

Environmental Antimicrobial Resistance in Africa: Wastewater, Aquatic Ecosystems and the One Health Surveillance Gap

Okoye Rosemary^{1*}, A.M. Ashafa²

¹Department of Microbiology, Federal University Gusau, Zamfara State, Nigeria. <https://orcid.org/000-0002-6090-6125>

²Department of Microbiology, Faculty of Life Sciences, Ahmadu Bello University, Zaria. <https://orcid.org/0000-0001-8332-0808>

ABSTRACT

Antimicrobial resistance (AMR) is increasingly recognised as an environmental problem and not only a clinical one, yet Africa's wastewater and aquatic ecosystems remain an understudied part of its emergence and spread. This review synthesises recent evidence on antimicrobial-resistant bacteria (ARB) and antimicrobial resistance genes (ARGs) in African wastewater, rivers and sediments, drawing on studies from Nigeria, Ghana, Kenya, Uganda, South Africa, Ethiopia, the Democratic Republic of Congo, Mali, Zambia and Tanzania. Hospital and municipal wastewater consistently carry high loads of ARB and clinically important ARGs, much of which survives treatment and is discharged into receiving rivers, where resistance levels often rise rather than fall downstream. Sediments and aquatic microbial communities act as additional, longer-term reservoirs. Because these environmental reservoirs connect directly to human, animal and agricultural compartments, a One Health perspective is essential, yet integrated cross-sectoral surveillance remains rare on the continent. The review identifies the principal surveillance gaps and outlines priority directions needed to close Africa's environmental AMR surveillance gap.

KEYWORDS: antimicrobial resistance; wastewater; Africa; One Health; aquatic ecosystems; surveillance

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I. INTRODUCTION

Antimicrobial resistance (AMR) occurs when bacteria and other microorganisms evolve mechanisms that reduce or eliminate the effectiveness of the drugs used to treat them. For decades AMR was framed largely as a clinical and hospital-based problem, tracked through susceptibility testing of patient isolates. This framing captures only part of the picture: environmental compartments such as wastewater harbour a wide diversity of antibiotic resistance genes (ARGs), often alongside residual antibiotic concentrations that create the selective pressure for resistant strains to persist and spread [1]. Resistance genes circulate continuously between clinical, agricultural and environmental settings through horizontal gene transfer.

Africa deserves particular attention. Antimicrobial use in food-producing animals has been reported by the large majority of farms surveyed across the continent [2], and environmental contamination routes linking hospitals, households, agriculture and waterways are increasingly well documented. Despite this, the region remains among the most

under-represented in global AMR surveillance data, and a systematic review of studies examining AMR simultaneously across human, animal and environmental compartments in Africa found integrated evidence to be genuinely scarce [3]. This review therefore focuses on the environmental dimension of AMR in Africa, wastewater, rivers, and the sediments and microbial communities within them, as reservoirs in their own right.

This focus is not simply a matter of completeness. Clinical surveillance alone cannot explain why resistant organisms recur in communities after treatment programmes appear to succeed, because it does not track what happens to resistant bacteria and their genes once they leave the human body. Wastewater and surface water are the connective tissue between the clinical, agricultural and community compartments: they receive input from all three and can redistribute resistance back to all three through drinking water, irrigation, recreational contact and the food chain. A review that stops at the hospital gate therefore risks describing only the first half of the AMR story. The

remainder of this paper is organised to follow that second half in order, from source, through wastewater, into rivers and sediments, and finally into the cross-sectoral surveillance systems that are meant to track the whole cycle but, as this review will show, presently do not.

II. MATERIALS AND METHODS

A. Search Strategy and Scope

This paper is a structured narrative review rather than a formal systematic review; it does not follow a PRISMA protocol with dual independent screening, and no review protocol was pre-registered. That distinction matters for how the findings should be read, and is stated here rather than left implicit. Within that scope, the search was carried out systematically. Google Scholar, PubMed and general web search were used to identify peer-reviewed studies and, where relevant, high-quality preprints and institutional reports published in English, using combinations of search terms structured around each chapter of the review: ‘antimicrobial resistance’ or ‘antibiotic resistance’ combined with ‘wastewater’, ‘river’, ‘surface water’, ‘sediment’, ‘One Health’ or ‘surveillance’, each further combined with ‘Africa’ or the name of an individual African country. Searches were run iteratively between mid-2025 and early 2026, with later

searches used to check whether earlier-identified preprints had since been published in peer-reviewed journals.

B. Study Selection

Studies were prioritised for inclusion if they reported primary data on antimicrobial-resistant bacteria, antimicrobial resistance genes, or antibiotic residues in an African wastewater, river, surface water or sediment sample, or if they addressed AMR surveillance capacity or policy at a regional or continental level relevant to interpreting that primary evidence. Purely clinical AMR studies with no environmental sampling component were excluded unless they were needed to establish One Health context. Where more recent or more geographically relevant studies were available on a given theme, they were prioritised over older or less specific sources, though older foundational studies were retained where no more recent equivalent existed. This selection approach means the 36 sources cited here are illustrative of the breadth and character of the literature rather than an exhaustive census of every environmental AMR study conducted in Africa; the true number of relevant primary studies is almost certainly larger, particularly in grey literature, theses and non-English-language regional journals that a English-language, database-driven search of this kind will systematically under-sample.

