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Prevalence of *Shigella* species and antimicrobial resistance patterns in Africa: systematic review and meta-analysis

Rabbi Nyarkoh¹, Alex Odoom¹ and Eric S. Donkor^{1*}

Abstract

Background Shigellosis continues to pose a significant public health problem in Africa; however, there is a lack of comprehensive knowledge regarding its prevalence, serogroup distribution, and antimicrobial resistance profiles. Therefore, the objective of this systematic review and meta-analysis was to determine the overall prevalence of *Shigella*, the distribution of species, and the patterns of antimicrobial resistance across Africa.

Methods Following PRISMA guidelines, a systematic search strategy was conducted using the PubMed, Web of Science and Scopus databases from January 31, 2024 to February 10, 2024. The study quality was assessed using the Joanna Briggs Institute checklist, and data were analyzed using the R statistical language and the R package 'meta'. The random effects model was employed to estimate the pooled prevalence, while heterogeneity was assessed using the l² statistic and prediction interval.

Results A total of 116 studies from 29 African countries were included in this meta-analysis, involving the examination of 99,510 samples. The overall pooled estimate of *Shigella* prevalence was determined to be 5.9% (95% CI: 4.9 – 7.0%). Regional prevalence showed prevalences of Southern Africa (6.9 [95% CI: 3.0 – 12.2%]), Northern Africa (6.7% [95% CI: 4.1 – 9.8%]), Eastern Africa (6.2% [95% CI: 4.9 – 7.6%]), Central Africa (4.5% [95% CI: 2.6 – 6.8%]) and Western Africa (4.0% [95% CI: 2.5 – 5.9%]). *Shigella* prevalence was found to be higher in children (6.6%, 95% CI: 3.2 – 11.1%) than in adults (3.6%, 95% CI: 1.6 – 6.3%). The most prevalent species was *S. flexneri* (53.6%, 95% CI: 46.1%—61.0%), followed by *S. sonnei* (11.5%, 95% CI: 7.7%—15.7%), *S. dysenteriae* (10.1%, 95% CI: 6.2 – 14.5%) and *S. boydii* (7.7%, 95% CI: 4.7 – 11.1%). Among the currently recommended first-line antibiotics, ciprofloxacin and ceftriaxone showed resistance prevalences of 10.0% (95% CI: 4.5%—16.9%) and 8.5% (95% CI: 2.4—16.9%) respectively.

Conclusion This review highlights the burden of shigellosis in Africa. *S. flexneri* remains the most prevalent species associated with shigellosis cases with *S. sonnei* being the second most dominant. The antimicrobial resistance patterns observed in the study suggest local antimicrobial patterns in choosing antibiotics for the treatment of Shigellosis.

Recommendation There is the need to explore alternative treatments for shigellosis with particular focus on vaccine development. There is also the need for more genomic epidemiology studies exploring the dissemination and risk of drug-resistant *S. sonnei* clones in Africa.

Keywords Shigellosis, Prevalence, Antimicrobial resistance, Serogroup, Meta-analysis, Africa

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Introduction

Shigellosis, an infectious disease resulting from *Shigella* bacteria, is a major contributor to global diarrheal-related mortality and morbidity, with an estimated 125 million cases of diarrhoea each year, resulting in roughly 160,000 fatalities[1]. The infection manifests with symptoms such as fever, abdominal cramps, nausea or vomiting, and tenesmus, as the bacteria invade and multiply within the large intestine's epithelial cells [2]. While shigellosis usually resolves without antibiotic treatment within two weeks, asymptomatic carriage of *Shigella* spp. is possible, although its prevalence and duration are unclear [3].

The historical distribution of *Shigella* in Africa has shown distinct phases. Previously, *Shigella* was uncommon as a cause of sporadic dysentery in most African countries [4, 5]. However, recent studies, such as the Global Enteric Multicentre Study (GEMS), have revealed that *Shigella* is the second most common cause of moderate to severe diarrhoea among *rotavirus*, *Cryptosporidium* spp., and *Salmonella* spp. in sub-Saharan Africa and South Asia [6], indicating a concerning incidence of shigellosis in these regions. Among the species, *S. flexneri* is the most prevalent, followed by *S. sonnei*, with variations in isolation across different countries in sub-Saharan Africa [7, 8].

The emergence of antibiotic resistance in Shigella was observed during the 1963–1964 epidemic in the Giohar district of Somalia [9]. Since 1990, all Shigella strains isolated have exhibited resistance to standard antibiotics, with uniform sensitivity only to newer quinolones and ceftriaxone [10]. Recent reports indicate increasing resistance to different antibiotic classes among Shigella spp. [11-20], with up to 41% of isolates exhibiting pentavalent resistance [21]. Nalidixic acid resistance is prevalent, with an overall rate of 57% [22]. This poses a significant public health threat, emphasizing the need for strategies to address the global issue of shigellosis. As new vaccines are being developed, up-to-date information on the burden of shigellosis in Africa is essential, as no study has specifically examined Africa in this context. Therefore, this review aimed to synthesize available data to provide an overview of Shigella prevalence trends, species distribution, and antibiotic resistance patterns in sub-Saharan Africa.

Methods

Guidelines and protocol

The PRISMA guidelines [23] were followed for conducting this systematic review and meta-analysis.

Literature search strategy and study selection

To find research on Shigellosis in Africa, we did an extensive search up until February 10, 2024. The databases searched were PubMed, Scopus, and Web of Science. Additionally, we explored other sources by following references from relevant articles. This was done through forward and backward tracking-looking at articles that cited a specific source (forward), and articles that a specific source cited itself (backward). The search keywords used were "Shigellosis" OR "Dysentery" AND "Africa", which led to the retrieval of all relevant articles. Additionally, we hand searched and selected relevant articles from Google Scholar by combining the search keywords and the individual African countries. The Mendeley Reference Manager version 1.19.8 was used to import the reference lists and check for the elimination of any duplicated studies. Additionally, the Covidence and Rayyan AI web applications (https://www.covidence.org/) (https:// new.rayyan.ai/) were used for screening, abstract review and full-text analysis to determine study eligibility.

Inclusion and exclusion criteria

For a study to be considered, it should have been conducted in Africa, reported the prevalence of *Shigella*, the distribution of the species and the antimicrobial resistance. Original cohort studies, case–control and crosssectional peer-reviewed studies were considered. Studies conducted outside Africa, nonhuman studies, studies connected to outbreaks, case reports, review articles, abstracts from conferences, commentaries, editorials, letters to editors and studies with sample sizes less than 50 were excluded. Additionally, studies that reported only *Shigella* prevalence without information on the species or antibiotic resistance patterns were excluded.

Data extraction

As a step towards accuracy reporting and minimization of errors, a data extraction template was created in Covidence software, which was used for data extraction and later exported to Microsoft Excel 2019 (version 2402, Microsoft Corp., Albuquerque, NM, USA) for further data manipulation. The following data were extracted from each eligible study: first author's name and year of publication, region of the study in Africa, country, age category of participants, study design, stool type (Diarrhoea and Non-Diarrhoea), study site (either health facility-based or community-based), prevalence of Shigella spp., species isolated, tested antibiotics and antibiotic resistance prevalence recorded for each antibiotic tested. Diarrhoea stools were those which meet the criteria of a minimum frequency threshold (≥ 3 loose/watery stools in a 24-h period) or the perception by a caregiver that it deviates from the individual's normal pattern according to WHO. The non-diarrhoea cases were either healthy controls or patients without diarrhoea. The CLSI 33rd edition suggest that for Shigella, the routine antibiotic

reporting should be on Ampicillin, a fluoroquinolone and Trimethoprim/Sulfamethoxazole. Also, it suggests that aminoglycosides, first- and second-generation cephalosporins, and cephamycins may appear active in vitro but are not effective clinically and should not be reported as susceptible[24]. This guided the antimicrobial resistance pattern reported in this study.

Study quality

The quality of each individual study was assessed through the use of the Joanna Briggs Institute (JBI) critical appraisal tool for prevalence studies [25]. The appraisal tool consists of 9 questions, each of which is assigned a score of 1 for a YES response or 0 for a NO or NOT SURE response. Studies with a total cumulative score of 50% or less were considered "high risk of bias" (poor quality); studies with a score ranging from 50 to 70% were considered "moderate risk of bias"; and studies with a score above 70% were considered "low risk of bias" (high quality) (Table S1).

Data analysis

The R Statistical language (version 4.4.1, R Core Team, 2024) (https://www.R-project.org/) [26] and the R package 'meta' version 7.0–0 were used for the meta-analysis[27]. The random effects model was used to calculate the pooled prevalence at a confidence interval of 95%. To assess heterogeneity, the I^2 statistic and the prediction interval were used. An I^2 value of 75% indicated substantial heterogeneity. A *p* value <0.05 indicated statistical significance [24]. Similarly, publication bias was assessed by plotting the prevalence estimates against the sample variance in a funnel plot. To validate the asymmetry of the funnel plot, Egger's test was used. The Freeman-Tukey double arcsine method was used in transforming the proportions before conducting a random-effects meta-analysis using conventional inverse-variance weighted methods [28].

Results

Search results

Our search across three databases (PubMed, Web of Science, and Scopus) initially yielded 1870 studies, which were supplemented by 88 articles from manual searches as well as from google scholar. Following the removal of 486 duplicates and an initial eligibility check, we examined the titles and abstracts of 1384 records. A total of 1284 studies were excluded, leaving 100 studies for full-text review. After reviewing the full texts, 60 studies were deemed ineligible for failing to meet our systematic review and meta-analysis objectives. Additionally, 15 of the manually retrieved studies were found to be duplicates already present in the PubMed and Scopus databases, and thus were eliminated. Ultimately, our

systematic review and meta-analysis included 113 studies (Fig. 1). From the 113 studies, 2 studies were conducted across multiple sites bringing the total included data points to 116.

