

Antimicrobial Resistance in a Changing Climate: Evidence, Causal Pathways, and Prevention Strategies

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Abstract

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Background: Antimicrobial resistance (AMR) and climate change are both significant global health threats increasingly recognized as mechanistically interconnected. This review critically synthesises evidence on the potential for climate-related hazards (temperature, heatwaves, flooding and drought) to alter and intensify AMR via biological, environmental and health-system pathways, and evaluates the causal strength and implications for prevention.

Methods: This critical narrative review involved a structured literature-identification and updating process, critically appraising systematic and scoping reviews, primary ecological and genomic studies, modelling analyses, and authoritative One Health reports through August 2026, organized by hazard and pathway rather than paper by paper.

Results: Association of temperature with AMR is one of the most consistent and replicated findings across

ecological studies in the United States, Europe and China, and is supported by a large multi-country genomic analysis of Salmonella resistance genes. This relationship is not generalizable: at least one adjusted analysis found no significant temperature-resistance

association, highlighting the organism- and context-specific nature of the relationship. Resistant pathogens and resistance genes are mobilized in the environment during flooding, but there is limited direct evidence of this leading to increased clinical infection. Ecological study design, residual confounding and limited data availability in South Asia, including Pakistan, limit causal inference across hazards. Evidence assessing climate-specific AMR prevention interventions remains limited.

Conclusions: Climate change must be considered as a potential contributing and amplifying factor for AMR, rather than a proven independent cause. Climate-informed surveillance is a priority for LMICs, including Pakistan, for One Health

AMR strategies.

Introduction

Antimicrobial resistance (AMR) is one of the greatest public health challenges in the 21st century. The most recent estimate puts the number of deaths directly caused by bacterial AMR at

1.14 million in 2021, and estimates that 4.71 million deaths are linked to resistant infections, with a projected 39 million deaths from 2025 to 2050 if action is not taken [1,2]. The drivers of AMR are well known and not controversial: overuse and misuse of antimicrobials in human medicine, veterinary medicine and agriculture are the most important selective pressures for the emergence and spread of antimicrobial resistance [3, 4]. More recently, climate change has been suggested as a further factor that could alter and intensify this pressure, but not as a direct driver. The United Nations Environment Programme has described AMR as part of a “triple planetary crisis” of climate change, pollution and biodiversity loss, where climate change and extreme

weather events, combined with other drivers of resistance, are expected to increase resistance [4]. There are plausible but distinct mechanistic pathways for each of the major hazards discussed here that lead to AMR: rising temperature, heatwaves, flooding, drought/water scarcity, and coastal change. This evidence base has been started to be mapped in prior reviews, such as van Bavel et al. [5] who performed a systematic scoping review, which identified 1,687 records and extracted data from 574, finding that the evidence base is “nascent” and fragmented, with gaps in hazards beyond temperature and resistance among fungi, viruses, protists, and helminths. This is further confirmed by subsequent reviews [6, 7] which document more and more links between climate variables and AMR, but do not clarify whether these are true causal links or confounded co-trends. Two gaps are especially noteworthy and are the basis of this review. First, the causal role of individual climate hazards in AMR is not clear and seems to vary depending on context and organism, rather than being a consistent and generalizable relationship. Second, evidence assessing interventions specifically developed to prevent or mitigate climate-associated AMR (as opposed to AMR-reduction interventions in general) is limited, and this is especially true for LMICs, including Pakistan and the broader South Asian region, which are repeatedly identified as having some of the highest future AMR burdens [8]. The purpose of this review is thus to critically synthesize evidence on pathways between climate-related hazards and AMR, evaluate the strength of the evidence in relation to causal-inference criteria, and identify priorities for prevention and research in LMIC and South Asian settings. Four questions guide this synthesis: (RQ1) What mechanisms link climate-related hazards with AMR emergence and dissemination? (RQ2) How strong and consistent is the evidence for the association between specific hazards and AMR? (RQ3) How strong is the evidence for a causal contribution of climate change to AMR and what are the key uncertainties? (RQ4) What are the plausible One Health strategies to mitigate AMR risk in the context of climate change and what are the gaps for LMICs and South Asia? This review builds on previous research that rates the causal strength hazard-by-hazard for South Asian contexts.

Methods

This is an important narrative review based on a structured process of identifying and updating the literature, not a systematic review or meta-analysis. The goal was not to comprehensively list all available literature, which has already been done in great detail in the 2024 systematic scoping review by van Bavel et al. [5] but to critically synthesize and update the evidence base, focusing on causal strength and

prevention-relevant findings by 2026.