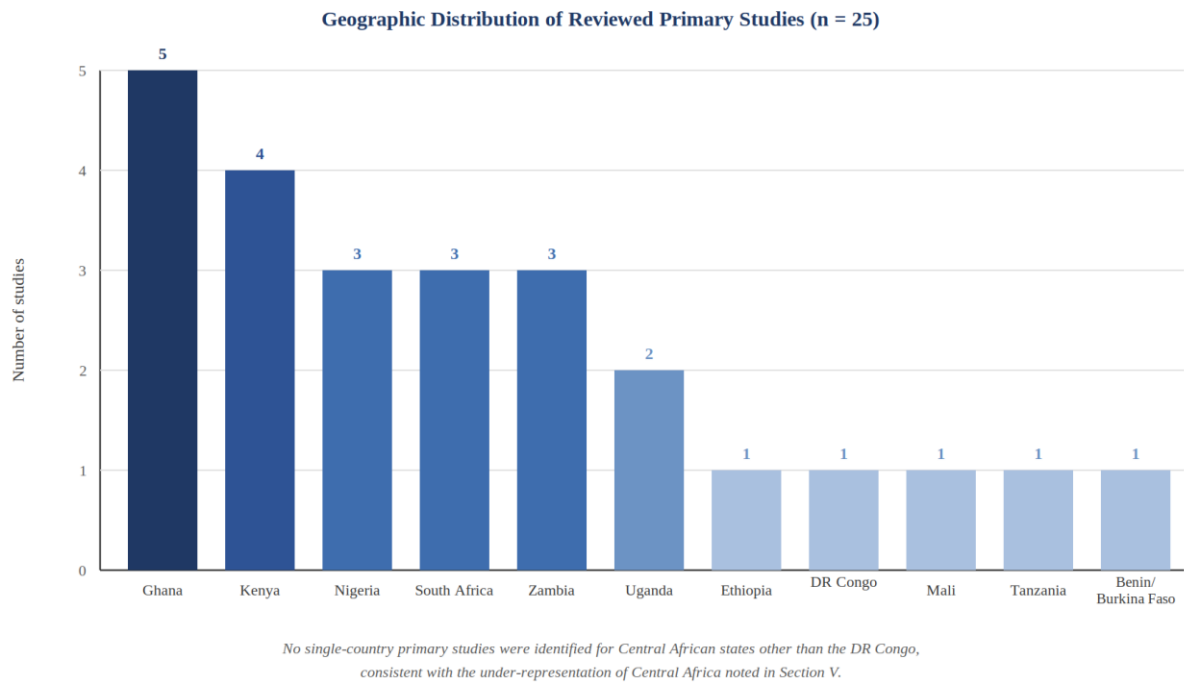


FIGURE 1. GEOGRAPHIC DISTRIBUTION OF THE PRIMARY STUDIES REVIEWED

The resulting evidence base, summarised in Figure 1, is concentrated in a relatively small number of countries with established environmental microbiology or genomics capacity, principally Ghana, Kenya, Nigeria, South Africa and Zambia, while large parts of the continent, and Central Africa in particular, are represented by at most a single

primary study or none at all. This imbalance is itself a finding of this review, not merely a limitation of the search: it mirrors, and likely partly explains, the laboratory-capacity gap discussed in Section V, since countries with more environmental AMR infrastructure naturally generate more publishable environmental AMR research.

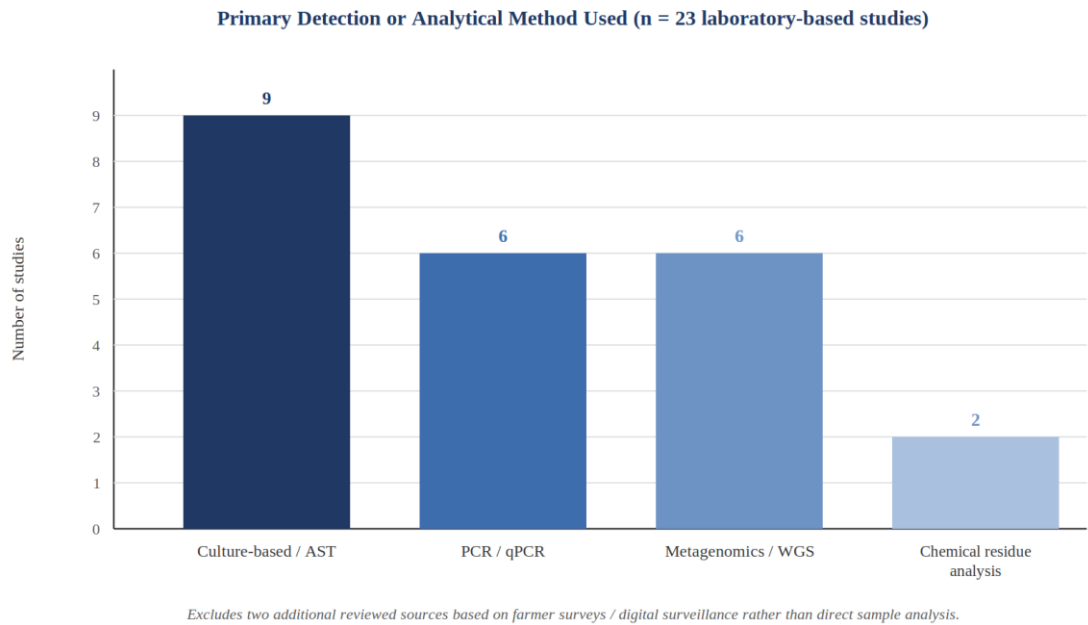


FIGURE 2. PRIMARY DETECTION OR ANALYTICAL METHOD USED ACROSS REVIEWED STUDIES

Figure 2 summarises the primary detection or analytical method used in each of the 23 reviewed studies that involved direct laboratory analysis of an environmental sample. Culture-based susceptibility testing remains the single most common approach, used in more studies than PCR-based and

sequencing-based methods combined. This distribution is returned to directly later in this review, where it is used to explain why studies using different methods are not always reporting genuinely different levels of contamination.

