NA	100(25)	NA	200(21)	Culture, biochemical tests, serotyping, PCR	NA
	[9]	2018	Egypt	Raw cow milk and Food (ice cream)	90(8)	NA	60(2)	NA	30(6)	Culture, biochemical tests, serotyping, PCR	NA
	[7]	2025	Morocco	Food (milk) animal (poultry and turkey)	310(34)	NA	150(17)	NA	160(17)	Culture, biochemical tests, PCR	NA
	[6]	2024	Egypt	Food, animal product, dairy products, Fresh vegetables, chicken, fish, sewage water, Urine and stool samples	266(105)	118(65)	51(10)	7(7)	90(28)	Culture, biochemical tests, ERIC- PCR	NA
	[5]	2009	Egypt	Human (stools, urine) Animal (sheep, cattle, seafood, and chicken, diary products), water,	384(10)	183(3)	71(6)	31(1)	99(0)	Culture, biochemical tests, PCR	NA
	[2]	2018	Egypt	Human (urine, ear), poultry, animal (cattle,)	207(60)	150(28)	57(32)	NA	NA	Culture, biochemical tests, PCR	NA
Western Africa	[17]	2012	Burkina Faso	Retail meats and intestines, human stool samples from children	778(21)	658(1)	120(200)	NA	0(0)	Culture, biochemical tests, serotyping, PCR PFGE	Disk diffusion
	[1]	2011	Nigeria	Animal (pigs, cattle, goats, sheep) and human	219(72)	10(10)	113(52)	NA	96(10)	Culture, biochemical tests, PCR	NA
Eastern Africa	[15]	2024	Ethiopia	fish samples, water samples, human stool samples)	750(18)	150(5)	450(7)	150(6)	NA	Culture, biochemical tests, serotyping, PCR, WGS	Disk diffusion

Table 1. Characteristics of Included studies. NA, Not available; WGS, Whole genome sequence; PCR.