Literature was identified by systematically reviewing and scoping the existing evidence base, followed by targeted updating and verification of relevant primary studies, modelling analyses, intervention studies, and authoritative One Health and public-health documents published up to August 2026. Combinations of antimicrobial-resistance terms (e.g., “antimicrobial resistance,” “antibiotic resistance,” “resistance genes,” and “ARG”) and climate-related terms (e.g., “climate change,” “temperature,” “heatwave,” “flood,” “drought,” and “extreme weather”) were used in the search concepts for updating. Peer-reviewed primary studies, systematic and scoping reviews, meta-analysis and authoritative reports from WHO, UNEP, FAO, WOH and the United Nations were prioritized. Sources were excluded if they were not substantive to a review question, or if bibliographic information could not be confirmed. We extracted the following information for each included source: author/year, geographic setting, study design, climate hazard examined, AMR outcome, main finding, and stated limitations, and summarized this evidence by hazard and pathway, not by paper-by-paper (summarized in Tables 1 and 2; the overall conceptual model is presented in Figure 1). We then used an explicit qualitative approach to evaluating the strength of the evidence and causal confidence, focusing on ecological-study limitations, direction of temporality, dose–response consistency, organism-specificity, and the separation of environmental detection of resistance determinants from clinically confirmed human infection. We stress that this process is not a systematic review or a meta-analysis, but rather critical narrative synthesis, and no formal risk-of-bias scoring tool or PRISMA flow diagram of screened/excluded records was used. Literature organization and drafting of the manuscript was aided by Generative AI (Claude, Anthropic) and all AI-generated content was reviewed and verified by the authors (see AI-Use Disclosure).

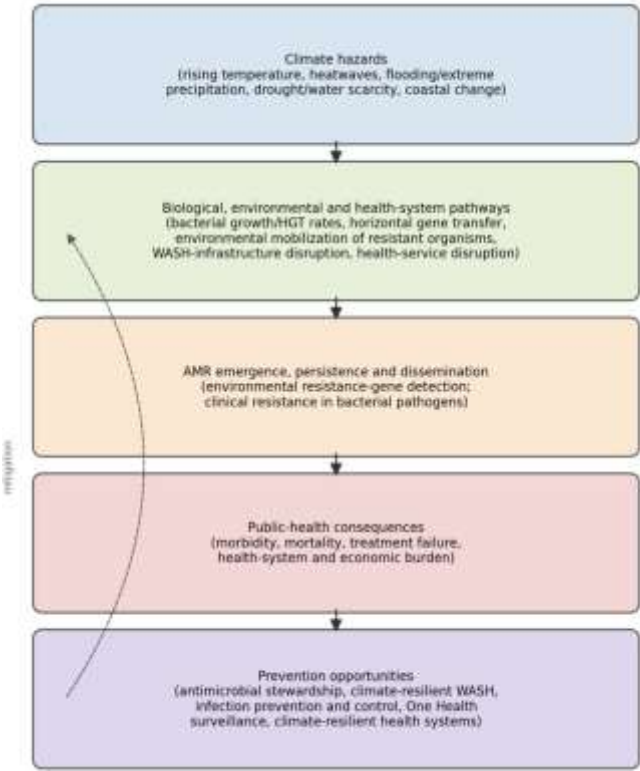


Figure 1. Conceptual framework that connects climate hazards, biological/environmental/health-system pathways, AMR emergence and

dissemination, public-health consequences, and prevention opportunities as summarized in Sections 3–5 of this review. Climate-Related Pathways Linking Climate Hazards and AMR

Temperature and heat

Mechanism. The following plausible biological pathways are suggested to explain how elevated ambient and environmental temperature may affect AMR: higher rates of bacterial growth and replication, higher mutation rates, and higher horizontal gene transfer (HGT) of resistance determinants between bacteria [3]. This relationship seems not to be linear, however. Zhao et al. [9] found that *Escherichia coli* showed a nonlinear temperature–resistance relationship, with maximum resistance at approximately 27°C, suggesting that the activation of efflux pumps and mutations in stress-response genes (*gyrA* and *marR*) were responsible for the resistance, not a simple “hotter equals more resistant” trend.

Empirical evidence. The temperature–AMR hypothesis was first proposed by MacFadden et al.