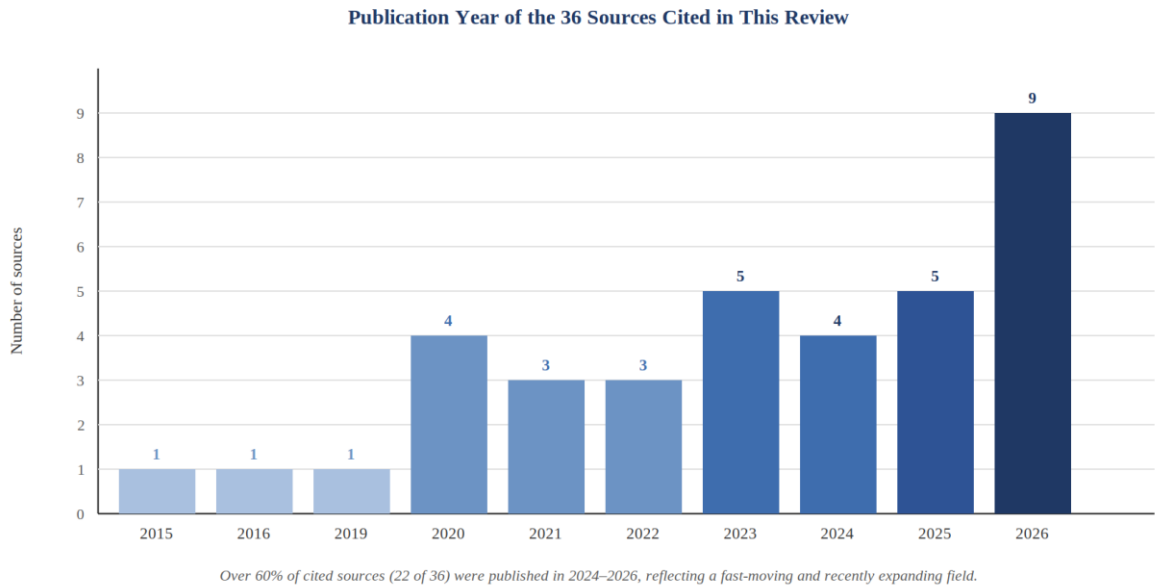


FIGURE 3. PUBLICATION YEAR OF THE 36 SOURCES CITED IN THIS REVIEW

Figure 3 shows the publication year of every source cited in this review. Over 60% (22 of 36) were published between 2024 and 2026, which reflects a field that has expanded rapidly in the last two to three years as sequencing costs have fallen and African genomics and environmental-microbiology capacity has grown. This recency is a strength of the evidence base assembled here, but it is also a caveat: much of the literature this review draws on is too recent to

have been independently replicated or extensively cited elsewhere, and conclusions that currently look consistent across studies may shift as this fast-moving field matures further.

III. WASTEWATER AS A RESERVOIR OF AMR

C. Sources of AMR in Wastewater

Wastewater accumulates resistant bacteria and resistance genes from several upstream sources before it ever reaches a treatment plant or a receiving river: hospitals, households, livestock and agriculture, and pharmaceutical or industrial activity. Antimicrobial use in food animals has been reported across most African countries surveyed [2], much of which is excreted and enters manure, runoff or wastewater. A study tracing *Salmonella* through Kampala, Uganda recovered the organism, resistant to multiple antimicrobials, from treatment-plant influent and effluent, waste stabilisation ponds, slaughterhouse flush-water, live-bird markets and a storm-water channel [4], a near-complete map of this source-to-wastewater chain. In Bamako, Mali, hospital wastewater discharged directly into the Niger River was identified as a key contributor of antibiotic residues to the river [5], while farmers interviewed along Tanzania's Msimbazi River basin pointed to manure use and uncontrolled drug disposal as the main perceived drivers of AMR spreading into their environment [6].

D. Resistant Bacteria and Resistance Genes

Once in wastewater, resistance is carried by resistant bacteria (ARB), the resistance genes (ARGs) they carry, and mobile genetic elements that let genes jump between species. In Mbarara, Uganda, quantitative PCR of hospital, municipal and treated wastewater detected six clinically relevant genes including the colistin-resistance gene *mcr-1*; treatment reduced overall bacterial counts substantially, yet the proportion of ESBL-producing *E. coli* did not fall significantly [7]. Shotgun metagenomic sequencing of hospital wastewater across nine hospitals in Benin and Burkina Faso found the carbapenemase gene *blaGES* to be the most widespread resistance determinant in Beninese samples, with *mcr* genes present across all sites studied [8]. A 2025 systematic review and meta-analysis focused specifically on carbapenemase genes in African hospital wastewater [9]. In Tamale, Ghana, combined Nanopore and Illumina sequencing found resistance genes and mobile genetic elements shared across the hospital–community interface, indicating active dissemination beyond hospital walls [10].

TABLE I. ENVIRONMENTAL AMR STUDIES IN AFRICAN WASTEWATER

Country	Wastewater source	Key ARB / ARG	Main finding
Uganda (Mbarara)	Hospital, municipal & treated	ESBL <i>E. coli</i> ; <i>mcr-1</i> , <i>sul1</i> , <i>blaTEM</i> , <i>blaOXA-48</i>	Treatment cut bacterial counts by 1.9–2.4 log, but ESBL proportion did not fall significantly [7].
Benin & Burkina Faso	Hospital (9 hospitals)	<i>mcr</i> genes; carbapenemase	<i>blaGES</i> Benin had the highest ARG abundance; <i>blaGES</i> was the dominant carbapenemase and reached hand-washing water [8].
Nigeria (Nsukka)	Treated sewage (irrigation reuse)	Enterotoxigenic & MDR <i>E. coli</i>	Treated sewage, irrigated soil and vegetables all carried resistant <i>E. coli</i> , unsafe for reuse [11].
Nigeria (Gombe)	Household, districts	7 <i>blaCTX-M</i> , <i>blaVIM</i> ; MDR <i>E. coli</i> , <i>Klebsiella</i>	MDR prevalence 60–96% across districts; <i>blaCTX-M</i> in nearly all ESBL isolates [12].
Nigeria (Abakaliki)	Tertiary hospital effluent	MDR <i>S. aureus</i> , <i>E. coli</i> , <i>Klebsiella</i> spp.	High multidrug resistance in untreated hospital effluent [13].
Ghana (Accra–Legon)	STP influent/effluent + stream	<i>E. coli</i> , <i>Aeromonas</i> , <i>Pseudomonas</i>	Plant cut bacterial counts >98%, yet resistance rose from upstream to outfall to downstream [14].
Ghana (Tamale)	Hospital & urban canalisations	Carbapenem/aminoglycoside genes, plasmids	Resistance genes and MGEs shared across the hospital–community interface [10].
Mali (Bamako)	Hospital → Niger River	Antibiotic residues (chemical)	Hospital effluent identified as a key antibiotic contributor to the river [5].
Kenya (multiple towns)	WWTP influent/effluent, surface water, sediment	Antibiotic residues	Surface water sometimes 2–5× higher in antibiotics than effluent; medium–high resistance-selection risk [15].