Characteristics of the included studies

A total of 99,510 samples were examined from 29 countries across Africa; Botswana, Burkina Faso, Cameroon, Central Africa Republic, Chad, Cote d'Ivoire, Djibouti, Egypt, Ethiopia, Gabon, Gambia, Ghana, Kenya, Libya, Madagascar, Malawi, Mali, Mozambique, Namibia, Niger, Nigeria, Senegal, Somalia, South Africa, South Sudan, Sudan, Tanzania, Tunisia and Zambia. The number of studies included Botswana (n=1), Burkina Faso (n=2), Cameroon (n=3), Central Africa Republic (n=4), Chad (n=1), Cote d'Ivoire (n=1), Djibouti (n=1), Egypt (n=5), Ethiopia (n=37), Gabon (n=1), Gambia (n=1), Ghana (n=5), Kenya (n=15), Libya (n=1), Madagascar (n=1), Malawi (n=2), Mali (n=1), Mozambique (n=1), Namibia (n=1), Niger (n=1), Nigeria (n=11), Senegal (n=2), Somalia (n=3), South Africa (n=4), South Sudan (n=1), Sudan (n=4), Tanzania (n=4), Tunisia (n=1) and Zambia (n=1). Most of the studies were conducted in East Africa (n=65), then in West Africa (n=24), North Africa (n=11), Central Africa (n=9) and South Africa (n=7). The age range of the study participants was between ≤ 5 years and ≥ 60 years. Considering the study sites, 16 were community-based studies, 89 were health facility-based studies and 3 were conducted in both community and health facilities. The reviewed studies included children and adults: 46 (39.7%) studies were carried out in children younger than 5 years, 10 (8.6%) studies in children of all ages, 14 (12.1%) studies in adults, and 38 (32.8%) studies in all age groups. The sample sizes ranged from 66 to 12,150. The prevalence of Shigella spp. ranged from 0.0% to 34.6% in the studies. The major characteristics of the 116 included studies are listed in Table S2.

Meta-analysis

Prevalence of Shigella

We developed forest plots to determine the prevalence of *Shigella* spp. across Africa. The meta-analysis indicated that the overall pooled prevalence of *Shigella* across Africa was 5.9% (95% CI: 4.9 - 7.0%, $I^2 = 97.4\%$, p < 0.001) (Fig. 2). We conducted sensitivity analysis by identifying and excluding studies with great impact on the heterogeneity and effect size of the data. The overall pooled prevalence was thus reported as 5.6% (95% CI: 4.7 - 6.6, $I^2 = 94.7\%$, p < 0.001). In terms of regional prevalence, Southern Africa had a pooled prevalence of 6.9% (95% CI: 3.0 - 12.2%), followed by Northern Africa 6.7% (95% CI: 4.1% - 9.8%), Eastern Africa 6.2% (95% CI: 4.9 - 7.6%),

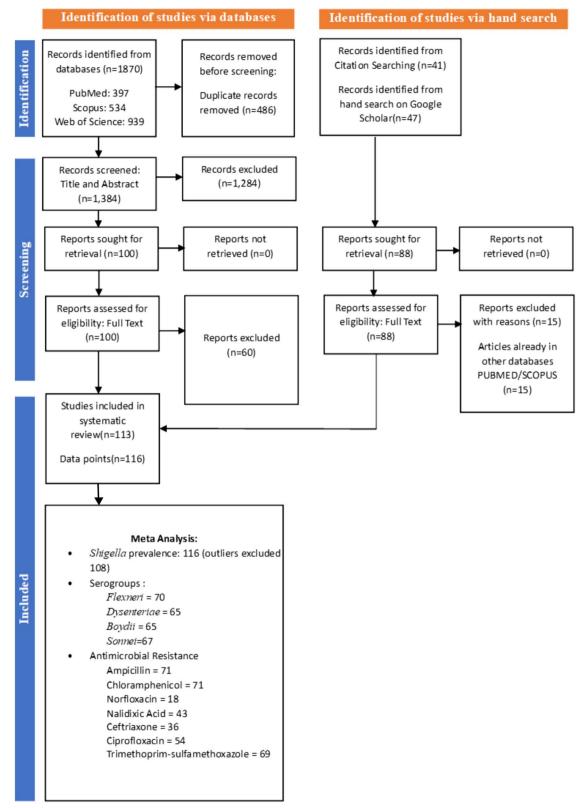


Fig. 1 Process of selecting published studies for the meta-analysis



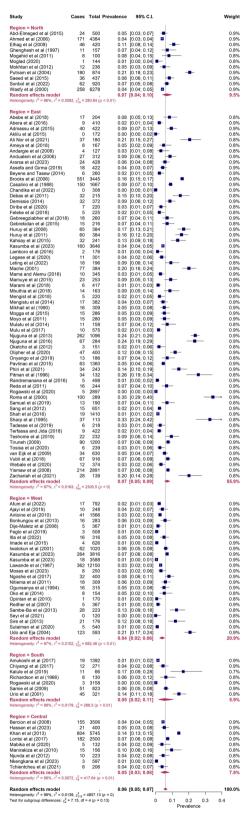


Fig. 2 Prevalence of Shigella spp. across Africa

Central Africa 4.5% (95% CI: 2.6 – 6.8%), and Western Africa 4.0 (95% CI: 2.5 – 5.9%). In terms of the different age groups, the prevalence of *Shigella* was highest in children of all ages [6.6% (95% CI: 3.2 – 11.1%)] followed by children <5 years [6.0% (95% CI: 3.2 – 11.1%)] all ages groups [5.7% (95% CI: 4.2 – 7.3%)] and adults [3.6% (95% CI: 1.6 – 6.3%)]. The prevalence in community and health facility-based studies were 3.7% (95% CI: 2.3 – 5.5%) and 6.2% (95% CI: 5.1 – 7.3%) respectively (Table S4). The between-study variability, expressed as the standard deviation (Tau), was 10.1% (95% CI: 8.8 – 11.8%). The 95% prediction interval, which provides an inference of a possible effect size for a future study conducted in similar settings was 0.0—18.3%.

Distribution of Shigella species

Seventy studies determined the prevalence of *S. flexneri*. These studies revealed a pooled prevalence of 53.6% (95% CI: 46.1 – 61.0%), with a significant *p* value of < 0.0001 (Fig. 3). Similarly, *S. dysenteriae* exhibited a pooled prevalence of 10.1% (95% CI: 6.2 - 14.5%) across 65 studies (Fig. 4). Furthermore, the pooled prevalence of *S. sonnei* reported from 67 studies was 11.5% (95% CI: 7.7%— 15.7%) (Fig. 5).The pooled prevalence of *S. boydii* was 7.7 (95% CI: 4.7 – 11.1%) across 65 studies (Fig. 6). We observed that in almost all the studies, *S. flexneri* was the predominant species isolated, except for a few studies that reported *S. sonnei* as the predominant species [29–36].

Antimicrobial resistance of Shigella spp.

The prevalence of antibiotic resistance among *Shigella* spp. in this study varied across different antibiotics, with Ampicillin exhibiting the highest resistance prevalence of 77.8% (95% CI: 71.7 – 83.4%) (Table S3). Trimethoprimsulfamethoxazole and Chloramphenicol also showed high resistance of 65.1% (95% CI: 56.1 – 73.4%) and 45.2% (95% CI: 38.2 – 52.2%) respectively. On the other hand, other antibiotics that showed low prevalences were ceftriaxone 8.5% (95% CI: 2.4 – 16.9%), ciprofloxacin 10.0% (95% CI: 4.5%—16.9%), norfloxacin 12.7% (95% CI: 3.6— 25.1%), and nalidixic acid 16.5% (95% CI: 9.5 – 24.6%).

Risk of Bias

The included articles showed high heterogeneity indicated by the I² test value of 97.6% and Cochrane Q test (Q=4857.1, p-value < 0.0001). Although a visual inspection of the funnel plot shows a slight asymmetrical distribution, the intercept of the Eggers regression model was 1.7527(95% CI: - 0.3233 - 3.8297) with a t statistic of 1.67 and a p-value of 0.0971. This suggests that potential publication bias in the included studies is unlikely (Fig. 7).