[10] in an ecological study in the USA, where they observed increases in resistance of 4.2%, 2.2%, and 2.7% for *E. coli*, *Klebsiella pneumoniae*, and *Staphylococcus aureus*, respectively, with a 10°C rise in local minimum temperature. This was independently replicated in a 28-country European analysis [11] and in a difference-in-differences design across 31 Chinese provinces by Zeng et al.

[12] who reported an increase of 4.7% and 10.7% in the prevalence of third generation cephalosporin resistant *K. pneumoniae* and carbapenem resistant *K. pneumoniae* respectively for every 1°C increase in annual mean temperature. This quasi-experimental design provides more temporal evidence than a correlational analysis, but not proof of causality. Zhou et al. [13] conducted a large-scale, longitudinal genomic and modelling study of 488,232 *Salmonella* genomes from 139 countries from 1940 to 2023, which revealed that climate change is linked to a 10% global rise in the abundance of antimicrobial resistance gene (ARG) in a non-linear fashion with temperature and precipitation. This estimate remained after accounting for measured antibiotic-use and socioeconomic covariates, but does not reflect a direct causal effect of the climate change, and abundance of ARG is a genomic indicator of resistance potential, not necessarily of actual resistance or clinical infection. Heatwaves are periods of extreme heat, and are biologically and epidemiologically different from a gradual warming. They can have indirect impacts on AMR by altering pathogen growth conditions, human susceptibility, health service demand and disruption of health services. But there are fewer studies of heatwave exposure and AMR outcomes than those of average temperature

Counter-evidence and context-dependence. Not every evidence is consistent with a temperature–AMR relationship. The study of distributed lag non-linear models in Queensland, Australia, did not show a significant relationship between temperature and resistance in *Salmonella* bacteraemia after adjusting for precipitation, seasonality and temporal trends [14] which is contrary to a simple extrapolation from the *E. coli*/*Klebsiella* literature. This is proof that the temperature–AMR relationship is not a universal biological law, but rather organism-specific and context-dependent. The heatwave pathway and the overall temperature–AMR relationship should be considered as hypothesis-generating and as priorities for longitudinal studies in exposed areas like South Asia.

Flooding and extreme precipitation

Mechanism. Flooding is thought to spread resistant bacteria and resistance genes in a number of ways: overwhelming of sewage and wastewater infrastructure,

mobilization of agricultural runoff (animal waste and remaining antimicrobials) into surface and groundwater, and disruption of water treatment systems [15].

Empirical evidence. Direct molecular and metagenomic evidence supports this pathway. In the aftermath of the devastating floods in 2024 in Bangladesh, shotgun metagenomic analysis of flood water samples from four districts revealed the presence of multidrug-resistant *K. pneumoniae*, toxigenic *Pseudomonas aeruginosa* and mobilizable resistance plasmids (IncP1 and Col440I) [16]. The floods in May 2024 in Rio Grande do Sul, Brazil, impacted more than 95% of the municipalities and an area of approximately 3,800 km², as described by Furlan et al. [15]. In the same event, Cardoso et al. [17] used qPCR to analyze 79 samples of flood water from Porto Alegre and found Enterobacteriaceae in all samples, including the presence of blaKPC in up to 100% of the pools and blaNDM in up to 89% of the pools. This pathway is not limited to LMIC settings, as a parallel study of urban flooding in Wrocław, Poland [18] reported changes in the bacterial community structure.

Critical caveat. It is important to differentiate between the detection of resistant organisms or resistance genes in flood water and actual increases in human clinical infection. The above studies have reported the presence of resistance determinants in the environment, but none have directly measured an increase in resistant clinical infections in the exposed human population. The mechanistic and exposure pathway is plausible and supported by direct environmental detection, but the causal chain from environmental contamination to measurable clinical AMR burden has not yet been empirically closed.

Drought, water scarcity and water-system disruption

The evidence specifically related to drought and water scarcity and AMR is significantly less developed than for temperature or flooding. Water scarcity can potentially affect concentrations of contaminants, sanitation conditions, and pathways of human exposure, and could exacerbate the water-system disruption already observed during flooding (Section 3.2) by concentrating pathogens and residual antimicrobials in reduced water supplies; however, there is limited evidence to date that links water-system disruption to AMR outcomes during water scarcity. This asymmetry is itself an important finding and justifies the focus on hazard-specific longitudinal and environmental monitoring of drought-affected contexts, such as much of Pakistan.