Note: sources are cited individually within each row above.

E. Why Findings Differ: Methodological and Contextual Heterogeneity

The studies summarised in Table I do not simply repeat one another with different place names; they differ in ways that plausibly explain much of the variation in reported

resistance levels, and a critical reading needs to hold these differences in view rather than treating every percentage as directly comparable. A first source of variation is detection method. Culture-based susceptibility testing, used for example in Ghana's drinking-water surveys [16], only detects

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bacteria that grow under the specific culture conditions chosen, and will systematically under-count viable-but-non-culturable organisms and genes carried on cell-free DNA. Quantitative PCR, as used in Mbarara, Uganda, targets a fixed panel of known genes and will miss anything outside that panel, however sensitive it is within it [7]. Metagenomic and whole-genome sequencing, used for the Benin/Burkina Faso and Accra studies, capture a much wider resistance-gene profile without needing culture at all [8,17], but are more expensive and remain concentrated in a handful of well-resourced labs. Comparing a culture-based prevalence figure from one country with a metagenomic abundance figure from another is therefore not a like-for-like comparison of how contaminated the two sites actually are; it is partly a comparison of what each method is capable of seeing.

A second source of variation is wastewater type and treatment status. Hospital effluent, sampled directly at source in Abakaliki, Nigeria [13] and across nine hospitals in Benin and Burkina Faso [8], concentrates antibiotic residues and resistant organisms far more than diluted municipal or household wastewater, so studies that sample at the hospital gate will tend to report higher resistance burdens than studies sampling downstream community wastewater almost regardless of location. Treated and untreated wastewater

differ for a related but distinct reason: treatment reduces total bacterial load, but several studies reviewed here found it does not proportionally reduce resistant organisms. In Mbarara, waste stabilisation ponds cut overall bacterial counts substantially while the proportion of ESBL-producing *E. coli* barely changed [7], and in Accra, a treatment plant removed more than 98% of bacteria yet resistance in the receiving stream rose rather than fell downstream [14]. This points to a further variable that is easy to overlook: the type of treatment infrastructure itself. The advanced, multi-stage plants studied in South Africa are not mechanically comparable to the waste stabilisation ponds used in Uganda and Ghana, which are not comparable again to the largely untreated hospital discharge reported in Mali and parts of Nigeria [5,13]; a review that treats ‘treated wastewater’ as a single category across these settings risks averaging over a genuinely important engineering difference. Finally, urban settings dominate this evidence base almost completely, every wastewater study reviewed here was conducted in a city or large town, so apparent country-level or regional patterns may in practice be urban-infrastructure patterns, and say comparatively little about rural on-site sanitation, which serves the majority of the region's population but is essentially unstudied for environmental AMR.

TABLE 2. ILLUSTRATIVE COMPARISON OF DETECTION METHODS USED ACROSS REVIEWED STUDIES

Method	What it detects	Key limitation	Relative cost / capacity need
Culture-based / AST	Viable, culturable resistant bacteria under chosen growth conditions	Misses viable-but-non-culturable organisms and cell-free DNA; results depend on media and incubation choices	Low cost; achievable in most existing clinical microbiology labs
PCR / qPCR	Presence and relative abundance of a pre-selected panel of known resistance genes	Cannot detect genes outside the chosen panel; panel design varies widely between research groups	Moderate cost; requires molecular biology capacity and reagent supply chains
Metagenomics / WGS	Full resistance-gene and mobile-genetic-element profile without prior culture	Expensive; requires bioinformatics expertise and computing infrastructure not evenly available	High cost; concentrated in a small number of well-resourced regional labs
Chemical residue analysis (LC-MS/MS)	Concentration of specific antibiotic compounds in water, not resistance itself	Indicates selective pressure, not actual resistance burden; residues can degrade before resistance does	High cost; requires analytical chemistry instrumentation rarely co-located with microbiology labs

Note. This comparison is illustrative rather than a formal technology assessment; costs and capacity requirements vary by country and by the specific assay or sequencing platform used.

Read alongside Figure 2, Table 2 helps explain a pattern that recurs throughout this review: the countries and studies using higher-resolution methods are not necessarily those with the worst underlying contamination; they may simply be the countries and research groups with access to the infrastructure those methods require. South Africa's prominent role in the metagenomic and extracellular-DNA literature reviewed here [18,19], for instance, likely reflects that country's comparatively well-established genomics

infrastructure at least as much as it reflects any underlying difference in contamination relative to countries represented only by culture-based studies.

F. Antibiotic Classes and Resistance Mechanisms Reported

Beyond method and source, the studies reviewed here also differ in which antibiotic classes and resistance mechanisms they were designed to detect, which further complicates any simple ranking of ‘how resistant’ a given site or country appears. Figure 4 summarises the resistance-gene families

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reported across the wastewater and aquatic-environment studies in Tables I and II. Beta-lactamase genes, the family that includes blaCTX-M, blaTEM, blaOXA-type and blaVIM enzymes, and confers resistance to penicillins and cephalosporins, are by far the most frequently reported group, appearing in eight of the studies summarised. This is unsurprising given that beta-lactams remain among the most

widely prescribed antibiotic classes in both human and veterinary medicine across the region, and that ESBL-screening protocols are comparatively inexpensive and well established in African clinical microbiology, so laboratories already equipped for ESBL surveillance can extend the same assays to environmental samples with little additional investment.