Study	Cases Tota	I Prevalence	95% C.I.	Weight
Region = North Abd-Elmeged et al (2015) Ahmed et al (2009) Elhag et al (2009) Ghenghesh et al (1997) Mogahid et al (2011) Mokhtari et al (2012) Putnam et al (2015) Saeed et al (2015) Sonbol et al (2022) Wasfy et al (2000) Random effects model Heterogeneity: $l^2 = 76\%$, $l^2 = 0.000$	$\begin{array}{cccccccccccccccccccccccccccccccccccc$	1 0.60 5 0.28 1 0.55 3 0.00 2 0.33 0 0.57 5 0.56 2 0.61 3 0.48 . 0.48	[0.42; 0.81] [0.53; 0.68] [0.15; 0.42] [0.00; 0.21] [0.00; 0.21] [0.00; 0.21] [0.39; 0.71] [0.42; 0.54] [0.42; 0.54] [0.37; 0.59]	> 1.5% > 1.7% → 1.6% → 1.3% 1.2% → 1.4% > 1.4% > 1.7% → 1.6% > 1.6% > 1.6% > 1.6%
Region = East Admassu et al (2015) Brooks et al (2006) Demissie (2014) Diriba et al (2020) Kasumba et al (2023) Leting et al (2022) Mbuthia et al (2014) Mikhail et al (2014) Mikhail et al (2014) Moyo et al (2011) Mulatu et al (2011) Mulatu et al (2011) Mulatu et al (2011) Paviinac et al (2015) Phiri et al (2021) Pawinac et al (2015) Phiri et al (2020) Randremanan et al (2016) Samuel et al (2019) Shah et al (2019) Torsias et al (2019) Torsias et al (2020) Randoremandel (1995) Teshome et al (2019) Torsias et al (2020) Randore flocts model Heterogeneity: I ² = 93%, z ² = 0.1	2 5 99 100 0 13 11 19 23 33 8 23 65 90 3 6 7 13	1 0.54 2 0.34 7 0.57 0 0.60 3 0.39 4 0.00 5 0.33 5 0.67 5 0.33 6 0.67 1 1.00 2 0.63 7 0.60 8 0.33 4 0.108 5 0.33 4 0.100 5 0.33 4 0.100 5 0.33 4 0.100 5 0.33 0 0.99 8 0.00 9 0.58 9 0.58 0 0.72 5 0.50 2 0.58 0 0.58	[0.29; 0.61] [0.50; 0.58] [0.14; 0.52] [0.52; 0.68] [0.10; 0.92] [0.11; 0.66] [0.00; 0.11] [0.11; 0.66] [0.30; 0.51] [0.40; 0.58] [0.40; 0.58] [0.40; 0.58] [0.40; 0.59] [0.40; 0.59] [0.40; 0.71] [0.00; 0.50] [0.23; 0.46] [0.23; 0.46] [0.00; 0.34] [0.00; 0.34] [0.00; 0.34] [0.00; 0.34] [0.00; 0.34] [0.00; 0.34] [0.00; 0.34] [0.00; 0.34] [0.46; 0.78] [0.46; 0.78] [0.46; 0.58] [0.46; 0.63]	$ \begin{array}{c} & & 1.6\% \\ & & & 1.7\% \\ & & & 1.6\% \\ & & & 1.2\% \\ & & & 1.2\% \\ & & & 1.2\% \\ & & & 1.4\% \\ & & & 1.4\% \\ & & & 1.4\% \\ & & & 1.4\% \\ & & & 1.4\% \\ & & & 1.4\% \\ & & & 1.4\% \\ & & & 1.1\% \\ & & & 1.7\% \\ & & & 0.9\% \\ & & & 0.9\% \\ & & & 0.9\% \\ & & & 1.6\% \\ & & & 1.6\% \\ & & & 1.6\% \\ & & & 1.6\% \\ & & & 1.6\% \\ & & & 1.6\% \\ & & & 1.5\% \\ & & & 1.7\% \\ & & & 1.1\% \\ & & & & 1.7\% \\ & & & 1.1\% \\ & & & & 1.5\% \\ & & & & 1.5\% \\ & & & & 1.5\% \\ & & & & & 1.5\% \\ & & & & & 1.5\% \\ & & & & & & 1.5\% \\ & & & & & & & 1.1\% \\ & & & & & & & & & \\ & & & & & & & & $
Region = West Afum et al (2022) Ajayi et al (2019) Antoine et al (2010) Bonkungou et al (2013) Dije-Maletz et al (2008) Feglo et al (2019) Illa et al (2022) Imade et al (2015) Iwalokun et al (2023) Kasumba et al (2023) Lawande et al (2023) Noses et al (2023) Nososhe et al (2017) Nitiema et al (2011) Ogunsanya et al (1994) Oko et al (2014) Opintan et al (2013) Sire et al (2013) Sire et al (2013) Sire et al (2013) Sire et al (2013) Judo and Eja (2004) Random effects model Heterogeneity: I ² = 56%, c ² = 0.0000000000000000000000000000000000	$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	0 0.50 1 0.54 5 0.56 5 0.40 6 0.56 5 0.40 4 1.00 2 0.52 8 0.67 4 0.64 2 0.52 2 0.42 4 0.61 8 0.25 2 0.47 3 0.33 5 0.31 5 0.41 1.00 1.00 5 0.40 5 0.40 5 0.40 6 0.79 1 0.76 3 0.58 0.58 0.60		$ \begin{array}{c} > 1.4\% \\ \rightarrow 1.3\% \\ \rightarrow 1.6\% \\ \rightarrow 1.4\% \\ \hline \qquad 1.1\% \\ > 0.9\% \\ \rightarrow 1.4\% \\ > 1.0\% \\ \rightarrow 1.6\% \\ > 1.0\% \\ \rightarrow 1.6\% \\ > 1.5\% \\ > 1.7\% \\ \rightarrow 1.2\% \\ \hline \qquad 1.5\% \\ \rightarrow 1.2\% \\ \hline \qquad 1.5\% \\ \rightarrow 1.5\% \\ \hline \qquad 1.5\% \\ \hline \qquad 1.5\% \\ \rightarrow 1.5\% \\ \hline \qquad 1.5\% \\ \hline \hline \qquad 1.5\% \\ \hline \qquad 1.5\% \\ \hline \hline \hline \hline \qquad 1.5\% \\ \hline $
Region = South Amukoshi et al (2017) Chiyangi et al (2017) Kalule et al (2019) Rogawski et al (2020) Urio et al (2001) Random effects model Heterogeneity: I ² = 89%, c ² = 0.1	$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	2 0.50 1 1.00 3 0.11 5 0.27 . 0.60	[0.57; 0.94] [0.22; 0.78] [0.83; 1.00] [0.00; 0.70] [0.15; 0.41] [0.23; 0.92]	→ 1.5% → 1.4% → 1.3% → 0.9% → 1.6%
Region = Central Bercion et al (2008) Khan et al (2013) Lontsi et al (2017) Manirakiza et al (2010) Nkengkana et al (2023) Random effects model Heterogeneity: I ² = 99%, t ² = 0.1	79 155 504 804 7 182 15 15 2 3 950, χ^2_4 = 326.73	4 0.63 2 0.04 5 1.00 3 0.67 . 0.56	[0.42; 0.60] [0.59; 0.66] [0.01; 0.07] [0.87; 1.00] [0.12; 1.00] [0.16; 0.93]	> 1.7% > 1.7% 1.7% > 1.4%
Random effects model Heterogeneity: $l^2 = 92\%$, $\tau^2 = 0.0$ Test for subgroup differences: χ_4^2	714, χ^2_{69} = 841.6 = 3.42, df = 4 (p	7 (p < 0.01)	[0.46; 0.61]	100.0% 0.1 0.2 0.3 0.4
across Africa				Prevalence

Fig. 3 Prevalence of S. flexneri across Africa

Study	Cases Total P	evalence 959	% C.I.	Weight
Region = North Abd-Elmeged et al (2015) Ahmed et al (2006) Elhag et al (2009) Ghenghesh et al (1997) Mogahid et al (2011) Mokhari et al (2012) Putnam et al (2004) Saeed et al (2015) Sonbol et al (2020) Wasfy et al (2000) Random effects model Heterogeneity: $l^2 = 88\%$, $r^2 = 0.040$	$\begin{array}{cccccccccccccccccccccccccccccccccccc$	0.12 [0.03; 0.09 [0.05; 0.50 [0.34; 0.00 [0.02; 0.38 [0.09; 0.00 [0.00; 0.17 [0.11; 0.11 [0.04; 0.00 [0.00; 0.18 [0.14; 0.12 [0.04;	0.14j 0.66j 0.30j 0.75j 0.27j 0.22j 0.27j 0.04j 	 − 1.6% 1.9% → 1.8% 1.3% 1.2% 1.4% 1.9% 1.7% 1.8% 2.0% 16.8%
$\label{eq:response} \begin{array}{l} \textbf{Region = East} \\ Admassu et al (2015) \\ Brooks et al (2006) \\ Demissie (2014) \\ Diriba et al (2020) \\ Kasumba et al (2023) Kenya \\ Leting et al (2022) \\ Mouthia et al (2018) \\ Mengistu et al (2014) \\ Mikhail et al (2014) \\ Mikhail et al (2015) \\ Moyo et al (2011) \\ Niuguna et al (2015) \\ Oketcho et al (2016) \\ Oketcho et al (2015) \\ Phirri et al (2021) \\ Phirman et al (2015) \\ Phirri et al (2015) \\ Randremanana et al (2016) \\ Rama et al (2016) \\ Samuel et al (2016) \\ Shah et al (2019) \\ Shah et al (2019) \\ Shah et al (2019) \\ Teshome et al (2019) \\ Tiruneh (2009) \\ Tosisa et al (2020) \\ Randormente al (2000) \\ Randorm$	$\begin{array}{cccccccccccccccccccccccccccccccccccc$	0.30 [0.15; 0.28 [0.24; 0.09 [0.00; 0.00 [0.00; 0.04 [0.01; 0.22 [0.01; 0.22 [0.01; 0.23 [0.16]; 0.18 [0.04; 0.12 [0.00; 0.67 [0.39; 0.33 [0.11; 0.10 [0.07; 0.16 [0.09; 1.00 [0.04; 0.00 [0.01; 0.74 [0.56; 0.00 [0.04; 0.00 [0.04; 0.05 [0.00; 0.23 [0.05; 0.05 [0.00; 0.25 [0.23; 0.10 [0.03; 0.00 [0.01; 0.75 [0.08; 0.15 [0.08;	0.32] 0.21] 0.41] 0.47] 0.37] 0.37] 0.37] 0.52] 0.15] 0.55] 0.55] 0.12] 0.52] 0.52] 0.54] 0.52] 0.688] 0.52] 0.54] 0.55] 0.68] 0.54] 0.55] 0.54] 0.54] 0.55] 0.55] 0.54] 0.55] 0.55] 0.55] 0.54] 0.54] 0.55] 0.56] 0.56] 0.58] 0.58] 0.58] 0.58] 0.58] 0.58] 0.58] 0.58] 0.58] 0.58] 0.58] 0.58] 0.58] 0.58] 0.58] 0.58]	$\begin{array}{c} & & 1.8\% \\ & & 2.0\% \\ & & 1.7\% \\ & & 1.1\% \\ & & 1.9\% \\ & & 1.5\% \\ & & 1.5\% \\ & & 1.5\% \\ & & 1.5\% \\ & & & 1.5\% \\ & & & 1.5\% \\ & & & 1.5\% \\ & & & 1.5\% \\ & & & 1.5\% \\ & & & & 1.5\% \\ & & & & 1.5\% \\ & & & & 1.5\% \\ & & & & 1.5\% \\ & & & & & 1.5\% \\ & & & & & 1.5\% \\ & & & & & & 1.5\% \\ & & & & & & & 1.5\% \\ & & & & & & & & \\ & & & & & & & & \\ & & & & & & & & \\ & & & & & & & & \\ & & & & & & & & \\ & & & & & & & & \\ & & & & & & & & \\ & & & & & & & & \\ & & & & & & & & \\ & & & & & & & & \\ & & & & & & & & \\ & & & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & \\ & & & & & & \\ & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & & \\ & & & & & & & \\$
Region = West Afum et al (2022) Ajayi et al (2019) Antoine et al (2010) Bonkungou et al (2013) Dije-Maletz et al (2008) Fegio et al (2019) Illa et al (2022) Imade et al (2015) Iwalokun et al (2023) Kasumba et al (2023) Moses et al (2023) Moses et al (2023) Nogshe et al (2017) Nitiema et al (2017) Nitiema et al (2010) Reither et al (2013) Udo and Eja (2004) Random effects model Heterogeneity: I ² = 82%, r ² = 0.033	$ \begin{array}{cccc} 1 & 18 \\ 52 & 362 \\ 6 & 8 \\ 12 & 32 \\ 0 & 18 \\ 2 & 16 \\ 0 & 1 \\ 0 & 5 \\ 0 & 21 \\ 0 & 123 \\ \end{array} $	0.12 [0.01; 0.00 [0.01; 0.00 [0.01; 0.00 [0.01; 0.00 [0.00; 0.00 [0.00; 0.00 [0.00; 0.04 [0.02; 0.66 [0.01; 0.75 [0.36; 0.38 [0.17; 0.00 [0.00; 0.12 [0.03; 0.00 [0.14; 0.00 [0.02; 0.00 [0.00; 0.04 [0.00; 0.04 [0.00; 0.04 [0.00; 0.01]	0.32] 0.12] 0.22] 0.22] 0.52] 0.21] 0.21] 0.57] 0.16] 0.28] 0.57] 0.18] 0.57] 0.15] 0.39] 0.57] 0.	1.5% 1.3% 1.8% 1.5% 0.8% 1.5% 2.0% 1.5% 2.0% 1.5% 2.0% 1.5% 2.0% 1.5% 2.0% 1.5% 2.0% 1.5% 2.0% 1.5% 1.5% 1.5% 1.5% 1.5% 1.5% 2.0% 2.0% 2.0% 2.0% 2.0% 2.0% 2.0% 2.0% 2.0% 2.0% 2.0% 2.0% 2.1.2% 2.0% 2.1.2% 2.1.2% 2.1.2% 2.1.2% 2.1.2% 2.1.2% 2.2% 2.2% 2.2% 2.2% 2.2% 2.2% 2.2%
Region = South Amukoshi et al (2017) Chiyangi et al (2017) Kalule et al (2019) Urio et al (2001) Random effects model Heterogeneity: $l^2 = 79\%$, $\tau^2 = 0.046$	$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	0.11 [0.01; 0.33 [0.12; 0.00 [0.01; 0.00 [0.00; 0.06 [0.00;	0.64] 0.29] • 0.08] •	 − − 1.6% 1.4% 1.3% 1.8% 6.1%
Region = CentralBercion et al (2008)Khan et al (2013)Lontsi et al (2017)Mabika et al (2020)Nkengkana et al (2023)Random effects modelHeterogeneity: $l^2 = 94\%$, $\tau^2 = 0.032$	47 155 181 804 6 182 0 5 0 3 3, $\chi_4^2 = 68.83 (p < 0.0$	0.30 [0.19; 0.23 [0.19; 0.03 [0.01; 0.00 [0.04; 0.00 [0.04; 0.11 [0.01; 1)	0.25] — — — — — — — — — — — — — — — — — — —	1.9% 2.0% 1.9% → 1.0% 0.8% 7.6%
Random effects model Heterogeneity: $l^2 = 89\%$, $\tau^2 = 0.044$ Test for subgroup differences: $\chi_4^2 = -$	4, χ ₆₄ ² = 594.72 (ρ < 0 4.25, df = 4 (ρ = 0.37	0.10 [0.06;	0.14] 0 0.1 0.2 0.1 Prevalence	100.0% 3 0.4

