

REVIEW

Cholera in Sub-Saharan Africa: From the Biology of *Vibrio Cholerae* to the Challenges of Surveillance, with a Focus on the Democratic Republic of the Congo

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Abstract: Cholera remains a major public health problem in sub-Saharan Africa, where limited water and sanitation, population displacement, conflict and weak health systems sustain recurrent epidemics. Interpreting its epidemiology requires linking the biology of *Vibrio cholerae*, antimicrobial resistance, and the performance of surveillance and diagnosis. We conducted a structured narrative review, prioritising meta-analyses, systematic reviews, epidemiological and genomic studies, and WHO and Global Task Force on Cholera Control (GTFCC) documents, with emphasis on sub-Saharan Africa, the Great Lakes region and the Democratic Republic of the Congo (DRC). Cholera in the region is a system combining an aquatic reservoir, foci of person-to-person transmission, structural determinants (water, sanitation and hygiene, rainfall, conflict, displacement) and repeated introductions of seventh-pandemic lineages. In the eastern DRC, genomic data show stable AFR10 sublineages persisting around the Great Lakes, with a ciprofloxacin-reducing *parC* mutation in one Lake Tanganyika clade. Resistance to historical antibiotics is common but heterogeneous across countries, years and laboratory methods, and data from central Africa are sparse, notably for macrolides. Laboratory surveillance is essential but limited by testing coverage, specimen transport and variable case definitions, which bias confirmed-case counts and positivity rates. Single-dose oral vaccination, water, sanitation and hygiene, and targeted response around cases are complementary, and none is sufficient alone. Understanding and controlling cholera in the DRC requires locally documented, standardised laboratory data reported with explicit denominators, stronger decentralised confirmation and genomic surveillance, and sustained investment in prevention. We identify four research priorities to guide integrated surveillance and outbreak response.

Keywords: Cholera, *Vibrio cholerae*, Sub-Saharan Africa, Democratic Republic of the Congo, great lakes region, antimicrobial resistance

1 Introduction

Cholera remains a major global public health problem, particularly in sub-Saharan Africa, where epidemics continue to occur against a background of limited access to safe water and sanitation, socioeconomic inequality, population displacement, humanitarian crises and constrained health systems. In 2024, 22 countries and territories of the World Health Organization (WHO) African Region reported 258,442 cholera cases and 4,976 deaths, illustrating the persistence of a substantial epidemiological burden [1]. Caused by the ingestion of water or food contaminated with toxigenic strains of *Vibrio cholerae*, this acute diarrhoeal disease can progress rapidly to severe dehydration and death without appropriate care [2]. Despite advances in prevention, diagnosis and treatment, the persistence of outbreaks underlines the need to better understand the biological, environmental and social mechanisms that sustain transmission.

The epidemiology of cholera results from the interaction of pathogen characteristics, environmental conditions, human behaviour and the capacity of health systems. The diversity of circulating strains, virulence mechanisms, the genomic evolution of *V. cholerae* and the emergence of antimicrobial resistance all shape the course of outbreaks and the options for controlling them. At the same time, the quality of epidemiological surveillance and microbiological investigation determines case detection, outbreak confirmation, strain characterisation and the interpretation of observed trends. According to the Global Task Force on Cholera Control (GTFCC), surveillance should produce reliable and timely information to support early outbreak detection, guide interventions and monitor progress of control strategies [3]. These data must nevertheless be read in light of the limits of surveillance systems, notably the availability of confirmatory tests, laboratory capacity, constraints on specimen transport and storage, and the likely incompleteness of notifications.

These issues are particularly important in sub-Saharan Africa, where transmission settings and surveillance capacities are heterogeneous. In the African Great Lakes region, cross-border population movements, conflict-related displacement, limited access to essential services and fragile water and sanitation infrastructure complicate prevention and outbreak control [4–7]. The Democratic Republic of the Congo (DRC) is a particularly relevant setting in which to examine these interactions, given the recurrence of epidemics in several provinces and the challenges of surveillance, microbiological confirmation and characterisation of circulating strains [5,6]. Analysing this situation requires going beyond a purely descriptive account of cases and deaths to consider the determinants of transmission, the microbiological characteristics of the pathogen and the limits of the available data.