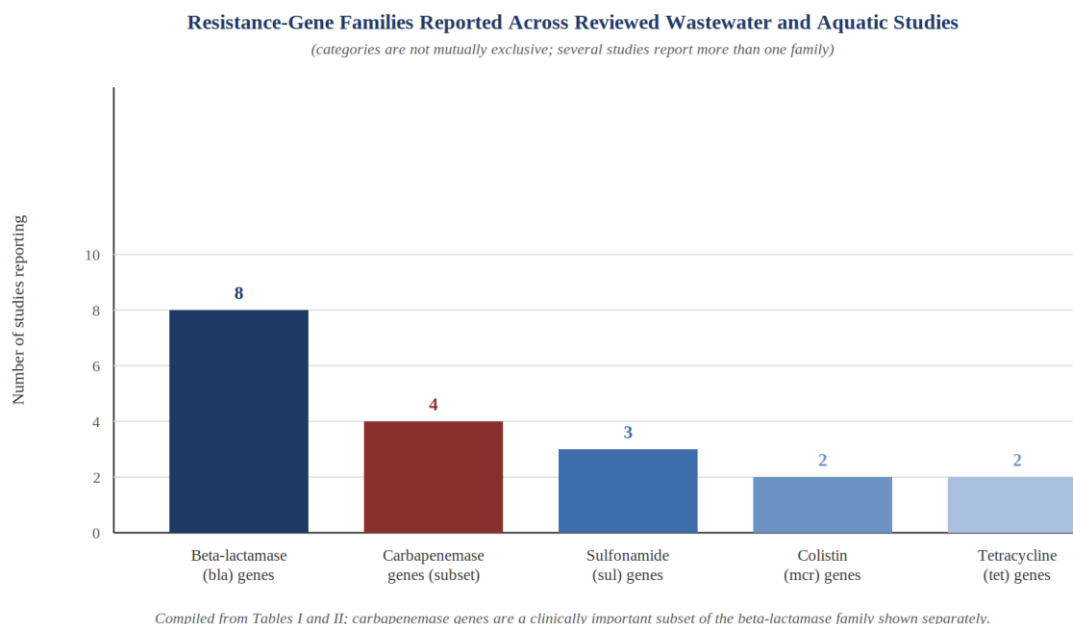


FIGURE 4. RESISTANCE-GENE FAMILIES REPORTED ACROSS REVIEWED STUDIES

Carbapenemase genes, a clinically critical subset of the beta-lactamase family, since carbapenems are typically reserved as last-resort agents for infections resistant to other beta-lactams, were reported in four studies, including the dedicated meta-analysis of African hospital wastewater discussed above [9]. Their presence in environmental samples at all is arguably more clinically alarming than the raw frequency suggests, since it indicates that resistance to antibiotics of last resort is not confined to hospital patients but is already circulating in wastewater and, in the Kinshasa and Tamale studies, in receiving rivers and community-facing wastewater networks [10,20]. Sulfonamide resistance genes (sul1, sul2, sul3) and tetracycline resistance genes (tet variants) were each reported in a smaller number of studies, concentrated in the South African and Democratic Republic of Congo river studies [19,20], which reflects both the older, cheaper class of antibiotics these genes correspond to and the qPCR-panel design choices of the specific research groups working in those countries, rather than necessarily reflecting an absence of sulfonamide or tetracycline resistance elsewhere. Colistin-resistance genes (mcr) were reported in only two studies, in Uganda and in the Benin/Burkina Faso metagenomic survey [7,8]; given that colistin is a last-resort antibiotic for multidrug-resistant Gram-negative infections, this low reported frequency should be read with particular caution, it may reflect genuine rarity, or it may simply reflect

that most of the studies in this review did not specifically screen for mcr genes in the first place, since targeted PCR panels only report what they were designed to look for.

IV. AQUATIC ENVIRONMENTS AND AMR DISSEMINATION

G. Rivers and Surface Water

Once discharged, wastewater carries resistant bacteria and genes into receiving rivers, and resistance often rises rather than falls moving downstream. Along River Sosiani in Eldoret, Kenya, sampling from upstream through town to downstream of several treatment plants found a high multi-antibiotic resistance index [21]. In Accra, Ghana, a treatment plant cut effluent bacterial counts by more than 98%, yet resistance to several antibiotics rose progressively from upstream to outfall to downstream in the receiving stream [14]. Whole-genome sequencing of multidrug-resistant *E. coli* from Accra's surface waters revealed diverse beta-lactamase genes and mobile genetic elements [17], while single-cell genomic profiling confirmed Ghana's Densu River as a natural AMR reservoir [22]. In Kenya, shotgun metagenomics of the Winam Gulf of Lake Victoria, a primary drinking-water and fish source, detected genes conferring resistance to beta-lactams and vancomycin [23].

H. Sediments and Aquatic Microbial Communities

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Sediments can act as longer-term reservoirs than the water column itself. Along Ethiopia's Akaki River, a panel of 84 targeted ARGs was assessed in water and sediment across five sites; water samples in the middle catchment carried 71–75% of targeted genes, while sediment-associated ARG data remained comparatively limited [24]. In Kinshasa, DR Congo, sediment sampled both downstream and upstream of rivers receiving animal-farm wastewater carried resistance genes including blaOXA-48 and blaCTX-M, showing farm effluent was not the sole contamination source [20]. In Durban, South Africa, sulfonamide and tetracycline resistance genes were detected in both culturable and non-culturable bacterial fractions of rivers and estuary, suggesting a hidden reservoir culture-based methods alone would miss [19]. A broader study across nine South African treatment plants found extracellular DNA stabilised high-risk, mobile ARGs enriched in effluent and downstream river stretches [18].

I. Environmental Factors

Wastewater discharge volume and treatment efficiency set the baseline resistance load entering a river, while broader pollution and nutrient enrichment add to that load independently of any single point source. In Kenya, chemical analysis across several treatment plants found antibiotic concentrations in surface water sometimes two to five times higher than in the corresponding effluent, with risk-quotient modelling indicating medium-to-high risk of resistance selection in both [15].

J. Explaining the Variability: What the Comparisons Across Studies Actually Show

Three further comparisons help explain why the aquatic-environment findings above do not all point in the same direction, and each carries a methodological caveat worth stating explicitly rather than leaving implicit. The first is water versus sediment. The Akaki River study is often read as showing that water carries more resistance genes than sediment, since targeted water samples recovered 71–75% of the ARG panel while sediment recovery was comparatively limited [24]. That may reflect a genuine biological difference, water samples integrate contamination from a larger catchment area and turn over faster, while sediment retains a smaller, more localised signal, but it may equally reflect that fewer extraction and amplification protocols in this literature have been optimised for sediment matrices, which contain more PCR-inhibiting organic material than water. The Kinshasa study, which found resistance genes in sediment both upstream and downstream of an animal-farm outlet,

suggests sediment is far from an inert reservoir once appropriately sampled [20]; the two findings are not necessarily in tension, but a review that reported only the Akaki result would understate how much sediment matters.