Fig. 4 Prevalence of S. dysenteriae across Africa

Study	Cases Tot	al Prevalence	95% C.I.	Weight
Region = North				
Abd-Elmeged et al (2015)			[0.00; 0.07]	1.6%
Ahmed et al (2006)	35 17		[0.14; 0.26]	2.0%
Elhag et al (2009) Ghenghesh et al (1997)		6 0.00 1 0.45		1.8% → 1.3%
Mogahid et al (2011)	5	8 0.62		→ 1.1%
Mokhtari et al (2012)		2 0.50		→ 1.3%
Putnam et al (2004)	42 18		[0.17; 0.29]	- 2.0%
Saeed et al (2015)			[0.19; 0.50]	→ 1.7%
Sonbol et al (2022)	20 6		[0.20; 0.44]	→ 1.9%
Wasfy et al (2000)	49 25	i8 0.19	[0.14; 0.24] —	2.0%
Random effects model Heterogeneity: $l^2 = 86\%$, $\tau^2 = 0.0$	$1561 \ \gamma^2 = 66.54$		[0.10; 0.38]	16.7%
	, xg	(+)		
Region = East	10		F0 44 0 001	1.00/
Admassu et al (2015)			[0.11; 0.39]	1.8% 2.0%
Brooks et al (2006) Demissie (2014)	41 5	0.07 0.03		2.0%
Diriba et al (2020)	1	7 0.14		→ 1.1%
Kasumba et al (2023)	41 10			
Leting et al (2022)		8 0.17		1.5%
Mbuthia et al (2018)	6	4 0.43	[0.17; 0.70]	→ 1.4%
Mengistu et al (2014)		7 0.35		→ 1.5%
Mikhail et al (1990)		6 0.00		1.5%
Mogga et al (2015)		5 0.00		1.4%
Moyo et al (2011) Njuguna et al (2013)	0 · 24 26	5 0.00 2 0.09		1.4% 2.0%
Njuguna et al (2013) Njuguna et al (2016)				2.0%
Oketcho et al (2012)	0		[0.00; 0.50]	→ 0.7%
Pavlinac et al (2015)			[0.33; 0.57]	→ 1.9%
Phiri et al (2021)			[0.00; 0.06]	1.7%
Pitman et al (1996)			[0.00; 0.11]	1.7%
Randremanana et al (2016		5 0.60	[0.19; 0.92]	→ 0.9%
Rogawski et al (2020)	0	3 0.07		→ 0.7%
Roma et al (2000)	0 10			1.9%
Samuel et al (2019)		3 0.77 9 0.26		> 1.4% → 1.5%
Shah et al (2016) Sharp et al (1995)		5 0.20 7 0.32	[0.08; 0.49]	→ 1.7%
Teshome et al (2019)		2 0.00		1.6%
Tiruneh (2009)		0.09		1.9%
Tosisa et al (2020)	1	6 0.17	[0.00; 0.58]	→ 1.0%
Webale et al (2020)	2 1		[0.01; 0.44]	→ 1.3%
Random effects model	2 004	. 0.13	[0.06; 0.20]	41.1%
Heterogeneity: $I^2 = 87\%$, $\tau^2 = 0.0$	1499, χ ₂₆ = 204.	+6 (<i>p</i> < 0.01)		
Region = West				
Afum et al (2022)			[0.01; 0.33]	1.5%
Ajayi et al (2019)		0 0.20		→ 1.2%
Antoine et al (2010)		1 0.27		→ 1.8%
Bonkungou et al (2013)	2 · 0		[0.02; 0.35]	
Dije-Maletz et al (2008) Feglo et al (2019)	0		[0.00; 0.30] • [0.00; 0.50] •	→ 0.7%
Illa et al (2022)		6 0.12		- 1.5%
Imade et al (2015)	0	4 0.00		0.8%
lwalokun et al (2001)		2 0.13	[0.06; 0.23]	1.9%
Kasumba et al (2023)		8 0.22		→ 1.5%
Kasumba et al (2023)	34 26		[0.08; 0.16]	2.0%
Lawande et al (1987) Moses et al (2023)	19 36		[0.03; 0.08]	2.0%
Ngoshe et al (2023)	0 5 3		[0.00; 0.19]	1.1% 1.7%
Nitiema et al (2011)		8 0.06		1.5%
Ogunsanya et al (1994)		6 0.06		1.5%
Opintan et al (2010)	0	1 0.00		→ 0.4%
Reither et al (2007)	0	5 0.00		0.9%
Sire et al (2013)			[0.04; 0.39]	1.6%
Udo and Eja (2004)	52 12		[0.33; 0.51]	→ 1.9%
Random effects model Heterogeneity: $l^2 = 80\%$, $\tau^2 = 0.0$	143, $\chi^2_{10} = 96.1$. U.11 (p < 0.01)	[0.06; 0.17]	27.8%
Region = South	~		10.04.0.001	
Amukoshi et al (2017)			[0.01; 0.30]	- 1.5%
Chiyangi et al (2017) Kalule et al (2019)			[0.00; 0.15] • [0.00; 0.17] •	1.3% 1.3%
Rogawski et al (2020)	0		[0.00; 0.52]	→ 0.9%
Urio et al (2001)			[0.02; 0.20]	1.8%
Random effects model		. 0.05	[0.01; 0.11]	6.8%
Heterogeneity: $I^2 = 0\%$, $\tau^2 = 0$, χ^2	$p_4^2 = 2.78 \ (p = 0.0)$	50)		
Region = Central				
Bercion et al (2008)	11 15	5 0.07	[0.00; 0.07] ——	2.0%
Khan et al (2013)	30 80		[0.02; 0.05]	2.0%
Lontsi et al (2017)	0 18	2 0.00	[0.00; 0.00] 🖪	2.0%
Mabika et al (2020)	2		[0.08; 0.81]	0.9%
Nkengkana et al (2023)	0		[0.00; 0.50]	→ 0.7%
Random effects model Heterogeneity: $l^2 = 85\%$, $\tau^2 = 0.0$	$(237, \gamma_{1}^{2} = 27.3)$. 0.01	[0.00; 0.11]	7.6%
	201, 14 - 21.02			
Random effects model	2	. 0.11	[0.08; 0.16]	100.0%
Heterogeneity: $I^2 = 88\%$, $\tau^2 = 0.0$ Test for subgroup differences: χ_4^2	$\chi_{66}^2 = 567.$	14 (p < 0.01) p = 0.05)	0 0.1 0.2	0.3 0.4
		/	Prevalence	
cross Africa				