An integrated understanding of cholera therefore requires linking knowledge on the biology of *V. cholerae*, the epidemiological burden and its determinants, antimicrobial resistance profiles, and the performance of surveillance and diagnostic systems. This perspective allows a better appreciation of what the available information can and cannot tell us, in order to guide public health interventions, strengthen laboratory capacity and improve early outbreak detection.

This article presents a narrative review of the literature on cholera in sub-Saharan Africa, with particular attention to the DRC and the Great Lakes region. It aims to synthesise knowledge on the biology and virulence of *V. cholerae*, to describe the epidemiological burden and the main determinants of transmission, to examine the available data on antimicrobial resistance, and to analyse the contributions and limitations of epidemiological surveillance and microbiological diagnosis. By connecting these dimensions, the review seeks to highlight the scientific and operational gaps that limit understanding and control of cholera in the region, and to identify ways of improving integrated surveillance and outbreak response.

2 Review approach

This is a structured narrative review. Unlike a systematic review, it does not aim to retrieve every available publication or to pool results quantitatively for a single research question. It offers a critical, contextualised synthesis that connects biological, epidemiological and microbiological evidence with the operational realities of cholera surveillance. Its conduct and reporting follow published guidance on narrative reviews, namely a clear statement of aims, transparency about the literature search, reasoned selection of sources and a coherent argument [8,9].

We gave priority to meta-analyses, relevant systematic reviews, epidemiological and genomic studies, and technical documents of the WHO and the GTFCC. Particular attention was paid to publications on sub-Saharan Africa, the Great Lakes region and the DRC, without excluding the international work needed to understand the biological mechanisms and the evolution of the disease. The review is organised around five questions:

- (1) What are the biological characteristics of *V. cholerae* and the pathophysiological basis of cholera?
- (2) What is the burden of cholera worldwide and in sub-Saharan Africa, and which factors drive transmission?
- (3) What is the epidemiological situation of cholera in the DRC and the Great Lakes region?
- (4) What data are available on antimicrobial resistance in *V. cholerae*?
- (5) What do epidemiological surveillance and microbiological diagnosis contribute to case detection, strain characterisation and data interpretation, and what are their limits?

A critical synthesis (section 11) brings these elements together to identify the main scientific

and operational gaps.

3 The pathogen: biology and virulence determinants

3.1 Taxonomy and diversity

Vibrio cholerae is a curved, Gram-negative, oxidase-positive bacillus, motile by a single polar flagellum, which grows well in alkaline media. This property underpins enrichment in alkaline peptone water and isolation on TCBS agar, where sucrose-fermenting colonies appear yellow [10, 11]. The species comprises more than 200 serogroups defined by the lipopolysaccharide O antigen. Only serogroups O1 and O139 carry the determinants needed to cause cholera epidemics. Non-O1/non-O139 strains cause sporadic gastroenteritis or, more rarely, extra-intestinal infections [10, 11].

Serogroup O1 is divided into two biotypes, classical and El Tor, and three serotypes, Ogawa, Inaba and Hikojima. Serotypes differ by methylation of the O antigen; Hikojima is an unstable intermediate form [10, 11]. The El Tor biotype has progressively replaced the classical biotype and is responsible for the seventh pandemic. “Atypical” or “hybrid” El Tor variants, which carry classical-biotype traits (notably the allele of the toxin B subunit), have spread and are associated with more severe clinical presentations [2, 12].

3.2 Virulence determinants and ecology

The virulence of *V. cholerae* O1 and O139 rests on two main elements. The first is cholera toxin, an AB₅ toxin whose *ctxAB* genes are carried by the filamentous bacteriophage CTX ϕ , integrated in the chromosome. Acquisition of this phage by lysogenic conversion turns a harmless environmental strain into an epidemic strain [13]. The second is the toxin-coregulated pilus (TCP), a small-intestine colonisation factor that also serves as the receptor for CTX ϕ . Expression of these factors is coordinated by a regulatory cascade (ToxR/ToxT) that responds to signals from the intestinal environment [2, 11].