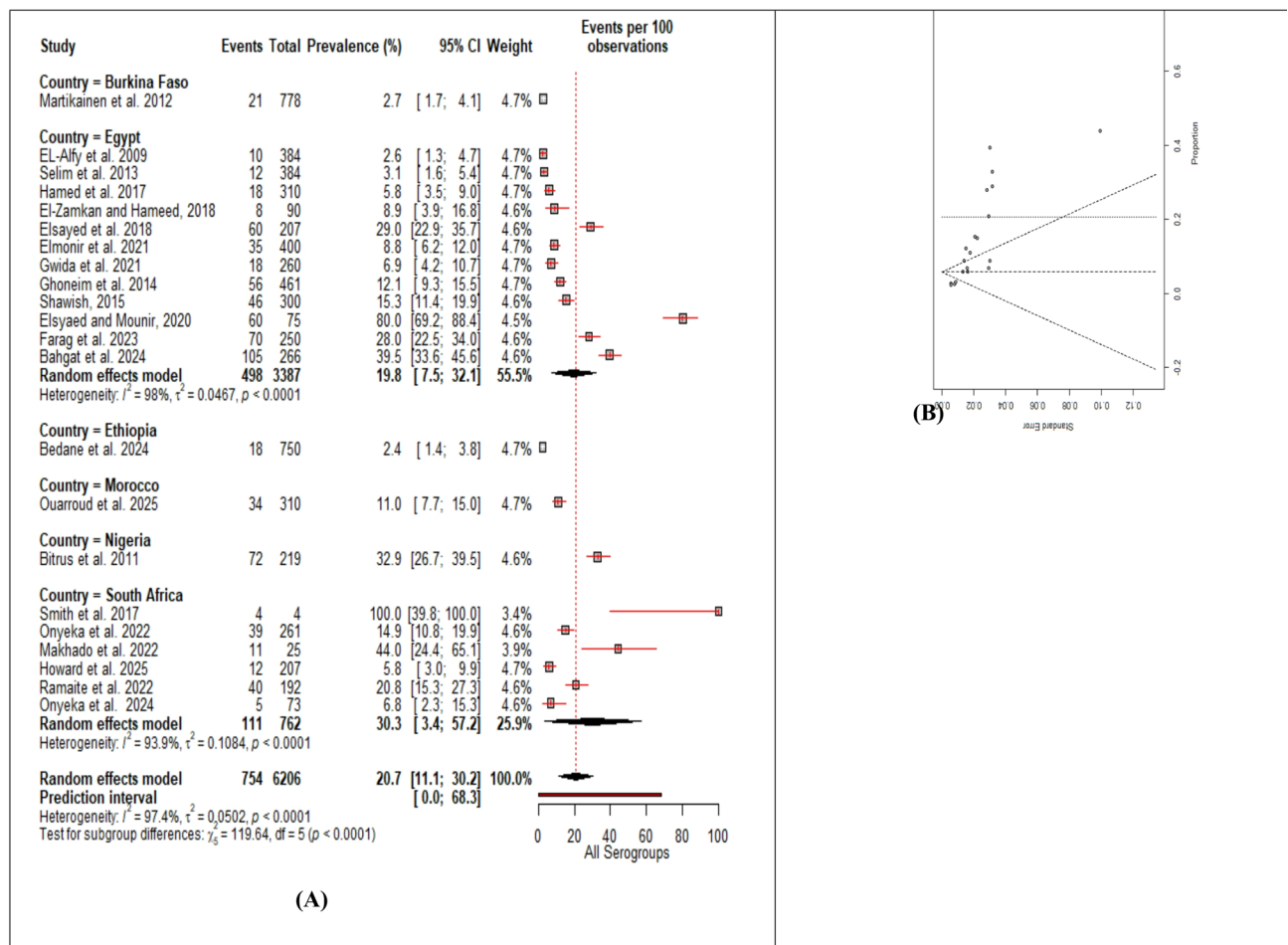


Fig. 2. (A) Forest plot of pooled prevalence of non-O157 STEC from the One Health perspective in Africa. (B) Funnel plot with the adjusted average prevalence of non-O157 STEC from the One Health perspective in Africa.

clinical presentations of diarrhea and gastroenteritis^{3,6,12,19} while other studies reported children and adult populations that were non-hospitalized but with clinical manifestations of diarrhea^{1,2,5,11,15,17,18}, gastroenteritis, enteric illness, and symptomatic UTI^{13,16,21}. Among the hospitalized populations, 4 out of 50 children admitted at Referral hospital in Giza Governorate, Egypt¹⁹, 24/50 patients at Aswan Governorate, in south of Egypt¹² and 19/118 patients admitted in hospital at Mansoura, Egypt⁶ presented non-bloody diarrhea and another 11/118 patients had UTI⁶, while children under 5 years in South Africa were reported to present bloody diarrhea and UTI³ due to non-O157 STEC infections. For non-hospitalized subjects, 10/100 adults in Egypt¹⁸, 1/21 in Burkina Faso¹⁷, 11/150 in Ethiopia¹⁵, 78/183 in Egypt¹³, 5/93 in Egypt¹¹, 2/179 in Egypt⁵, and 9/10 in Nigeria¹ presented non-bloody diarrhea but 11/118 from Egypt⁶ showed UTI and 9/150² and 7/80¹⁶ in Egypt had other infections including otitis media.

Risk of publication bias

The included studies showed high heterogeneity with the I^2 more than 97.8% at $p < 0.0001$. Funnel plot showed a slight asymmetrical distribution while the intercept of the Eggers regression model at p -value < 0.0001 suggests potential publication bias. To further validate our findings, an influential analysis was carried out by sequential omission of individual studies and no alteration was recorded with the estimated pooled prevalence but kept fluctuating at 0.14% (95% CI: 0.08–0.22) (SF-Figs. 28 and 29; Table 5).

Discussion

Considering the number of studies reporting non-O157 STEC in African countries, South Africa and Egypt had a higher number of studies compared to other African countries. A High number of specialized research institution, improved socio-economic conditions of South Africa and Egypt with considerable investment in research studies, have enhanced studies on non-O157 STEC. Low reports from other African countries do not connote the absence of these strains but poor and inadequate laboratory infrastructure and funding, impact reporting of these pathogens. Despite low reports on inter-relatedness of human, animal, and environment within the One Health context, the included reports reveal the implication of zoonotic potential of non-O157