The second is the disconnect between antibiotic residues and ARG abundance, which are frequently treated as interchangeable evidence of contamination but measure different things. The Kenyan wastewater study reviewed here measured chemical antibiotic concentrations directly, using these to model resistance-selection risk rather than measuring resistance itself [15], whereas most other studies in this review measured ARGs or resistant bacteria without paired chemical data. High antibiotic residues indicate selective pressure is present, but resistance can persist in an environment long after residues have degraded or diluted below detection, particularly where resistance genes are linked to other, unrelated selective advantages on the same mobile genetic element. A site with low measured antibiotic concentrations but high ARG abundance, or the reverse, is therefore not necessarily a contradictory finding; it may simply reflect that the two measurements capture different stages of the same contamination process, and few studies in this literature measure both simultaneously at the same site and time.

The third is sampling design. Nearly every study reviewed in this paper relies on grab sampling at a single or a small number of time points, which cannot capture seasonal variation in rainfall, dilution, and temperature that is known elsewhere to affect both antibiotic persistence and bacterial survival; a river sampled during the dry season, when flow is low and contamination is concentrated, would be expected to show a different resistance profile than the same river sampled during heavy seasonal rains, independent of any underlying change in contamination sources. None of the aquatic-environment studies reviewed here report repeated seasonal sampling at the same sites, so the field currently cannot distinguish a genuine site-to-site difference in contamination from a difference that simply reflects when in the year each study happened to sample. Taken together, these three issues, water versus sediment recovery, residues versus gene abundance, and single-timepoint versus seasonal sampling, mean that the cross-country comparisons elsewhere in this review should be read as showing a consistent overall pattern of environmental AMR contamination across the continent, rather than as precisely ranking which country or site is 'worse' than another.

TABLE 3. AMR IN AFRICAN AQUATIC ENVIRONMENTS

Country	Environment	Key ARB / ARG	Main finding
Kenya (Eldoret)	River Sosiani	MDR Gram-negative bacteria	High multi-antibiotic resistance index across upstream/downstream sites near WWTPs [21].

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Ghana (Accra)	Urban surface water	MDR E. coli; beta-lactamase genes	WGS revealed diverse resistance genes, sequence types and MGEs [17].
Ghana (Densu River)	River water	AMR-carrying E. coli	Single-cell genomics confirmed the river as a natural AMR reservoir [22].
Ethiopia (Addis Ababa)	Akaki River, water & sediment	84 targeted ARGs (blaOXA-10, qnrS, etc.)	Water in the mid-catchment carried 71–75% of targeted genes [24].
South Africa (9 WWTPs)	Effluent + receiving rivers	High-risk mobile (extracellular DNA)	ARGs enriched in effluent and further downstream [18].
South Africa (Durban)	River, estuary, STP effluent	sul1, sul2, sul3, tet(M)	Genes present in non-culturable as well as culturable fractions [19].
DR (Kinshasa)	Congo Rivers near animal farms	blaOXA-48, sul1-3, tet(B)	Resistance genes present upstream as well as downstream of the farm outlet [20].
Kenya (Lake Victoria)	(Lake Winam Gulf lake water	Beta-lactamase & vancomycin-resistance genes	Lake is a primary drinking-water/fishery source for surrounding communities [23].

Sources: compiled from references cited in each row above.

V. ONE HEALTH CONNECTION AND ENVIRONMENTAL SURVEILLANCE

K. Human–Animal–Environment Connection

The One Health concept frames human, animal and environmental health as interconnected compartments through which resistance can move in either direction [25]. Antimicrobial use in food-producing animals is reported by the large majority of African farms surveyed, poultry most commonly [2]. Among broiler poultry farm workers in Lusaka, Zambia, E. coli from stool samples showed high resistance to tetracycline, trimethoprim-sulfamethoxazole and ampicillin, with a minority of isolates confirmed as ESBL-producers [26]. A related study of layer poultry across Zambia found similarly high resistance rates in apparently healthy laying hens [27,28]. Despite this accumulating compartment-level evidence, a systematic review of African studies examining two or more compartments simultaneously found genuinely integrated evidence to still be scarce [3].

The mechanism connecting these compartments is worth making explicit rather than leaving implied by Figure 1 alone. Poultry, fish and livestock raised in or near contaminated water can carry resistant organisms into the food chain directly; workers and residents in close daily contact with farm effluent or wastewater-irrigated produce face a further, occupational and dietary exposure route documented in the Zambian poultry-worker study above [26]; and household members who never set foot on a farm can still be exposed indirectly through drinking water drawn from a contaminated river or shallow well downstream. Each of these routes has been documented individually somewhere in the literature reviewed here, but almost never for the same population at the same time, which is precisely the integrated, multi-compartment study design that Escher and colleagues found to be lacking across the region [3]. Until such studies exist, it remains difficult to say with confidence which of these three exposure routes contributes most to human resistant-infection

risk in any given African setting, even though the environmental reservoir feeding all three is now reasonably well documented in isolation.

L. Current Environmental AMR Surveillance

Four broad approaches recur across the literature: culture-based susceptibility testing (used from Ghana's drinking-water sources [16] to Zambia's poultry farms [26]); PCR/qPCR for targeted genes (Uganda's wastewater [7], South African rivers [19]); metagenomic sequencing (Benin/Burkina Faso [8], Lake Victoria [23], South African extracellular DNA [18]); and whole-genome sequencing of isolates (Accra [17], Tamale [10]). A 2015–2025 scoping review of genomic AMR studies across Africa found that while genomic capacity has expanded meaningfully, significant gaps persist in veterinary and environmental laboratory capacity specifically [29]. Kenya's One Health AMR Surveillance System (OHAMRS) already integrates human and animal AMR data through a shared DHIS2-based dashboard and has been designed to be scalable to future inclusion of environmental, food and aquaculture data [30], that environmental data is not yet part of even one of the continent's more advanced integrated systems is itself a useful marker of where the field currently stands.