The bacterium is primarily an aquatic organism. It persists in brackish, estuarine and lacustrine waters, often associated with zooplankton and chitinous surfaces, as biofilms or viable-but-non-culturable states that escape conventional culture [2, 10]. After passage through the human gut, the vibrio becomes transiently hyperinfectious: freshly shed bacteria are much more infectious than in vitro-grown cells, which facilitates explosive transmission during outbreaks [14]. This dual environmental and human life cycle explains why cholera combines local cycles of person-to-person transmission with resurgences linked to environmental conditions.

4 Pathophysiology, clinical features and management

Cholera toxin irreversibly activates adenylate cyclase in enterocytes, raising intracellular cyclic AMP and causing massive secretion of chloride, bicarbonate and water. The result is watery diarrhoea that can reach several litres per hour in the most severely ill patients, with up to 10⁷–10⁸ vibrios per millilitre of stool [2, 12]. The incubation period ranges from a few hours to five days, with a median of about 1.4 days [15]. Roughly three in four infections are asymptomatic or mild, yet carriers still shed the organism and sustain transmission. Among symptomatic patients, about one in five develops severe disease, marked by major dehydration that can lead within hours to hypovolaemic shock, acute kidney injury and death [2, 12].

Rehydration, oral or intravenous according to severity, is the cornerstone of treatment; when early and adequate it brings case fatality below 1% [16, 17]. Antibiotic therapy is an adjunct reserved for severe cases and patients at risk. A Cochrane review showed that it reduces the duration of diarrhoea, the volume of fluid losses and the duration of bacterial shedding [16]. The therapeutic value of antibiotics therefore depends directly on the susceptibility of circulating strains, which is why resistance surveillance matters (section 8). Confirmatory diagnosis, based on culture and PCR, is discussed in section 9. Figure 1 summarises the transmission cycle of *V. cholerae* and the points at which each control intervention acts.

5 Cholera worldwide

The seventh pandemic, triggered by the El Tor biotype in 1961, reached Africa in 1970 and has not been interrupted since [12]. In 2024, the WHO recorded more than 560,000 cases and 6,028 deaths reported by 60 countries, compared with 45 countries in 2023. Cases rose by 5% and deaths by 50% relative to 2023, for an overall case fatality of 1.1% [1]. Africa accounts

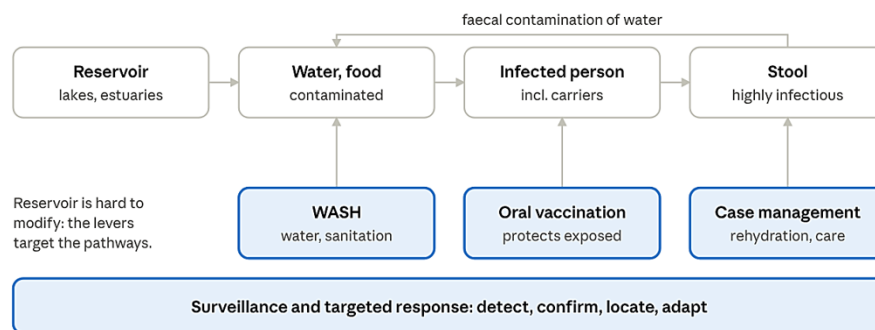


Figure 1 Transmission cycle of *Vibrio cholerae* and points of action of control interventions (Interventions target the transmission pathways, not the reservoir)

for nearly half of reported cases and the large majority of deaths, with a case fatality of about 1.9%, above the 1% threshold set as a target for quality of care [1]. These figures are floors: modelling of the burden in endemic countries yields estimates of 1.3 to 4 million cases and 21,000 to 143,000 deaths per year, illustrating the scale of under-reporting [18]. Long-term data for the WHO African Region show the same pattern of recurrent, large epidemics [19].

The GTFCC global roadmap, launched in 2017, set three targets for 2030: to reduce cholera deaths by 90%, to eliminate the disease in up to 20 countries, and to implement funded multisectoral national plans in all priority countries [20]. The 2024 assessment shows that progress is insufficient: the number of affected countries has almost doubled since 2017 and very large outbreaks have tripled [1]. The main drivers identified are conflict, population displacement, climate change and persistent deficits in water, sanitation and hygiene (WASH) [1].