Subgroup (Antibiotics)	Number of studies	Total number of samples	Number of resistant Isolates	Prevalence % (95%CI)	I ² (%)	P-value
CHL	5	2071	131	18.9(0.0–45.4)	98.1	0.001
CIP	5	2184	30	1.1(0.4–1.8)	69.0	0.0117
IMP	5	1950	44	2.2(0.1–4.2)	80.7	0.0004
NA	5	2379	83	4.0(0.9–7.1)	81.2	0.0003
TET	9	3037	184	16.3(1.6–31.1)	96.0	0.0001
AMK	2	467	5	1.0(0.1–1.9)	-	0.4929
NOR	2	396	9	14.4(0.0–45.3)	81.9	0.0187
RA	2	396	10	19.0(0.0–58.2)	87.5	0.0046
SPT	5	1695	42	2.7(0.5–4.8)	79.3	0.0007
TRIM	4	1407	40	9.7(0.0–26.0)	89.0	0.0001
AMP	9	2313	108	18.9(0.0–38.7)	96.7	0.00001
AUG	6	1249	29	2.3(0.4–4.1)	75.0	0.0013
CFX	4	2099	44	1.6(0.2–3.0)	84.5	0.0002
GN	5	1460	31	3.0(0.1–6.0)	87.2	0.0001
STR	5	1581	86	10.0(0.0–20.9)	95.5	0.0001
CAZ	4	1924	48	9.5(0.0–26.1)	87.6	0.0001
CEF	5	1064	62	7.2(0.0–14.4)	92.0	0.0001
ERY	5	889	82	25.5(2.9–48.1)	95.7	0.0001
NIT	2	396	7	14.2(0.0–45.6)	82.5	0.0168
DA	4	1381	64	13.0(0.0–28.7)	94.9	0.0001
PEN	5	1613	114	13.2(0.4–26.0)	95.1	0.0001

Table 2. ; Pooled antibiotic resistance prevalence based on meta-analysis. (CHL, chloramphenicol; CIP, ciprofloxacin; IMP, imipenem; NA, nalidixic acid; TET, tetracycline; AMK, amikacin; NOR, norfloxacin; RA, rifampicin; SPT, sulfamethoxazole; TRIM, trimethoprim; AMP, ampicillin; AUG, amoxicillin-clavulanate; CFX, cefotaxime; GN, gentamicin; SPT, streptomycin; CAZ, ceftazidime; CEF, cefuroxime; ERY, erythromycin; NIT, nitrofurantoin; DA, clindamycin; PEN, penicillin)

Authors	Year	Country	Sources	Antimicrobial resistance genes
[4]	2025	South Africa	Animal (Sheep, goat, manure), water	blaSHV (25), aadA (5), ampC (5), and Methionine sulfoxide reductase A (MsrA) (4).
[8]	2022	South Africa	Animal manure, soil and water	sul1 (40), blaOXA (39), blaCTX-M (37), sul2 (36), blaTEM (33), qnrS (9), blaTEM, sul2 and blaTEM genes (8), sul2, blaCTX-M, and blaTEM (8)
[2]	2018	Egypt	Human (urine, ear), poultry, animal (cattle,)	blaCTX-M-1(8), blaTEM (3), tetA (44), tetB (2), tetC (2), tetE(6), tetG (27)
[9]	2018	Egypt	Raw cow milk and Food (ice cream)	sul1(40), sul2 (36), blaCTXM-M (37), blaOXA (39), blaTEM (35) qnrS (17)
[18]	2021	Egypt	Food (raw beef, milk), animal (fecal), human (stool)	blaVIM (7), blaNDM (1), blaTEM (15), blaCTX (10), blaOXA (1)
[19]	2017	Egypt	Food (meat samples) Human (stool)	blaTEM (9), tetA (2).

Table 3. Antibiotic resistance genes distribution from different sources.

STEC on human populations^{12,24} but there is no reported study to genetically link the transmission pathways. Conventional culture-based methods that are usually deployed in Africa, with the commonly used disc diffusion assay for estimation of phenotypic resistance, could limit serogroup characterization and antibiotic resistance evaluation. Molecular-based methods such as PCR, WGS, PFGE; provide robust and explicit data for reliable outcomes³.