Fig. 5 Prevalence of S. sonnei across Africa

Study	Cases To	al Prevalence	95% C.I.	Weight
Region = North Abd-Elmeged et al (2015)			[0.10; 0.47]	■ 1.6%
Ahmed et al (2006) Elhag et al (2009)			[0.04; 0.13] — — [0.10; 0.36] — —	2.1% 1.9%
Ghenghesh et al (1997)			[0.02; 0.30]	1.3%
Mogahid et al (2011)	0	8 0.00	[0.00; 0.37]	1.1%
Mokhtari et al (2012)			[0.03; 0.48]	→ 1.3%
Putnam et al (2004) Saeed et al (2015)			[0.00; 0.06] -	2.1% 1.8%
Sonbol et al (2022)			[0.00; 0.14]	1.9%
Wasfy et al (2000)	38 2		[0.11; 0.20]	- 2.1%
Random effects model Heterogeneity: I^2 = 79%, τ^2 = 0.0	158, χ ₉ ² = 42.7		[0.03; 0.14]	17.2%
Region = East				
Admassu et al (2015)			[0.00; 0.07] •	1.8% 2.2%
Brooks et al (2006) Demissie (2014)			[0.00; 0.17]	1.7%
Diriba et al (2020)	2		[0.04; 0.71]	→ 1.0%
Kasumba et al (2023)			[0.04; 0.14]	2.1%
Leting et al (2022)			[0.01; 0.47]	$\xrightarrow{\bullet} 1.5\%$ $\longrightarrow 1.4\%$
Mbuthia et al (2018) Mengistu et al (2014)			[0.29, 0.83]	→ 1.4% → 1.5%
Mikhail et al (1990)			[0.00; 0.37]	1.4%
Mogga et al (2015)			[0.01; 0.23]	1.4%
Moyo et al (2011) Niuguna et al (2013)			[0.00; 0.21]	
Njuguna et al (2013) Njuguna et al (2016)			[0.01; 0.07] —	2.1% — 1.9%
Oketcho et al (2012)	õ		[0.07; 0.66]	→ 0.6%
Pavlinac et al (2015)		63 0.02	[0.00; 0.09] 🔳	1.9%
Phiri et al (2021) Pitman et al (1996)			[0.01; 0.11]	1.8% 1.8%
Pitman et al (1996) Randremanana et al (2016)			[0.00; 0.09] — —— [0.11; 0.80] — ——	■ 0.9%
Roma et al (2000)			[0.00; 0.00]	2.0%
Samuel et al (2019)			[0.00; 0.25]	1.3%
Shah et al (2016)			[0.01; 0.33]	1.5%
Sharp et al (1995) Teshome et al (2019)			[0.13; 0.44]	■ 1.8% ■ 1.6%
Tiruneh (2009)			[0.02; 0.15]	2.0%
Tosisa et al (2020)	2		[0.07; 0.76]	■ 1.0%
Webale et al (2020)	2		[0.01; 0.48]	→ 1.3% 41.1%
Random effects model Heterogeneity: $I^2 = 76\%$, $\tau^2 = 0.0$	269, χ ₂₅ = 105		[0.03; 0.12]	41.170
Region = West Afum et al (2022)	1	17 0.06	[0.00; 0.29]	1.5%
Ajayi et al (2019)			[0.09; 0.64]	→ 1.2%
Antoine et al (2010)		41 0.17	[0.06; 0.32]	1.8%
Bonkungou et al (2013)			[0.13; 0.58]	■ 1.4%
Dije-Maletz et al (2008) Feglo et al (2019)	2 0		[0.11; 0.81] [0.00; 0.71] •	■ 0.9% → 0.6%
Illa et al (2022)	2		[0.02; 0.38]	1.4%
Imade et al (2015)	0		[0.00; 0.61]	→ 0.8%
Iwalokun et al (2001)			[0.00; 0.16]	1.9%
Kasumba et al (2023) Kasumba et al (2023)			[0.01; 0.28] I	- 1.5% - 2.1%
Lawande et al (1987)		62 0.21		2.1%
Moses et al (2023)	0		[0.03; 0.38]	1.1%
Ngoshe et al (2017) Nitiema et al (2011)			[0.00; 0.06] • [0.00; 0.25]	1.7% 1.5%
Ogunsanya et al (1994)			[0.06; 0.46]	■ 1.4%
Opintan et al (2010)	0	1 0.00	[0.14; 0.82] • —	→ 0.3%
Reither et al (2007)	3		[0.18; 0.91]	→ 0.9%
Sire et al (2013) Udo and Eja (2004)			[0.00; 0.24] –	
Random effects model Heterogeneity: $l^2 = 81\%$, $\tau^2 = 0.0$. 0.08	[0.03; 0.15]	28.0%
Region = South		(
Amukoshi et al (2017)	0	19 0.00	[0.00; 0.18]	1.5%
Chiyangi et al (2017)	2	12 0.17	[0.04; 0.49]	→ 1.3%
Kalule et al (2019)			[0.01; 0.29]	1.3%
Urio et al (2001) Random effects model	29		[0.49; 0.78] [0.00; 0.52]	> 1.8%
Heterogeneity: $I^2 = 94\%$, $\tau^2 = 0.1$	385, χ ₃ ² = 48.6			
Region = Central Bergion et al (2008)	18 1	55 0.12	[0.02; 0.13]	2.1%
Bercion et al (2008) Khan et al (2013)			[0.07; 0.12]	2.1%
Lontsi et al (2017)		82 0.00	[0.00; 0.01]	2.1%
Mabika et al (2020)	0	5 0.00	[0.04; 0.52]	→ 0.9%
Nkengkana et al (2023) Random effects model	1		[0.06; 0.85]	■ 0.6% 7.8%
Random effects model Heterogeneity: $I^2 = 92\%$, $\tau^2 = 0.0$	251, χ ₄ ² = 52.3	. υ.υ.3 δ (p < 0.01)	[0.00; 0.14]	7.8%
Random effects model			[0.05; 0.11]	100.0%
Heterogeneity: $I^2 = 84\%$, $\tau^2 = 0.0$	295, $\chi^2_{64} = 404$.49 (p < 0.01)	1 1	
Test for subgroup differences: χ_4^2	= 1.80, df = 4	(p = 0.77)	0 0.1 Pre	0.2 0.3 0.4 valence
cross Africa				

Fig. 6 Prevalence of S. boydii across Africa

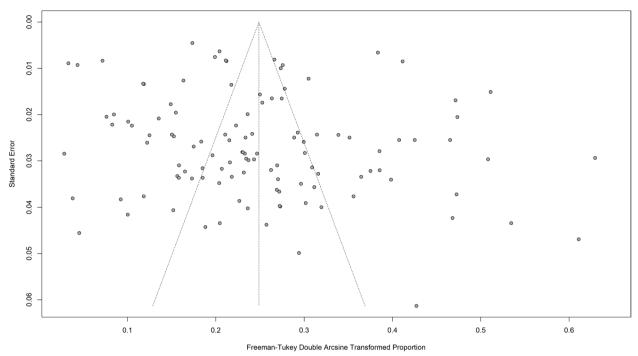


Fig. 7 Funnel plot of publication bias. The graph displays the standard error of the estimate (prevalence) on the Y-axis, while the X-axis represents the transformed proportions (prevalence) with individual studies represented by dots. The 95% confidence interval is indicated by dashed lines on the graph

Discussion

This meta-analysis focused on determining the overall prevalence of Shigella spp., their serogroups and antimicrobial resistance across sub-Saharan Africa. The pooled prevalence of Shigella spp. among 116 data points included was 5.9% (95% CI: 4.9 - 7.0%). Systematic reviews in South Asia and Southeast Asia have reported similar prevalences of 7% (95% CI: 6-7%) and 4% (95% CI: 4–5%) respectively[37, 38]. Other systematic reviews in some parts of Africa have also reported similar prevalences of 6.2% (95% CI-0.20-12.60) in East Africa, and 6.6% (95% CI 4.7-8.8) in Ethiopia. Our systematic review covered a larger region and included a greater number of studies (n=113) compared to South Asia (n=7) and Southeast Asia (n=21), East Africa (n=22) and Ethiopia (n=27), consequently offering a more comprehensive and precise understanding of the prevalence of Shigella spp. Although variations in the number of studies and study designs could contribute to differences in Shigella prevalence, other factors like seasonal variations might also be important. For instance, certain studies have found a correlation between shigellosis and factors like rainfall and temperature, indicating a higher incidence of shigellosis during periods of above-average rainfall [39]. However, it is important to note that other studies have not observed such associations [40, 41].

The estimated prevalence of 5.9% for Shigellosis in the African subregion is a cause for concern, as it indicates a significant risk of exposure to Shigella spp. among the population. This high prevalence is particularly alarming considering the severe nature of the disease and its potential for rapid transmission. The primary mode of Shigellosis transmission is through the faecal-oral route, often due to inadequate sanitation and the consumption of contaminated food or water. It is important to note that as few as 10 colony-forming units (CFU) of S. dysenteriae or 180 CFU of S. flexneri and S. sonnei can lead to the contraction of the disease [42, 43]. This makes shigellosis particularly prevalent in densely populated areas with poor sanitation and personal hygiene practices. Given the escalating tensions from conflicts in certain parts of Africa, the prevalence of shigellosis becomes even more worrisome. The movement of troops and the possibility of overcrowding in military camps or refugee settlements could further elevate the risk of Shigella spp. transmission, resulting in outbreaks and the spread of the disease to a larger population. It is worth noting that among deployed US troops in Africa, the reported prevalence of Shigella was 6.6% [44]. Another study reported the potential of using S. dysenteriae as a bioterrorism agent [45]. The seriousness of S. dysenteriae type 1 is a result of its ability to produce Shiga toxin, leading to

severe and life-threatening dysentery with high rates of illness and death, particularly in areas with limited access to healthcare[46]. Additionally *S. dysenteriae* could be potentially genetically engineered to increase its virulence and ease of spread diagnosing and treating it is also challenging [46].

Other factors, such as age, are strongly associated with the incidence of *Shigella* spp. According to our review, the prevalence of Shigellosis was higher in children compared to adults. According to the Global Enteric Multicentre Study (GEMS), Shigella ranks as the second most prevalent pathogen causing medically attended moderate-to-severe diarrhoea (MSD) among children aged 12-23 months across 7 sites in sub-Saharan Africa and South Asia. Additionally, Shigella emerged as the primary pathogen among children aged 24-59 months in the same regions [39]. We also observed that the prevalence in health facility-based studies was twice as much as the prevalence in community-based studies. The lower prevalence observed in community-based studies suggests a relatively lower presence of shigellosis within the general population outside healthcare settings. In contrast, the higher prevalence in health facility-based studies indicates a greater burden of shigellosis among individuals seeking healthcare services. This could be due to more severe cases or specific healthcare-seeking behaviours associated with shigellosis. Patient seeking healthcare are assumed to reflect the relative importance of prevalent illnesses within a region[47].

In our study, the most dominant species was S. flexneri, accounting for 53% (95% CI: 46.1-61.0%) of the cases. This finding agrees with other studies, which have reported that during outbreaks of diarrheal disease, S. *flexneri* is the dominant cause of shigellosis in developing countries, responsible for 44.5% to 80% of the cases [20, 48-50]. The second most prevalent species was S. sonnei [11.5% (95% CI: 7.7 – 15.7%)], followed by S. dysenteriae [10.1% (95% CI: 6.2—14.5%)] and the least prevalent was S. boydii [7.7% (95% CI: 4.7-11.1%)]. Another review conducted between 1985 and 2005 in low- and middleincome countries, reported S. sonnei as the second most prevalent species, accounting for 25% of shigellosis cases following S. flexneri [51]. Similarly, in another study, S. flexneri accounted for 62% of shigellosis cases, while S. sonnei accounted for 25% of the cases in Asia and sub-Saharan Africa [52]. However, S. sonnei is more prevalent in industrialized and developed countries [53]. Improved hygiene conditions and economic growth have led to an observed trend where S. flexneri is replacing S. sonnei as the predominant species in the incidence of shigellosis[54]. This shift has been documented in various regions, including China [55], Vietnam [56], Bangladesh [57] and India [58]. Interestingly, a positive correlation has been found between a country's gross domestic product (GDP) and the prevalence of *S. sonnei*, which is the primary causative agent of shigellosis [51]. Several mechanisms have been proposed to explain this correlation. For instance, it has been suggested that individuals living in resource-limited environments develop natural immunity to *S. sonnei* as a result of exposure to contaminated surface water containing *Pleisiomonas shigelloides* O17, which possesses an O antigen closely resembling that of *S. sonnei* [51].

Although this study did not examine the prevalence of serotypes in the various Shigella spp. serogroups, different studies have emphasized that serotypes 2a and 1b are the most endemic species of S. flexneri isolated in developing countries [7, 59]. The development of vaccine programs against Shigella has led to the development of an effective Shigella vaccine comprising at least S. flexneri 2a and S. sonnei. This activity is believed to provide 40-50% protection against *Shigella* [38]. Recent advancements in Shigella vaccine development have yielded several promising candidates. These include a quadrivalent vaccine covering key Shigella serotypes 2a, 3a, and 6 and S. sonnei, which has completed phase II trials in Kenyan infants, potentially offering broad protection worldwide [60, 61]. Another candidate, the 1790GAHB S. sonnei vaccine, which utilizes the GMMA platform, demonstrated efficacy, safety, and immunogenicity in phase 2b trials in adults [60]. Additionally, a synthetic glycan-based vaccine targeting S. flexneri 2a showed safety, immunogenicity, and protective efficacy against moderate to severe SF2a shigellosis in controlled human infection model studies [60, 62] These developments marked significant progress towards combating *Shigella* infections. Children, as a key group susceptible to *Shigella* infection, are a priority for the provision of Shigella vaccines. Other groups, such as HIV-infected individuals, charity workers, missionaries, business travellers, tourists and militants, are also a target group for vaccination because the nature of their work exposes them to possible Shigella infection, especially in very endemic regions.