Genomics has profoundly changed how this pandemic is read. Analysis of several hundred genomes showed that African epidemics result from successive introductions, at least eleven since 1970, of seventh-pandemic lineages from South Asia, followed by regional spread and lineage replacement [21]. These lineages, designated with the prefix AFR, differ in geographical distribution and in resistance determinants. This genomic reading means that African cholera is no longer viewed as an “autochthonous” disease sustained by local reservoirs, but as a system in which local persistence, population mobility and reintroductions combine [21].

6 Cholera in sub-Saharan Africa

6.1 A burden concentrated in persistent hotspots

An analysis of cholera burden in sub-Saharan Africa across geographical scales showed marked spatial heterogeneity: incidence is concentrated in a limited number of areas of persistent transmission, which argues for interventions targeted at these hotspots rather than uniform national action [22]. Eliminated from high-income countries through sanitation, cholera remains in Africa a preventable disease that strikes the poorest populations first, hence the image of a “lion in the village” [23]. Determinants vary by region: along the coast they are heterogeneous and tied to the local environment [24], whereas in the Great Lakes region they combine a lake reservoir, high population density and high mobility (section 7).

6.2 Water, sanitation and hygiene

Limited access to safe water, inadequate sanitation and poor hygiene practices are the best-established determinants. A meta-analysis of case-control studies showed that household water treatment, handwashing and access to sanitation facilities are associated with lower cholera risk [25]. A systematic review of WASH interventions concluded that a protective effect is plausible, but rests on evidence that is often of low quality, especially for sanitation and hygiene interventions [26]. National meta-analyses point the same way: in Ethiopia, consumption of untreated water, lack of latrines and poor hygiene practices are associated with outbreaks [27]; in Ghana, epidemics have been linked to inadequate sanitation, consumption of street-vended food and proximity to refuse dumps [28].

6.3 Climate and seasonality

Cholera seasonality is not uniform worldwide: it depends on climate, rainfall regime and local context [29]. In sub-Saharan Africa many outbreaks coincide with rainy seasons and

floods, which promote faecal contamination of water sources and proliferation of *V. cholerae* in the environment [30,31]. Analyses of environmental factors show that rainfall and temperature, combined with access to water and sanitation, are major determinants of endemic risk [31,32]. Climate change, by increasing the frequency of extreme events, is likely to worsen this exposure [33]. These findings argue for characterising the seasonal profile locally rather than deducing it from regional patterns, all the more so because the effects of rainfall vary from year to year.

6.4 Conflict, displacement and urbanisation

Humanitarian crises occupy a growing place among the drivers of the epidemic: the 2024 global assessment identifies conflict, population displacement and infrastructure deficits as major factors in the current dynamics [1]. Displacement camps and precarious urban neighbourhoods combine overcrowding, restricted access to safe water and inadequate sanitation. They also weaken the health system itself: supply-chain disruption, difficulties in transporting specimens and loss of trained staff. The recent crisis in Goma illustrates the public health consequences of such disruption in the eastern DRC [7]. These conditions have a double effect: they increase incidence and degrade the quality of the data that measure it.

6.5 Fine-scale spatio-temporal structure and targeting

At very small scale, cholera cases cluster in time and space: risk is high in the immediate neighbourhood of a case and in the days that follow [34]. This property underlies targeted response strategies around cases (chlorination, hygiene promotion, ring vaccination). It presupposes a surveillance system able to locate confirmed cases rapidly, which brings back the laboratory constraints described in section 9. (see Table 1)

Table 1 Determinants of cholera in sub-Saharan Africa: summary of the literature

Determinant	Main mechanism	Sources
Unsafe water, inadequate sanitation and hygiene	Faecal contamination of water and food; person-to-person transmission	[25–28]
Rainfall, floods, temperature	Contamination of water resources; survival and multiplication of the vibrio in the environment	[29–33]
Conflict, displacement, precarious urbanisation	Overcrowding, service breakdown, delayed response	[1, 7]
Spatio-temporal clustering of cases	Proximity transmission (households, neighbourhood)	[34]