The “big six” non-O157 STEC is a global public health challenge and the prevalence was similarly observed in this study²⁵. Recording high prevalence rates of O26, O111, and O78 in human, animal, food and environmental samples from several African countries suggest an emerging threat to population health and a potential public health hazard associated with the consumption of contaminated animal or food products^{7,10,26}. The carriage of virulent toxins (including *stx* variants, *eae*, *hlyA*) among these serogroups provide epidemiological information and implication for population health and risk of foodborne infection particularly in low income settings in Africa.

Globally, the rising prevalence of non-O157 STEC threatens the population health²⁷, which was also reported in Africa^{1,15,28}. The estimated pooled non-O157 STEC prevalence of 20.7% from reported studies in the One Health context in Africa needs to be considered for surveillance in order to prevent possible international spread. This pooled prevalence further suggest major international health concern, primarily linked to food, water and animal products that could affect public health, economic outcome, hospital visit and livestock management. The

Authors	Year	Country	Design (dates)	Population (clinical condition)	Number of subjects ^b	Number of subjects with clinical presentations for non-O157 STEC ^b				
						Non-bloody diarrhea	Bloody diarrhea	HUS	UTI	*Other infections
[15]	2024	Ethiopia	Cross-sectional study (December 2020 to June 2022)	Children and adults (diarrheic out-patients)	150	11	0	0	0	0
[6]	2024	Egypt	Prospective study (November 2018 to April 2020)	All ages (hospitalized) ^a	118	19	0	0	11	0
[12]	2023	Egypt	Prospective study (February 2021 to November 2022)	All ages (hospitalized gastroenteritis) ^a	50	24	0	0	0	0
[18]	2021	Egypt	Prospective study (March and August 2016).	Diarrheic children and adult (non-hospitalized)	100	10	0	0	0	0
[16]	2021	Egypt	Prospective study	All ages (non-hospitalized) ^a	80	0	0	0	0	7
[21]	2020	Egypt	Prospective study (November 2017 to November 2018)	Children and adult (non-hospitalized)	20	0	0	0	15	0
[2]	2018	Egypt	Prospective study (2018)	Adult (non-hospitalized) ^a	150	0	0	0	19	9
[19]	2017	Egypt	Prospective study (January 2012 to August 2016)	Children with diarrhea (Hospitalized)	50	4	0	0	0	0
[3]	2017	South Africa	Retrospective study (13 February 2017)	Children under 5 years (Hospitalized for diarrhea)	4	0	2	2	0	0
[11]	2014	Egypt	Prospective study (2014)	All ages (gastroenteritis) ^a	93	5	0	0	0	0
[13]	2013	Egypt	Prospective study (2013)	All ages with enteric illness/ symptomatic UTI ^a	183	78	0	0	0	0
[17]	2012	Burkina Faso	Prospective study (March and August 2016).	Children (non-hospitalized)	21	1	0	0	0	0
[1]	2011	Nigeria	Prospective study (July and October, 2009).	Children and adult with gastroenteritis	10	9	0	0	0	0
[5]	2009	Egypt	Prospective study (September 2008 through October 2009)	All ages (Non-hospitalized Symptomatic UTI ^a)	179	2	0	0	1	0

Table 4. Clinical presentations of subjects with non-O157 STEC infections. (Note: a; no defined age group or classification of the population; b, only human population reported in the selected studies; *Other samples include urine, stool, ear swab (diagnosed for otitis media); UTI, urinary tract infection)

high prevalence rates in Northern and Southern Africa countries could be attributed to increase contamination of water sources with livestock fecal material^{8,29} and frequent contamination of vegetables with the use of animal manure^{4,30}. Low pooled prevalence or studies from other African countries should not be interpreted as the absence of non-O157 STEC but could be attributed to poor detection and inadequate diagnostic facilities mostly in rural settings across African sub-regions. Similar pooled prevalence rates were observed in the USA^{31,32} and Germany³³ due to retailing of contaminated meat and meat products, which was identified as major sources of transmission. Having recorded significant pooled prevalence in human-animal-environment samples, or inter-related samples of human, animal, food and environment, this One Health meta-analysis provided evidence for possible spread of these pathogen, increase infection rates, and debilities with resultant morbidities.