As demonstrated in this review, AMR in Shigella strains is a growing concern as it affects the effectiveness of antibiotic treatment options. Considering the variations in antimicrobial resistance patterns across different regions, the choice of antibiotic treatment for shigellosis should be based on the specific AMR profiles observed in each region. The 2023 report on essential medicines by the WHO recommends that ciprofloxacin be the first choice for treating adults and children with dysentery, and azithromycin, cefixime, sulfamethoxazole+trimethoprim and ceftriaxone as second choices for children >41 weeks of corrected gestational age for ceftriaxone [63]. In line with this recommendation, there have been various studies conducted in sub-Saharan Africa that have highlighted susceptibility to ciprofloxacin, ceftriaxone, and azithromycin [31, 64-73]. Moreover, a study conducted in Nigeria, discouraged the use of ampicillin, tetracycline, cotrimoxazole, and streptomycin as first-line drugs but instead advocated the use of third-generation cephalosporins and fluoroquinolones [74]. Similarly in Zimbabwe, the use of ampicillin, cotrimoxazole, chloramphenicol, or tetracycline is not recommended for Shigella dysenteriae type 1 infections [75, 76] whereas azithromycin is the preferred treatment for shigellosis in Cameroon [77]. A high resistance prevalence has also been observed against ampicillin, chloramphenicol, and trimethoprim-sulfamethoxazole for Shigella isolates in Eritrea [78]. Our study revealed that the resistance prevalence for ciprofloxacin and ceftriaxone was 10.0% (95% CI: 4.5-16.9%) and 8.5% (95% CI: 2.4 - 16.9%) indicating that these antibiotics may retain their effectiveness in treating *Shigella* infections in Africa. This suggests that ciprofloxacin and ceftriaxone could be viable treatment options for Shigella-related illnesses in African regions, particularly in cases where other antibiotics demonstrate higher resistance prevalence.

Shigella spp. have developed various mechanisms to resist quinolone and fluoroquinolone antibiotics, posing a significant challenge in treating shigellosis. One such mechanism is the acquisition of chromosomal targetsite mutations [59, 79]. Recent studies have identified two novel mutations in the parE gene of Shigella isolates, located at codons 408 and 458 [80, 81]. These mutations were found in Shigella strains collected in India in 2013 and Jiangsu Province in China in 2016 [48, 82]. The presence of a mutation at codon 458 is thought to cause resistance to both ciprofloxacin and nalidixic acid. On the other hand, a single isolate with a mutation at codon 408 in parE is associated with resistance to nalidixic acid but remains sensitive to ciprofloxacin [81]. The presence of new mutations in the parE gene of S. flexneri isolates appears to be linked to higher minimum inhibitory concentration (MIC) values for ciprofloxacin and the development of resistance to fluoroquinolones [20]. Evidence suggests that mutations linked to resistance against fluoroquinolones in S. sonnei might also promote resistance to other antimicrobial agents^[83]. Whole-genome sequencing of contemporaneous ciprofloxacin-resistant S. sonnei revealed that a single clone that is widespread in South Asia may be driving the intercontinental surge of ciprofloxacin-resistant S. sonnei and has the ability to establish endemic transmission in new locations [84]. Ceftriaxone is recommended for the treatment of ciprofloxacin-resistant Shigella isolates [20]. It is nevertheless noteworthy that certain strains of Shigella spp. have developed resistance genes to cephalosporins, as confirmed in this study. Reports indicate that class C β -lactamases, referred to as AmpC-type enzymes, provide significant resistance to cephalosporins. These AmpC β -lactamases are encoded by genes found on both plasmids and chromosomes [20, 62]. These findings also emphasize the need to consider local antimicrobial patterns when choosing first- and second-line antibiotics for the treatment of shigellosis. Continuous surveillance of antimicrobial resistance patterns to guide appropriate treatment strategies is therefore of great importance.

Conclusion

The pooled prevalence of Shigella spp. in Africa was estimated to be 5.9%. The resistance pattern of Shigella spp. to antibiotics in this study poses a significant public health concern, as it restricts the available treatment options. The most commonly found species in Africa were S. flexneri and S. sonnei. The prevalence of S. sonnei suggests mild symptoms from infections which in turn impacts the use of antibiotics in treatments. The misuse of antibiotics in Africa has contributed to the escalation of resistance, emphasizing the necessity for alternative approaches to reduce reliance on antibiotics. Vaccine development is a prioritized area; however, progress has been impeded by the diverse range of *Shigella* serotypes and the complexity involved in developing effective vaccines. The substantial burden of Shigellosis in Africa emphasizes the urgent requirement for interventions and innovative solutions to address this issue.

Limitations

This current review was subject to some limitations that need to be addressed. First, the estimated prevalence of 5.9% may not be an accurate representation of the situation in Africa. This can be attributed to different factors. Almost all studies included in this analysis used stool culture for the isolation of Shigella. Stool culture, even though the standard for isolating Shigella spp., has been criticized for its lower sensitivity and lack of specificity, potentially leading to underestimation [85]. Furthermore, our systematic review had more health-based studies compared to communitybased studies. The limited community-based studies suggests that the overall estimation of Shigella prevalence reported may not accurately represent the entire African population. Conversely, the inclusion of more community studies could have revealed an even higher prevalence, considering the presence of asymptomatic Shigella spp. and individuals engaging in self-medication, which is common in Africa due to diarrhoea. Another limitation of our study is the distribution of the studies in Africa. Out of the 54 African countries, only 29 were represented in this meta-analysis, with a majority from East African countries. This introduces a potential bias and highlights a knowledge gap regarding the distribution of shigellosis across Africa. To address these limitations, it is recommended that more surveillance studies be conducted in African countries lacking epidemiological data to obtain comprehensive reporting on the burden of shigellosis. Despite these limitations, this study presents a recent assessment of the burden of shigellosis in Africa, offering updated information. Moreover, it contributes valuable insights into the distribution of *Shigella* species and the prevalence of antimicrobial resistance among *Shigella* strains in Africa.

Recommendation

There is the need to explore alternative treatments for shigellosis with particular focus on vaccine development. There is also the need for more genomic epidemiology studies exploring the dissemination and risk of drug-resistant *S. sonnei* clones in Africa.

Supplementary Information

The online version contains supplementary material available at https://doi. org/10.1186/s12879-024-09945-2.

Supplementary Material 1. Table S1 JBI critical appraisal checklist for studies reporting prevalence data. Key: Y = Yes; N = No; NR = Not reported. Table S2 Characteristics of included studies. Table S3 Antimicrobial resistance pattern of Shigella across Africa. Table S4 Subgroup Analysis of Shigella species prevalence across Africa

Clinical trial number

Not applicable.

Consent to publish declaration

All authors have agreed to the publication of the paper.

Authors' contributions

R.N.: Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Validation, Visualization, Writing - original draft, Writing - review editing. A.O.: Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Validation, Visualization, Writing - original draft, Writing - review editing. E.S.D.: Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Visualization, Writing - original draft, Writing - review editing.

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Availability of data and materials

Data is provided within the manuscript or supplementary information

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

All participants (or their legal guardians) involved in this study have provided written informed consent for the publication of the data and images contained in this manuscript.

Competing interests

All authors declare no competing interests.

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References

- Kirk MD, Pires SM, Black RE, Caipo M, Crump JA, Devleesschauwer B, et al. World Health Organization estimates of the global and regional disease burden of 22 foodborne bacterial, protozoal, and viral diseases, 2010: a data synthesis. PLoS Med. 2015;12(12):e1001921. Available from:https:// journals.plos.org/plosmedicine/article?id=10.1371/journal.pmed.10019 21. Cited 2024 Aug 24.
- Aslam A, Hashmi MF, Okafor CN. Shigellosis. StatPearls. 2024 Feb 26; Available from: https://www.ncbi.nlm.nih.gov/books/NBK482337/. Cited 2024 Jun 23.
- Lampel KA, Formal† SB, Maurelli AT. A Brief History of Shigella. EcoSal Plus. 2018;8(1). Available from: /pmc/articles/PMC8559768/. Cited 2024 Jun 23.
- Wittenberg DF. The Spread of Shigella dysenteriae Type I in Africa. Jpn J Med Sci Biol. 1998;51:36–42.
- Guerin PJ, Brasher C, Baron E, Mic D, Grimont F, Ryan M, et al. Shigella dysenteriae serotype 1 in west Africa: intervention strategy for an outbreak in Sierra Leone. Lancet. 2003;362(9385):705–6 Available from: https:// pubmed.ncbi.nlm.nih.gov/12957094/.
- Kotloff KL, Blackwelder WC, Nasrin D, Nataro JP, Farag TH, Van Eijk A, et al. The Global Enteric Multicenter Study (GEMS) of diarrheal disease in infants and young children in developing countries: Epidemiologic and clinical methods of the case/control study. Clin Infect Dis. 2012;55(SUPPL. 4):S232–45.
- Breurec S, Rafaï C, Onambele M, Frank T, Farra A, Legrand A, et al. Serotype distribution and antimicrobial resistance of shigella species in Bangui, Central African Republic, from 2002 to 2013. Am J Tropical Med Hyg. 2018;99(2):283–6 Available from: https://www.scopus.com/inward/ record.uri?eid=2-s2.0-85051083508&doi=10.4269%2fajtmh.17-0917& partnerlD=40&md5=aceeeb4c3759b03836152c1e521b64ee.
- Kasumba IN, Badji H, Powell H, Hossain MJ, Omore R, Sow SO, et al. Shigella in Africa: New Insights From the Vaccine Impact on Diarrhea in Africa (VIDA) Study. Clin Infect Dis. 2023;76(Suppl 1):S66 Available from: / pmc/articles/PMC10116563/.
- Cahill KM, Davies JA, Johnson R. Report on an Epidemic Due to Shigella dysenteriae, Type 1, in the Somali Interior. Am J Trop Med Hyg. 1966;15(1):52–6 Available from: https://www.ajtmh.org/view/journals/ tpmd/15/1/article-p52.xmll.
- Engels D, Madaras T, Nyandwi S, Murray J. Epidemic dysentery caused by Shigella dysenteriae type 1: a sentinel site surveillance of antimicrobial resistance patterns in Burundi. Bull World Health Org. 1995;73(6):787–91 Available from: https://www.ncbi.nlm.nih.gov/pubmed/8907772.
- 11. Ahmed S, Chowdhury MIH, Sultana S, Alam SS, Marzan M, Islam MA. Prevalence of antibiotic-resistant *Shigella* spp. in Bangladesh: a systematic review and meta-analysis of 44,519 samples. Antibiotics. MDPI; 2023;12:817.