7 Cholera in the Democratic Republic of the Congo

7.1 Endemicity anchored in the east

The DRC is one of the most affected countries in Africa. The Great Lakes of the East (Tanganyika, Kivu, Albert, Edward, Mweru) have been identified as foci from which epidemics spread [35]. Disease dynamics in the Great Lakes region follow multi-year cycles of resurgent transmission, with repeated resurgences from these foci [4]. An analysis of epidemics from 2008 to 2017 confirms the recurrence of outbreaks and the central role of the eastern provinces as a zone of persistence [5]. A spatio-temporal analysis from 1973 to 2018 shows that spread towards the west of the country is not unidirectional from east to west [36], and an eco-epidemiological study of 2000–2018 examined the drivers of this spread [6]. Around Goma and Lake Kivu, an eight-year survey linked cholera epidemics to war and disasters [37].

Armed conflict in the east, which worsened in early 2025 with the occupation of Goma and Bukavu, has disrupted supply routes, the functioning of health facilities and the continuity of surveillance and vaccination in these provinces [7]. The situation is changing rapidly and the most recent WHO and Ministry of Health bulletins should be consulted for current figures.

7.2 What genomics reveals

Genomic data from the eastern DRC converge. Isolates from 2014 to 2017, sequenced from 78 clinical strains, belonged to the seventh-pandemic lineage AFR10 (introduction “T10” of the third wave), with two subsets of distinct geographical distribution, one largely restricted to the Lake Tanganyika basin and the other disseminated along the Albertine rift [38]. An analysis of isolates from the Congolese shore of Lake Tanganyika from 2001 to 2020 showed

that they belonged to two of the five clades of the AFR10 sublineage, and that one clade became predominant after acquiring a *parC* mutation that reduces susceptibility to ciprofloxacin [39]. More recently, sequencing of 263 O1 isolates collected between 2018 and 2024 in North Kivu, South Kivu, Tanganyika and Kasai Oriental placed all isolates in sublineages AFR10d and AFR10e; AFR10e spread across the four provinces whereas AFR10d appeared extinct by the end of 2020, and rearrangements of the CTX prophage element were observed from 2022 [40].

In Bukavu, four years of epidemiological and genomic surveillance revealed bimodal annual peaks of AFR10e clade infections and a clonal outbreak fourteen months after a campaign of more than one million vaccine doses, explained in part by 9% coverage in informal neighbourhoods. The effectiveness of one dose of killed whole-cell vaccine, estimated at 24 months, was 59.8% (95% CI 19.7–79.9) [41]. These results show both the remarkable stability of the lineages circulating in the region and the role of vaccination coverage gaps in the persistence of transmission.

7.3 Goma and North Kivu

Goma holds a singular position. The July 1994 epidemic among Rwandan refugees, which caused tens of thousands of deaths within a few weeks, remains one of the most dramatic examples of the vulnerability of displaced populations to cholera [42]. The 2023–2027 multi-sectoral strategic plan for cholera elimination classifies the health zones of Goma, Karisimbi, Nyiragongo and Kirotshe as high-risk, because of rapid urbanisation, insufficient access to water and sanitation, and strong cross-border mobility [43]. The area is thus durably exposed to transmission, while published longitudinal microbiological data on its health zones remain scarce.

7.4 Surveillance: a weakened system

Surveillance relies on the national integrated disease surveillance and response system, supported by the WHO and the National Institute for Biomedical Research. A framework for evaluating cholera surveillance systems in Africa identifies three interdependent pillars: resource allocation (staff, funding, digital infrastructure), organisational structures (governance, coordination, laboratory–surveillance network) and core functions (detection, confirmation, analysis, feedback, response) [44]. The weaknesses described in Africa, and often echoed for the DRC, are late detection, under-reporting, weak integration of epidemiology and laboratory, limited decentralised confirmation capacity and the lack of robust cross-border coordination [44,45]. The absence or fragility of any one pillar compromises the speed of case confirmation and the quality of resistance data.