Since the antibiotic resistance rate was collectively reported by several studies, estimation of more than 10% pooled antibiotic resistance among the strains to commonly used antibiotics (including chloramphenicol, tetracycline, norfloxacin, rifampicin, ampicillin, streptomycin and erythromycin) highlights common AMR spread from animal, food and water to human or vice versa. The use of antibiotics as prophylaxis or growth promoters in livestock and poultry have increase antimicrobial accumulation as residue in meat and also serve as possible reservoir for ARG accumulation^{20,34}. There is need for public enlightenment on the use of antibiotics in animals and implication of AMR transfer through the food chain^{9,35}. Despite less number of studies reporting ARG carriage in non-O157 STEC pathogens, the detection of *blaCTX-M-1*, *blaTEM*, *blaNDM*, *blaOXA*, suggested high level beta-lactamase drug resistance, while *tetA*, *tetB*, *tetC*, *tetE*, *tetG*, *sul1* and *sul2* indicate multiple carriage of resistant gene among different serogroups. Detection of ARG in human stool, food (milk, ice cream), animal products (raw beef) and environment (animal manure, soil, water) justifies the need to step up AMR control with One Health perspective in Africa. Rigorous surveillance for ARG-encoded non-O157 STEC serogroups in Africa countries is very important towards prevention of zoonotic transmission and possible community outbreak of non-O157 STEC infection.

With 14 studies reporting non-O157 STEC infections, these pathogens pose a substantial threat to clinical outcomes mostly among children under 5 years. Clinical presentations of non-O157 STEC-associated infection among the hospitalized and non-hospitalized adult populations substantiate the clinical manifestation of diarrhea, gastroenteritis and symptomatic UTI^{36,37}. In this study, the observed clinical manifestations of non-O157 STEC infection associated with non-bloody diarrhea, gastroenteritis, and UTI could progress to severe hemorrhagic colitis, HUS, and death if poorly managed, as a result of shiga toxins and other virulence factors production^{38,39}. Non-O157 STEC infection in children less than 5 years with non-bloody diarrhea was reported to be contracted through consumption of contaminated food product mostly animal milk or water^{9,40}, leading to severe clinical outcomes. Similar non-O157 STEC infection in children less than 5 years was also reported

in Ghana⁴¹, Nigeria⁴², USA⁴³, Japan⁴⁴ etc. To curtail possible infection spread, rapid diagnosis of non-O157 STEC infections should be emphasized particularly in resource limited settings to assist clinical decision-making regarding the administration of fluid and antibiotics. Detection of non-O157 STEC infections in children and adult populations should instigate a rapid response of the public health authorities for investigation and prevention towards possible outbreak.

Strengths and limitations of the study

The current evidence provides insight into prevalent non-O157 STEC serogroups in Africa, and for robust national and international One Health strategic surveillance, research, and prevention awareness. Data from this study further substantiate non-O157 STEC-associated infection and clinical outcomes for improved clinical management and rapid response against possible outbreak. However, paucity of report from other African countries limit the generalization of the study outcome and small sample size cause bias in the interpretation of statistical analysis. Inadequate clinical details and small number of hospitalized and non-hospitalized individuals were insufficient to determine the implications of the virulence factors for different clinical outcomes.

Conclusions

The prevalence rates of non-O157 STEC serogroups, mostly O26, O111, and O78 in human, animal, food and environment indicate emerging threat to African populations and potential public health hazard associated with the consumption of contaminated animal and food products. The rising AMR prevalence and carriage of ARGs among the serogroups require public enlightenment on the use of antibiotic in animals and human and their implications on population health. Adequate deployment of rapid diagnosis of non-O157 STEC infections should be emphasized to guide clinical decisions and surveillance.

Data availability

This study is a systematic review and meta-analysis. The data analyzed were extracted from previously published studies. As such, the original data are not owned by the authors. However, the dataset compiled and used for the meta-analysis is available from the corresponding author upon reasonable request.

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Author contributions

Each author made a substantial contribution as follows; PA and ES conceived and developed the review and meta-analysis protocol. PA, AO, UE and ES; performed data extraction and quality assessment for the systematic review and contributed to the preparation of all tables and meta-analysis. PA, SD and AO conducted data extraction for the meta-analysis, carried out statistical analyses, and produced the corresponding figures and tables. PA, YA, ES. Supervised the project, and drafted the manuscript, and revised its final version. All authors contributed to data interpretation, critically reviewed the manuscript, and approved the final version for submission.

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Declarations

Competing interests

The authors declare no competing interests.

Additional information

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