- 12. Ashbaugh H, Pomeroy CD, Baishya M, Creppage K, Bazaco S, Johnson M, et al. Antimicrobial resistance of enteric pathogens in the Military Health System, 2009–2019. BMC Public Health. 2022;22(1):2300.
- Áyele B, Beyene G, Alemayehu M, Dekebo A, Mekonnen Z, Nigussie G. Prevalence and antimicrobial-resistant features of *Shigella* species in East Africa from 2015–2022: a systematic review and meta-analysis. Interdiscip Perspect Infect Dis. 2023;2023:1–10.
- Baker S, Scott TA. Antimicrobial-resistant Shigella: where do we go next? Vol. 21, Nature Reviews Microbiology. Nature Research; 2023. p. 409–10.
- Brooks JT, Ochieng JB, Kumar L, Okoth G, Shapiro RL, Wells JG, et al. Surveillance for Bacterial Diarrhea and Antimicrobial Resistance in Rural Western Kenya, 1997–2003. 2006. Available from: https://academic.oup. com/cid/article/43/4/393/387096
- Gaudreau C, Ratnayake R, Pilon PA, Gagnon S, Roger M, Lévesque S. Ciprofl oxacin-resistant Shigella sonnei among men who have sex with men, Canada, 2010. Emerg Infect Dis. 2011S;17(9):1747–50.
- Hoffmann C, Sahly H, Jessen A, Ingiliz P, Stellbrink HJ, Neifer S, et al. High rates of quinolone-resistant strains of Shigella sonnei in HIV-infected MSM. Infection. 20130;41(5):999–1003.
- Opintan JA, Newman MJ. Distribution of serogroups and serotypes of multiple drug resistant Shigella isolates. 2007;41:8–29.
- Phiri AFND, Abia ALK, Amoako DG, Mkakosya R, Sundsfjord A, Essack SY, et al. Burden, antibiotic resistance, and clonality of *Shigella* spp. implicated in community-acquired acute diarrhoea in Lilongwe, Malawi. Trop Med Infect Dis. 2021;6(2):63.
- Ranjbar R, Farahani A. Shigella: Antibiotic-Resistance Mechanisms And New Horizons For Treatment. Infect Drug Resist. 2019;12:3137 Available from: /pmc/articles/PMC6789722/. Cited 2024 Apr 5.
- MacLennan CA, Steele AD. Frontiers in Shigella Vaccine Development. Vaccines (Basel). 2022;10(9). Available from: /pmc/articles/PMC9503259/. Cited 2024 May 28
- 22. Teimourpour R, Babapour B, Esmaelizad M, Arzanlou M, Peeri-Doghaheh H. Molecular characterization of quinolone resistant Shigella spp isolates from patients in Ardabil. Iran Iran J Microbiol. 2019;11(6):496 Available from: /pmc/articles/PMC7048964/. Cited Jun 23
- Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. The BMJ. 2021;29:372.
- Lewis JS, Weinstein MP, Bobenchik AM, Cameau Shelley, Cullen SK, Dingle Tanis et al. M100 : performance standards for antimicrobial susceptibility testing. Clinical and Laboratory Standards Institute; 2023. 358 p.
- Munn Z, Moola S, Lisy K, Riitano D, Tufanaru C. Methodological guidance for systematic reviews of observational epidemiological studies reporting prevalence and cumulative incidence data. Int J Evid Based Healthc. 2015;13(3):147–53 Available from: https://pubmed.ncbi.nlm.nih.gov/ 26317388/. Cited 2024 Jul 1.
- R Core Team. R: A Language and Environment for Statistical Computing. Vienna, Austria; 2024. Available from: https://www.R-project.org/
- 27. Balduzzi S, Rücker G, Schwarzer G. How to perform a meta-analysis with R: a practical tutorial. Evid Based Ment Health. 2019;22:153–60.
- Wang N. Conducting Meta-analyses of Proportions in R. Journal of Behavioral Data Science. 2023N 7;3(2):64–126.
- 29. Randremanana RV, Razafindratsimandresy R, Andriatahina T, Randriamanantena A, Ravelomanana L, Randrianirina F, et al. Etiologies, risk factors and impact of severe diarrhea in the under-fives in moramanga and antananarivo, Madagascar. PLoS One. 2016;11(7):e0158862.
- Mabika RM, Oyegue-Liabagui SL, Dibakou SE, Gabrielle M, Centre M, Régional H, et al. Etiology and biodistribution of enterobacteria and parasites, and their associated environmental risk factors among children under 5 years old with diarrhea in East-Central Gabon. 2020; Available from: https://doi.org/10.21203/rs.3.rs-131619/v1
- 31. Mengistu G, Mulugeta G, ... TLJMB, 2014 undefined. Prevalence and antimicrobial susceptibility patterns of Salmonella serovars and Shigella species. researchgate.netG Mengistu, G Mulugeta, T Lema, A AseffaJ Microb Biochem Technol, 2014-researchgate.net. 2014; Available from: https://www.researchgate.net/profile/Gebru-Weldearegay/publication/ 261097734_Prevalence_and_Antimicrobial_Susceptibility_Patterns_of_ Salmonella_serovars_and_Shigella_species/links/02e7e5332e8ea6fe7100 0000/Prevalence-and-Antimicrobial-Susceptibility-Patterns-of-Salmo nella-serovars-and-Shigella-species.pdf

- 32. Pavlinac PB, Denno DM, John-Stewart GC, Onchiri FM, Naulikha JM, Odundo EA, et al. Failure of syndrome-based diarrhea management guidelines to detect shigella infections in kenyan children. J Pediatric Infect Dis Soc. 2016;5(4):366–74 Available from: https://www.scopus. com/inward/record.uri?eid=2-s2.0-85014742610&doi=10.1093%2fjpids% 2fpiv037&partnerlD=40&md5=efc3801d6d94f3fb2856a0e4be813901.
- 33. Mokhtari W, Nsaibia S, Majouri D, Ben Hassen A, Gharbi A, Aouni M. Detection and characterization of Shigella species isolated from food and human stool samples in Nabeul, Tunisia, by molecular methods and culture techniques. J Appl Microbiol. 2012;113(1):209–22 Available from: https://doi.org/10.1111/j.1365-2672.2012.05324.x.
- Samuel SK, Moses NM, Emily TJ. Prevalence of Enterobacteriaceae Isolated from Childhood Diarrhoea in Mukuru Slums. Nairobi- Kenya J Adv Microbiol. 2019;25:1–9.
- Mogahid MEH, Naglaa ME, Misleyeen AE, Ahmed AAJ. Diarrheal diseases among internally home displaced (idps) in Khartoum state. Egypt Acad J biolog Sci. 2011;3(1):1–5 Available from: www.eajbs.eg.net .
- Mbuthia OW, Mathenge SG, Oyaro MO, Ng'ayo MO. Etiology and pathogenicity of bacterial isolates: A cross sectional study among diarrheal children below five years in central regions of Kenya. Pan Afr Med J. 2018;31(88).
- Muzembo BA, Kitahara K, Mitra D, Ohno A, Khatiwada J, Dutta S, et al. Burden of Shigella in South Asia: a systematic review and meta-analysis. J Travel Med. 2023;30(1). Available from: https://pubmed.ncbi.nlm.nih.gov/ 36331282/. Cited 2024 May 30.
- Muzembo BA, Kitahara K, Mitra D, Ohno A, Khatiwada J, Dutta S, et al. Shigellosis in Southeast Asia: A systematic review and meta-analysis. Travel Med Infect Dis. 2023;1(52):102554.
- Kasumba IN, Badji H, Powell H, Hossain MJ, Omore R, Sow SO, et al. Shigella in Africa: New Insights from the Vaccine Impact on Diarrhea in Africa (VIDA) Study. Clin Infect Dis. 2023;1(76):S66–76.
- Li Z, Wang L, Sun W, Hou X, Yang H, Sun L, et al. Identifying high-risk areas of bacillary dysentery and associated meteorological factors in Wuhan, China. Sci Rep. 2013;3. Available from: /pmc/articles/PMC3836034/. Cited 2024 Mar 26.
- Vubil D, Acácio S, Quintò L, Ballesté-Delpierre C, Nhampossa T, Kotloff K, et al. Clinical features, risk factors, and impact of antibiotic treatment of diarrhea caused by Shigella in children less than 5 years in Manhiça District, rural Mozambique. Infect Drug Resist. 2018;11:2095 Available from: / pmc/articles/PMC6219103/. Cited 2024 Mar 26.
- Bhattacharya SK, Sur D, von Seidlein L. Shigellosis. Reference Module in Biomedical Sciences. 202; Available from: https://linkinghub.elsevier.com/ retrieve/pii/B9780323999670000545. Cited 2024 Apr 3.
- HI D, Mm L, Rb H, Sb F. Inoculum size in shigellosis and implications for expected mode of transmission. J Infect Dis. 1989;159(6):1126–8 Available from: https://pubmed.ncbi.nlm.nih.gov/2656880/. Cited 2024 Apr 4.
- 44. Connor P, Porter CK, Swierczewski B, Riddle MS. Diarrhoea during military deployment: Current concepts and future directions. Curr Opin Infect Dis. 2012;25(5):546–54 Available from: https://journals.lww.com/co-infectious diseases/fulltext/2012/10000/diarrhoea_during_military_deployment__ current.11.aspx. Cited 2024 May 30.
- Sanford SM. Shigella dysenteriae (Shigellosis) Attack. Ciottone's Disaster Medicine. 2024;1:769–71.
- Rathish B, Pillay R, Wilson A, Pillay W. Comprehensive Review of Bioterrorism. StatPearls. 2023; Available from: https://www.ncbi.nlm.nih.gov/ books/NBK570614/. Cited 2024 Aug 24.
- Wasfy MO, Oyofo BA, David JC, Ismail TF, El-Gendy AM, Mohran ZS, et al. Isolation and Antibiotic Susceptibility of Salmonella, Shigella, and Campylobacter from Acute Enteric Infections in Egypt. Source: Journal of Health. 2000;18(1):33–8. Available from: https://www.jstor.org/stable/23499061? seq=1&cid=pdf-
- 48. Bhattacharya D, Bhattacharya H, Thamizhmani R, Sayi DS, Reesu R, Anwesh M, et al. Shigellosis in Bay of Bengal Islands, India: Clinical and seasonal patterns, surveillance of antibiotic susceptibility patterns, and molecular characterization of multidrug-resistant Shigella strains isolated during a 6-year period from 2006 to 2011. Eur J Clin Microbiol Infect Dis. 2014;33(2):157–70.
- Kotloff KL, Riddle MS, Platts-Mills JA, Pavlinac P, Zaidi AKM. Shigellosis. Lancet. 2018F 24;391(10122):801–12.