8 Antimicrobial resistance in *Vibrio cholerae*

8.1 Mechanisms of acquisition and spread

V. cholerae has several routes to acquire resistance: chromosomal mutations in antibiotic target enzymes, efflux pumps, class 1 integrons carrying resistance gene cassettes, integrative conjugative elements of the SXT/R391 family, and conjugative plasmids [46]. The SXT element, first described in serogroup O139, carries determinants of resistance to sulfamethoxazole, trimethoprim and streptomycin and can transfer to other strains [47]. Multidrug-resistant IncC plasmids identified during the Haiti epidemic illustrate the rapid acquisition of broad resistance panels [48]. Mutations in *gyrA* and *parC* decrease susceptibility to quinolones, as in the Congolese clade mentioned above [39]. Figure 2 summarises these mechanisms and their phenotypic consequences.

8.2 Global trends

Available meta-analyses draw a contrasting picture. A meta-analysis of 131 articles on antibiotics that inhibit cell-wall synthesis shows increasing resistance over time to cefalotin, ceftriaxone, amoxicillin and meropenem, and decreasing resistance to penicillin, ceftazidime and cefotaxime [49]. A meta-analysis on tetracyclines (tetracycline and doxycycline) examined the global status of resistance and confirms that this class, first-line in many protocols, needs close monitoring [50]. These studies underline the strong heterogeneity of rates between countries, years and susceptibility testing methods.

8.3 Sub-Saharan Africa

A systematic review covering 16 sub-Saharan African countries retained only 25 studies on antibiotic susceptibility published between 2000 and 2017. Isolates belonged to serogroup O1,

Five genetic routes lead to reduced susceptibility in *V. cholerae*

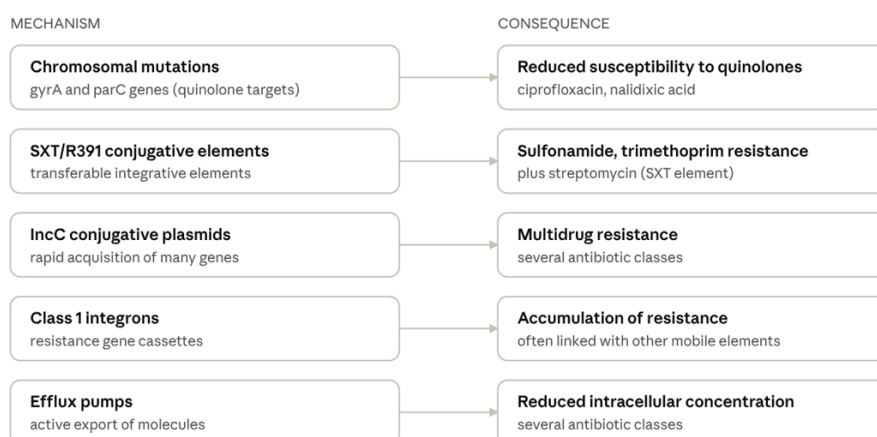


Figure 2 Mechanisms of acquisition of antimicrobial resistance in *Vibrio cholerae* and their phenotypic consequences

biotype El Tor, with alternating Ogawa and Inaba serotypes. Resistance was documented for several classes, including cell-wall-active agents and antimetabolites, with signs of phenotypic and genotypic resistance to fluoroquinolones; resistance mechanisms were investigated in only 40% of studies [51]. In the DRC itself, an analysis of 1,093 isolates collected between 1997 and 2012 found increasing antimicrobial resistance over time [52]. National series confirm this picture: in Kenya, strains from the 2017 epidemic showed high resistance to ampicillin, amoxicillin and cotrimoxazole [53]; in Zambia, multidrug-resistant El Tor strains emerged as early as the epidemics of the 2000s [54]; in Mozambique, strains from 2012–2015 showed extensive resistance [55]; in West Africa, a variant with reduced susceptibility to ciprofloxacin was described as early as 2010 [56]. More recent data from Zambia [57, 58] and Ethiopia [59], a genomic analysis of the 2022 outbreak in India [60] and a narrative review on Africa and the Middle East [61] confirm that resistance evolves quickly and that the profile of one region at one time cannot be extrapolated to others. (see Table 2)