It is worth being specific about what 'integrating environmental data' into a system like OHAMRS would actually require, since the gap is often stated as a goal without the underlying technical obstacles being named. Human and animal AMR surveillance typically reports a manageable number of standardised data points per case, organism, specimen type, resistance profile against a defined antibiotic panel, collected through clinical or veterinary laboratory workflows that already feed national reporting systems. Environmental data, by contrast, arrives in far more heterogeneous forms, as this review's own Figure 3 illustrates: a culture-based prevalence percentage, a qPCR gene-copy-

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number result, or a metagenomic relative-abundance profile are not natively compatible data types, and no African One Health platform reviewed here has yet published a data model for reconciling them. Building that data model, rather than simply agreeing in principle that environmental data should

be included, is the concrete technical task standing between the current human–animal integration achieved in Kenya and the fully cross-sectoral system envisioned in national AMR action plans across the region.

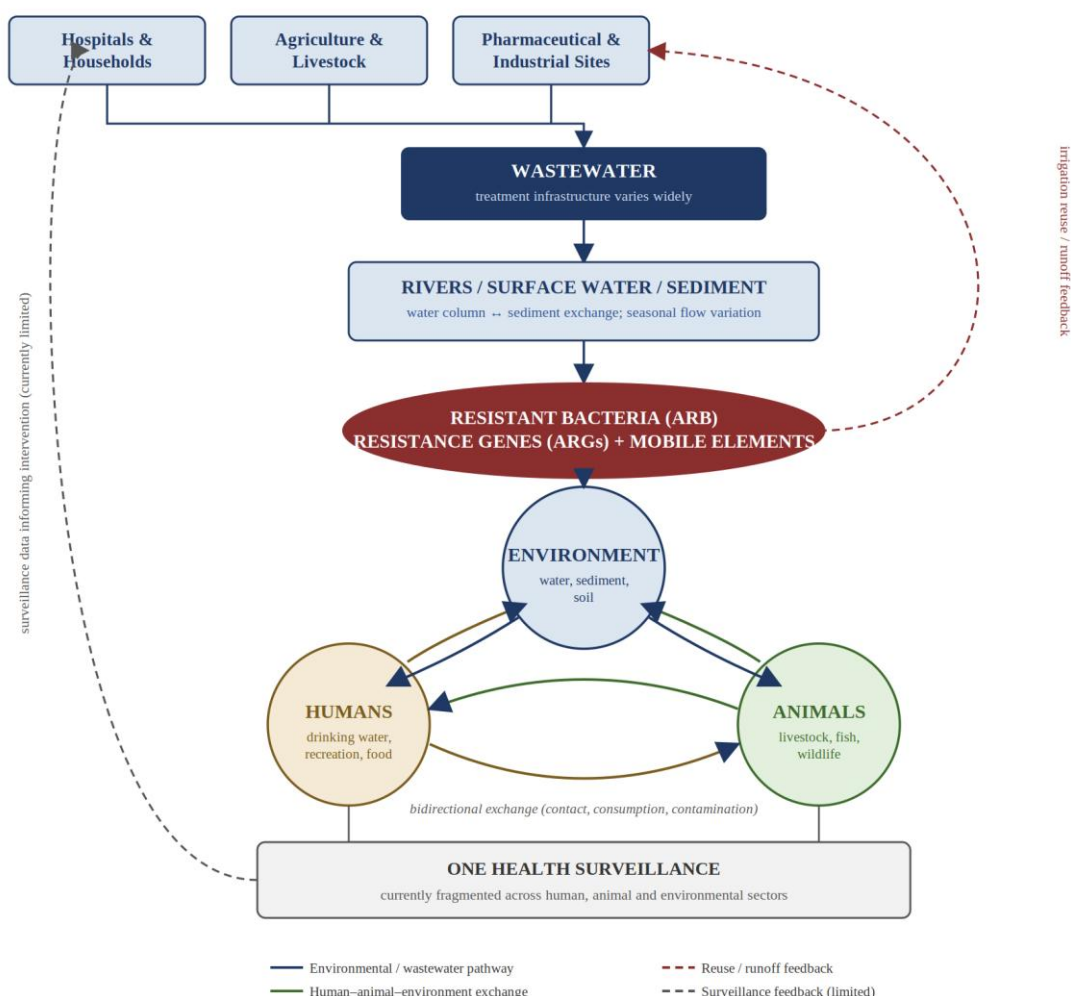


FIGURE 5. CONCEPTUAL MODEL OF ENVIRONMENTAL AMR DISSEMINATION, ONE HEALTH EXCHANGE, AND FEEDBACK PATHWAYS

Note. Solid navy arrows trace the wastewater-to-environment pathway from sources through treatment to receiving waters. Solid gold and green arrows show bidirectional exchange within the human–animal–environment triangle (contact, consumption, contamination). Dashed arrows show two feedback loops that are frequently omitted from simpler schematics: reuse of contaminated water for irrigation or runoff back toward source compartments, and the (currently limited) flow of surveillance data back into intervention.

M. Policy and Practice Implications

The pattern shown in Figure 1 has direct practical implications that go beyond the academic observation that compartments are connected. First, because treatment consistently reduced bacterial counts without proportionally reducing resistant fractions in every treated-wastewater study reviewed here [7,14], investment decisions that measure success purely in terms of total coliform or *E. coli* reduction may be missing the resistance dimension of water quality entirely; national and municipal wastewater standards in the region do not currently include any resistance-specific target, and this review suggests they arguably should. Second, the

repeated finding that hospital effluent is a concentrated source of both resistant organisms and antibiotic residues [5,8,13] points toward a relatively low-cost, high-yield intervention: on-site pre-treatment or disinfection of hospital wastewater before it enters the municipal system, which several of the studies reviewed here explicitly recommend but which is not yet standard practice at most of the facilities studied. Third, the irrigation-reuse pathway shown as a feedback loop in Figure 1 is not hypothetical: the Nsukka, Nigeria study found treated sewage used for vegetable irrigation to be an active reservoir of resistant *E. coli* on the vegetables themselves [11], meaning that wastewater-reuse

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guidance for urban and peri-urban agriculture, an increasingly common practice in water-scarce African cities, needs to incorporate resistance testing rather than conventional faecal-indicator testing alone.