- Livio S, Strockbine NA, Panchalingam S, Tennant SM, Barry EM, Marohn ME, et al. Shigella isolates from the global enteric multicenter study inform vaccine development. Clin Infect Dis. 2014;59(7):933–41.
- Ram PK, Crump JA, Gupta SK, Miller MA, Mintz ED. Part II Analysis of data gaps pertaining to Shigella infections in low and medium human development index countries, 1984–2005. Epidemiol Infect. 2008;136(5):577–603.
- 52. Gu B, Cao Y, Pan S, Zhuang L, Yu R, Peng Z, et al. Comparison of the prevalence and changing resistance to nalidixic acid and ciprofloxacin of Shigella between Europe-America and Asia-Africa from 1998 to 2009. Int J Antimicrob Agents. 2012J 1;40(1):9–17.
- Thompson CN, Duy PT, Baker S. The Rising Dominance of Shigella sonnei: An Intercontinental Shift in the Etiology of Bacillary Dysentery. PLoS Negl Trop Dis. 2015;9(6). Available from: /pmc/articles/PMC4466244/. Cited 2024 Jun 22.
- Ranjbar R, Bolandian M, Behzadi P. Virulotyping of Shigella spp. isolated from pediatric patients in Tehran. Iran Acta Microbiol Immunol Hung. 2017;64(1):71–80 Available from: https://akjournals.com/view/journals/ 030/64/1/article-p71.xml. Cited 2024 Aug 27.
- Qu M, Zhang X, Liu G, Huang Y, Jia L, Liang W, et al. An eight-year study of Shigella species in Beijing, China: Serodiversity, virulence genes, and antimicrobial resistance. J Infect Dev Ctries. 2014;8(7):904–8.
- Vinh H, Baker S, Campbell J, Van Minh HN, Loan HT, Chinh MT, et al. Rapid emergence of third generation cephalosporin resistant Shigella spp. Southern Vietnam J Med Microbiol. 2009;58(2):281–3.
- Nuzhat S, Das R, Das S, Islam S Bin, Palit P, Haque MA, et al. Antimicrobial resistance in shigellosis: a surveillance study among urban and rural children over 20 years in Bangladesh. PLoS One. 2022;17(11 November):e0277574.
- Das A, Natarajan M, Mandal J. The emergence of quinolone resistant Shigella sonnei, Pondicherry, India. PLoS One. 2016;11(8):e0160290.
- Azmi IJ, Khajanchi BK, Akter F, Hasan TN, Shahnaij M, Akter M, et al. Fluoroquinolone Resistance Mechanisms of Shigella flexneri Isolated in Bangladesh. PLoS One. 2014;9(7):102533. Available from: /pmc/articles/ PMC4100904/. Cited 2024 May 28.
- Meron-Sudai S, Asato V, Adler A, Bialik A, Goren S, Ariel-Cohen O, et al. A Shigella flexneri 2a synthetic glycan-based vaccine induces a long-lasting immune response in adults. npj Vaccines 2023 8:1. 2023;8(1):1–10. Available from: https://www.nature.com/articles/s41541-023-00624-y. Cited 2024 May 28.
- MacLennan CA, Grow S, Ma LF, Steele AD. The Shigella Vaccines Pipeline. Vaccines (Basel). 2022;10(9). Available from: /pmc/articles/PMC9504713/. Cited 2024 May 30.
- 62. Hausdorff WP, Scheele S, Giersing BK. What Drives the Value of a Shigella Vaccine? Vaccines. 2022;10(2):282 Available from: https://www.mdpi.com/2076-393X/10/2/282/htm. Cited 2024 May 28.
- WHO. WHO Model List of Essential Medicines 23rd List (2023). 2023. Available from: https://www.who.int/publications/i/item/WHO-MHP-HPS-EML-2023.02. Cited 2024 Mar 25.
- Abd-Elmeged GM, Khairy RM, Abo-Eloyoon SM, Abdelwahab SF. Changing patterns of drug-resistant Shigella isolates in egypt. Microb Drug Resist. 2014;21(3):286–91. https://doi.org/10.1089/mdr.2014.0187.
- 65. Assefa A, Girma M. Prevalence and antimicrobial susceptibility patterns of Salmonella and *Shigella* isolates among children aged below five years with diarrhea attending Robe General Hospital and Goba Referral Hospital, South East Ethiopia. Trop Dis Travel Med Vaccines. 2019;5(1):19.
- 66. Feleke H, Medhin G, Abebe A, Beyene B, Kloos H, Asrat D. Enteric pathogens and associated risk factors among under-five children with and without diarrhea in Wegera district, northwestern Ethiopia. Pan African Medical Journal. 2018;24:29.
- 67. Gebreegziabher G, Asrat D, W/Amanuel Y, Hagos T. Isolation and antimicrobial susceptibility profile of Shigella and Salmonella species from children with acute diarrhoea in Mekelle Hospital and Semen Health Center. Ethiopia Ethiop J Health Sci. 2018;28(2):197–206.
- Kasumba IN, Badji H, Powell H, Hossain MJ, Omore R, Sow SO, et al. Shigella in Africa: New Insights from the Vaccine Impact on Diarrhea in Africa (VIDA) Study. Clinical Infectious Diseases. 2023;76:S66–76 https:// www.scopus.com/inward/record.uri?eid=2-s2.0-85160347556&doi=10. 1093%2fcid%2fciac969&partnerID=40&md5=53f14e75ccfead051517 33df491324a5.

- 69. Mama M, Alemu G. Prevalence, antimicrobial susceptibility patterns and associated risk factors of *Shigella* and Salmonella among food handlers in Arba Minch University, South Ethiopia. BMC Infect Dis. 2016;16(1):686.
- Mogga J, Oundo J, Journal GKSSM, 2015 undefined. Epidemiological and antibiotic susceptibility profiles of infectious bacterial diarrhoea in Juba, South Sudan. ajol.infoJJH Mogga, J Oundo, G KikuviSouth Sudan Medical Journal, 2015-ajol.info. 2015;8. Available from: https://www.ajol.info/index. php/ssmj/article/view/132330. Cited 2024 Feb 9.
- Tadesse G, Mitiku H, Teklemariam Z, Marami D. Salmonella and Shigella Among Asymptomatic Street Food Vendors in the Dire Dawa city, Eastern Ethiopia: Prevalence, Antimicrobial Susceptibility Pattern, and Associated Factors. Environ Health Insights. 2019;13.
- Terfassa A, Jida M. Prevalence and antibiotics susceptibility pattern of Salmonella and Shigella species among diarrheal patients attending Nekemte Referral Hospital, Oromia. Ethiopia Int J Microbiol. 2018;2018(24):9214689.
- Tiruneh M. Serodiversity and antimicrobial resistance pattern of Shigella isolates at Gondar University teaching hospital. Northwest Ethiopia Jpn J Infect Dis. 2009;62(2):93–7.
- 74. Iwalokun BA, Gbenle GO, Smith SI, Ogunledun A, Akinsinde KA, Omonigbehin EA. Epidemiology of shigellosis in Lagos, Nigeria: trends in antimicrobial resistance. J Health Popul Nutr. 2001;19(3):183–90 Available from: https://www.ncbi.nlm.nih.gov/pubmed/11761772.
- Mason PR, Nathoo KJ, Wellington M, Mason E. Antimicrobial susceptibilities of Shigella dysenteriae type 1 isolated in Zimbabwe–implications for the management of dysentery. Cent Afr J Med. 1995;41(4):132–7 Available from: https://www.ncbi.nlm.nih.gov/pubmed/7788685.
- Olaru I. Understanding Gram-negative infections and antimicrobial resistance in Zimbabwe. 2022; Available from: https://researchonline.lshtm.ac. uk/id/eprint/4670825/1/2022_ITD_PhD_Olaru_ID.pdf. Cited 2024 Jan 30.
- Njunda AL, Assob JCN, Nsagha DS, Kamga HLF, Awafong MP, Weledji EP. Epidemiological, clinical features and susceptibility pattern of shigellosis in the buea health district, Cameroon. BMC Res Notes. 2012;5. Available from: https://pubmed.ncbi.nlm.nih.gov/22264300/. Cited 2024 Apr 15.
- Naik DG. Prevalence and antimicrobial susceptibility patterns of Shigella species in Asmara, Eritrea, northeast Africa. J Microbiol Immunol Infect. 2006;39(5):392–5.
- 79. Kim JY, Kim SH, Jeon SM, Park MS, Rhie HG, Lee BK. Resistance to fluoroquinolones by the combination of target site mutations and enhanced expression of genes for efflux pumps in Shigella flexneri and Shigella sonnei strains isolated in Korea. Clinical Microbiology and Infection. 2008;14(8):760–5 Available from: http://www.clinicalmicrobiologyandin fection.com/article/S1198743X14621224/fulltext. Cited 2024 May 28.
- Cao M, Wang W, Zhang L, Liu G, Zhou X, Li B, et al. Epidemic and molecular characterization of fluoroquinolone-resistant Shigella dysenteriae 1 isolates from calves with diarrhea. BMC Microbiol. 2021;21(1):1–11 Available from: https://bmcmicrobiol.biomedcentral.com/articles/https://doi.org/10.1186/s12866-020-02050-9. Cited 2024 May 29.
- Qin T, Qian H, Fan W, Ma P, Zhou L, Dong C, et al. Newest data on fluoroquinolone resistance mechanism of Shigella flexneri isolates in Jiangsu Province of China. BMC. 2017;6(97).
- Xue C, Cai J, Kang H, Chen Y, Wang K, Qian H, et al. Two novel mutations in parE among Shigella flexneri isolated from Jiangsu Province of China, 2016. Ann Transl Med. 2018;6(15):306–306.
- Gaudreau C, Pilon PA, Cornut G, Marchand-Senecal X, Bekal S. Shigella flexneri with ciprofloxacin resistance and reduced azithromycin susceptibility, Canada, 2015. Emerg Infect Dis. 2016;22(11):2016–8.
- Chung The H, Rabaa MA, Pham Thanh D, De Lappe N, Cormican M, Valcanis M, et al. South Asia as a reservoir for the global spread of ciprofloxacin-resistant *Shigella* sonnei: a cross-sectional study. PLoS Med. 2016;13(8):e1002055.
- WHO. WHO Global Foodborne Infections Network 'A WHO network building capacity to detect, control and prevent foodborne and other enteric infections from farm to table' Laboratory Protocol: 'Isolation of Salmonella and Shigella from Faecal Specimens'. 2010;

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