Table 2 Resistance of *V. cholerae* to the main antibiotic classes in sub-Saharan Africa: qualitative synthesis of the reviews and series cited

Antibiotic class	Profile reported in sub-Saharan Africa	Sources
Aminopenicillins (ampicillin, amoxicillin)	Frequent resistance	[51, 53]
Cotrimoxazole, chloramphenicol	Frequent to near-universal resistance in several series	[51, 53–55]
Tetracyclines (doxycycline)	Variable; to be monitored as a first-line drug	[50, 51]
Fluoroquinolones	Signals of reduced susceptibility (<i>gyrA/parC</i> mutations)	[39, 51, 56]
Macrolides (erythromycin, azithromycin)	Limited data; possible heterogeneity by molecule and area	[51]

8.4 Interpreting antibiograms: methodological limits

Interpretive criteria of the Clinical and Laboratory Standards Institute (document M45) and of EUCAST are not strictly equivalent and do not cover all molecules of interest in the same way [62, 63]. Laboratories in resource-limited countries use discs, media and reference standards that are sometimes heterogeneous. This heterogeneity complicates comparison over time and between regions: an observed change in resistance may reflect a change in method as much as a biological change. The frequent grouping of “intermediate” and “resistant” categories as “non-susceptible” adds a difficulty of clinical interpretation.

Particular attention is required for macrolides. Resistance to erythromycin with preserved susceptibility to azithromycin, sometimes reported, could reflect mechanisms of limited expression, differences in reading of inhibition zones, or molecule-specific effects. These hypotheses can only be resolved by genotypic investigation (screening for macrolide resistance determinants)

and by reference methods (minimum inhibitory concentration). Few published data document the geographical distribution of erythromycin resistance in central Africa.

9 Surveillance and microbiological diagnosis

9.1 The surveillance framework

GTFCC guidance describes surveillance built on detection of suspected cases, laboratory confirmation of a share of them, and identification of priority areas of transmission. It stresses the need to document the proportion of suspected cases actually tested and the test positivity rate in order to interpret trends [3]. The suspected-case definition itself matters: in seven African countries, the WHO case definition had a sensitivity of 92.7% but a specificity of only 8.1% against culture [64]. The link between clinic, laboratory and public health decision is therefore central, and a break at any link affects the whole chain.

9.2 Confirmation methods

Culture remains the reference method for isolation, serotyping, antibiogram and later genomic characterisation, but it is demanding: it requires transport under conditions that preserve vibrio viability, suitable media and several days before a result. PCR is more sensitive and less dependent on bacterial viability, but yields no isolate for antibiogram. Rapid diagnostic tests, useful for early field detection, have moderate performance. A meta-analysis of 20 studies reports modest sensitivity and specificity relative to culture or PCR, with higher sensitivity in studies conducted in Africa [65]. A prior enrichment step improves their performance [66,67]. These tests therefore guide the response but do not replace laboratory confirmation when the antibiogram or serotype is needed. (see Table 3)

Table 3 Cholera confirmation methods: contributions and limits

Method	Main contribution	Main limits
Rapid diagnostic test	Early field detection, result in about 30 minutes	Moderate performance; no isolate; confirmation required [65–67]
PCR	High sensitivity, independent of viability	No isolate for antibiogram; equipment and reagents needed
Culture	Reference: isolate, serotype, antibiogram, genomics	Delay of several days; sensitive to transport conditions

9.3 A major bias: testing activity

The number of confirmed cases and the positivity rate depend both on the disease and on how testing is done. Burden estimates rely on surveillance data whose quality varies widely between countries [68,69]. When specimen collection criteria change (extension of sites, introduction of PCR, change of suspected-case definition), the number of confirmed cases can rise without any rise in true incidence. Operational difficulties described in several African countries (specimen transport, delays in reporting results, insufficiently trained staff, non-harmonised procedures) add to this bias and affect the reliability of antibiograms [70]. Interpreting laboratory series therefore requires a critical reading of denominators, that is, of the number of people tested, and an explicit description of the operational context.