VI. SURVEILLANCE GAPS AND FUTURE DIRECTIONS

N. Major Surveillance Gaps

Six gaps recur across the African environmental AMR literature reviewed here: limited environmental and veterinary laboratory capacity relative to clinical microbiology [29,31]; fragmented, sector-siloed data systems, even in relatively advanced platforms such as Kenya's OHAMRS [30]; inconsistent sampling and detection methods across studies, which complicates cross-country comparison [3]; genomic and metagenomic surveillance still concentrated in a handful of countries and labs [32,33]; inconsistent national reporting to global surveillance systems [34]; and a scarcity of long-term monitoring, since nearly every study reviewed here, from Eldoret's River Sosiani [21] to Ethiopia's Akaki River [24], represents a single cross-sectional snapshot rather than sustained monitoring.

O. Future Directions

Closing these gaps points toward five priorities: extending integrated One Health platforms such as Kenya's OHAMRS to formally include environmental and wastewater data [30]; adopting standardised environmental sampling protocols to make cross-country comparison meaningful; scaling up molecular and genomic approaches through coordinated regional efforts such as the Africa CDC's Pathogen Genomics Initiative [35] and the SeqAfrica network's regional sequencing hubs [34]; extending wastewater-based surveillance, already demonstrated as feasible for tuberculosis resistance monitoring in South Africa [36] and community-level AMR monitoring in Uganda [7] and Nigeria [12]; and strengthening collaboration between human, animal and environmental health sectors, informed by the poultry-farm evidence from Zambia and Tanzania [6,26].

Translating these priorities into practice requires attention to research design as much as to infrastructure funding. Based on the heterogeneity issues identified throughout this review, future primary studies would add the most value to the evidence base if they: (i) paired at least two detection

methods at the same site and time point, for example culture-based screening alongside targeted qPCR, so that method-driven differences can be distinguished from genuine site differences; (ii) sampled the same wastewater or river site repeatedly across at least one full wet–dry seasonal cycle, rather than relying on a single grab sample, given that no aquatic-environment study reviewed here reported repeated seasonal sampling; (iii) reported paired sediment and water-column data wherever feasible, rather than one matrix alone, to help resolve whether the apparent water–sediment differences seen in the Akaki River data [24] reflect biology or methodology; and (iv) explicitly distinguished hospital, municipal and household wastewater sources within the same study design, rather than sampling only one source type, so that source-driven differences in resistance burden can be separated from genuine geographic differences. None of these changes requires substantially more advanced technology than is already used somewhere in the studies reviewed here; they require research designs that build comparison into the study from the outset, rather than relying on readers to compare across differently-designed studies after the fact, which is precisely the exercise this review has had to undertake.

Funding and capacity-building efforts should also be matched to where this review found the evidence base to be thinnest. Figure 2 shows that Central, Francophone and Lusophone Africa are severely under-represented relative to Anglophone West, East and Southern Africa; targeted funding calls and regional training partnerships, for example through the Africa CDC's Pathogen Genomics Initiative working with regional economic communities rather than only with individual national reference laboratories [35], could do more to close this geographic gap than continued investment concentrated in the handful of countries already generating most of the evidence. Similarly, because rural on-site sanitation was not represented in any study reviewed here, funders and national AMR action plans should consider ring-fencing a specific share of environmental AMR surveillance funding for rural and peri-urban settings, rather than allowing urban wastewater systems to continue absorbing the great majority of research attention simply because they are logistically easier to sample.

TABLE 4 MAJOR SURVEILLANCE GAPS AND PRIORITIES

Current gap	Why it matters	Recommended approach
Limited environmental & veterinary lab capacity	Most functioning microbiology capacity is clinical, not environmental	Invest in regional environmental-microbiology and sequencing hubs (e.g., SeqAfrica)
Fragmented, sector-siloed data systems	Human, animal and environmental AMR data are rarely integrated	Build interoperable One Health platforms, extending models such as Kenya's OHAMRS
Inconsistent sampling & methods	Limits cross-country and cross-study comparison	Adopt harmonised protocols (WHO GLASS/Tricycle, FAO InFARM)

Low genomic/metagenomic uptake	Surveillance concentrated in a few countries and labs	Expand WGS/metagenomics via the Africa CDC Pathogen Genomics Initiative
Limited long-term monitoring	Most studies are single cross-sectional snapshots	Establish sentinel wastewater/river monitoring sites with sustained funding
Under-studied regions & compartments	Central Africa, rural areas and aquaculture are under-represented	Prioritise funding and sampling toward under-represented regions and sectors

P. Limitations of This Review

This review has its own limitations, which follow directly from the search approach described in the Materials and Methods section and should be weighed alongside its conclusions. It is a structured narrative review rather than a formal systematic review; sources were identified through iterative, English-language database and web searching rather than a pre-registered, dual-screened protocol, and so it cannot claim to be an exhaustive census of the literature. The geographic concentration shown in Figure 1 may partly reflect the search strategy itself, English-language database searches will preferentially surface research from countries with stronger English-language publishing traditions and better-indexed national journals, although this is unlikely to fully explain the pattern, since it is consistent with independent reports of laboratory and genomic capacity concentration discussed in Section V. Grey literature, national reports, theses and non-English-language regional publications were not systematically searched and almost certainly contain additional relevant studies, particularly from Francophone and Lusophone African countries, which are visibly under-represented in Figure 1 relative to their population and geographic extent. Finally, because the review draws on studies using different detection methods, sampling designs and reporting conventions, as discussed at length earlier in the methodological-heterogeneity discussions of Sections II and III, the quantitative comparisons offered here, including the resistance-gene tallies in Figure 4, should be read as indicative of broad patterns across the literature rather than as a precise, standardised measurement of relative resistance burden between countries.

CONCLUSION

The evidence reviewed here traces a consistent chain: wastewater from hospitals, households, agriculture and livestock accumulates resistant bacteria and resistance genes; that wastewater, even after treatment, discharges into rivers where resistance levels often rise rather than fall; sediments and aquatic microbial communities then act as longer-term reservoirs; and because these environmental reservoirs connect directly back to human and animal populations, a One Health approach is necessary to understand and manage them. Closing the surveillance gap, through standardised sampling, expanded genomic capacity, and genuinely integrated cross-sectoral data systems, is the central task

facing environmental AMR research and policy in Africa going forward.

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