10 Prevention and response

Prevention rests on three complementary levers. The first, structural, is lasting improvement of water, sanitation and hygiene, whose protective effect is plausible but for which the evidence is often of limited quality [25,26].

The second is oral vaccination. A meta-analysis showed that killed whole-cell vaccines protect against cholera, with more modest protection in very young children and declining protection over time [71]. An effectiveness study during an outbreak in Guinea confirmed the value of reactive vaccination [72]. Since 2013 a global stockpile has allowed rapid deployment of campaigns, and use rose sharply between 2013 and 2018 [73]; WHO recommendations on cholera vaccines specify their indications [74]. Faced with vaccine shortages, a single-dose strategy was adopted for reactive campaigns in 2024 [1]. Effectiveness of one dose remains moderate: it was estimated at 59.8% at 24 months in a surveillance study in Bukavu [41], and

a matched case-control study in an endemic community of the DRC also assessed one-dose effectiveness [75]. The third lever is targeted response around cases, which relies on the spatio-temporal clustering of cases [34]. None of these levers is sufficient alone: a review of cholera resurgence in Africa stresses the need to combine them with effective surveillance to reach the 2030 elimination target [76].

11 Critical synthesis, knowledge gaps and research priorities

Three findings emerge from this review. First, cholera in sub-Saharan Africa is a system in which an environmental reservoir, foci of person-to-person transmission, structural determinants (water, sanitation, conflict, displacement) and introductions of genomic lineages combine, so reasoning based on any single element is inadequate [21,22,35]. Second, antimicrobial resistance is widespread for historical drugs and remains strongly heterogeneous over time, across space and between laboratory methods; signals for fluoroquinolones and the place of macrolides are points of vigilance [39,51,52]. Third, laboratory surveillance data are indispensable but subject to testing biases that must be accounted for when interpreting trends [3,68]. These findings point to four gaps, summarised in (see Table 4) together with the research priorities they imply.

Table 4 Knowledge gaps and research priorities

Gap identified in the literature	Research priority
Few local data on the spatio-temporal and seasonal distribution of laboratory-confirmed cholera in high-risk urban areas such as Goma [29,43]	Multilevel analyses of test positivity by season, year, age, sex and health zone, with local rather than regional seasonal profiles
No integrated longitudinal description of trends, serotypes and antibiotic susceptibility that accounts for testing activity [3,68,70]	Multi-year laboratory surveillance series reported with explicit denominators (suspected cases and persons tested)
Geographical heterogeneity of resistance profiles in the DRC poorly documented, notably for erythromycin and macrolides [51,52]	Standardised susceptibility testing, MIC confirmation and genotypic screening, compared across provinces
Few analyses that account for the hierarchical structure of surveillance data and the limits of routine data [64,68]	Multilevel and penalised regression models, and explicit discussion of testing bias in published analyses

12 Conclusion

Cholera in sub-Saharan Africa, and in the DRC in particular, cannot be understood through case counts alone. Its persistence reflects the interplay of environmental reservoirs, lineages introduced from outside the continent and then maintained locally, structural vulnerabilities, and surveillance systems whose capacity to detect, confirm and characterise cases is uneven. Antimicrobial resistance is evolving quickly and varies by place, time and laboratory method, so regional profiles cannot be assumed to apply elsewhere. Local, well-documented laboratory data, reported with explicit denominators and standardised susceptibility methods, are needed to support treatment guidance and targeted control. Strengthening decentralised laboratory confirmation, genomic surveillance and cross-border coordination, alongside sustained investment in water, sanitation and vaccination, is essential to progress towards the 2030 elimination goals.

Abbreviations

AFR: African lineage designation of the seventh pandemic;
 AMR: antimicrobial resistance;
 CTX ϕ : cholera toxin phage;
 DRC: Democratic Republic of the Congo;
 EUCAST: European Committee on Antimicrobial Susceptibility Testing;
 GTFCC: Global Task Force on Cholera Control;
 MIC: minimum inhibitory concentration;
 OCV: oral cholera vaccine;
 TCP: toxin-coregulated pilus;
 WASH: water, sanitation and hygiene;
 WHO: World Health Organization.

Conflicts of interest

The authors declare no conflicts of interest.

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