Other climate-related environmental and health-system pathways

The effects of coastal and sea-level change on aquatic microbial ecosystems could affect the mechanisms of AMR dissemination in a mechanistically plausible manner, but there is little direct evidence in the current literature that links this hazard to AMR outcomes. Climate hazards can also have indirect impacts on AMR, by affecting the capacity of healthcare systems to deliver services and control infections during extreme events (e.g., during heatwaves, the demand for healthcare services may exceed capacity, or during floods, water/sanitation infrastructure may be damaged, which reduces capacity to control infections). These health-system pathways are not well tested empirically and are explored in more detail in Section 5. The hazard-by-hazard pathways and evidentiary status summarized in this section are presented in Table 1.

Table 1. Climate hazards, proposed pathways and AMR outcomes

Climate hazard	Proposed pathway(s)	AMR-related outcome reported	Key source(s)
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Rising ambient temperature	Increased bacterial growth/mutation rate; enhanced horizontal gene transfer; organism-specific stress responses	Higher phenotypic resistance detection (E. coli, K. pneumoniae, S. aureus); higher ARG* abundance in Salmonella genomes; nonlinear dose-response	[9-13]
Heatwaves	Acute pathogen growth conditions; healthcare demand surges; possible health-service disruption	Hypothesis-generating; direct empirical evidence sparse	Section 3.1
Flooding / extreme precipitation	Sewage/wastewater overflow; agricultural runoff mobilization; water-treatment disruption	Environmental detection of MDR* organisms, carbapenemase genes (blaKPC, blaNDM) and resistance plasmids in floodwater; shifts in bacterial community structure	[15-18]
Drought / water scarcity / water-system disruption	Concentration of contaminants and residual antimicrobials in shrinking water sources; altered sanitation conditions	Evidence base markedly underdeveloped; no direct AMR-outcome studies identified	Section 3.3
Coastal / sea-level change	Modification of aquatic microbial ecosystems	Evidence essentially absent	Section 3.4

ARG = antimicrobial resistance gene; MDR = multidrug-resistant.

Strength, Consistency and Causal Limitations of the Evidence

The evidence reviewed above has several common limitations that must be addressed when assessing whether climate change is a true causal contributor to AMR (as opposed to being associated with AMR through confounding or shared anthropogenic drivers), which are summarized and presented with key study characteristics in Table 2.

Ecological study design.

Most population-level studies of the association between temperature and AMR [10, 11] are of an ecological design, meaning that associations are observed at the population level and are susceptible to confounding by factors that are associated with both temperature and AMR at the regional level, such as population density, access of healthcare, antimicrobial use, and socioeconomic development, as well as the ecological fallacy, which is the assumption that population-level associations are also present at the individual level.

Residual confounding. Even with the adjustment for measured antibiotic-use and socioeconomic factors, the association of climate remained independent in Zhou et al.'s [13] genomic analysis, but unmeasured factors (such as the intensity of surveillance, sanitation investment, or land-use change that co-occurred with warming) cannot be ruled out in any observational study, regardless of its size.

Temporality and dose-response. The Zeng et al. [12] difference-in-differences design provides a more robust temporal inference than cross-sectional studies alone, but a single quasi-experimental study in one country does not provide generalizable causal inference. The nonlinear temperature-resistance relationships reported by Zhao et al. [9] and Zhou et al. [13] further complicate any simple “more warming, more resistance” interpretation.

Organism-specificity. The negative finding for Salmonella bacteraemia in Queensland [14] contrasts with the positive findings for E. coli, Klebsiella and S. aureus elsewhere. This is not a statistical fluke, but rather an indication that

climate–AMR relationships are organism- and context-specific, and should not be extrapolated beyond pathogens or settings without direct evidence.

Environmental detection versus clinical infection.

As mentioned in Section 3.2, there is a general trend in this literature that the environmental metagenomic or molecular detection of resistance genes or resistant organisms [16, 17] does not equal demonstrated increases in human clinical AMR infections or outcomes, and the two should not be conflated.

Model projections versus observed outcomes.

Forecasting studies, such as those by Li et al. [8], did not rely on observations, but rather on scenario modelling, with a high-emissions, low-development scenario contrasted with a lower-emissions, higher-development scenario. This modelling showed a difference of about 2.4 percentage points in the estimated prevalence of AMR in the world by 2050 between the scenarios, with a larger difference for LMICs (about 4.1 points) than for high-income countries (about 0.9 points). These are comparative figures based on assumptions as to the future, and do not represent an actual or guaranteed increase.

Synthesis statement.

The evidence as a whole suggests a plausible mechanistic pathway for how climate-related hazards, especially rising temperature, can lead to increased AMR in several bacterial pathogens and settings, but does not support the unqualified claim that climate change causes AMR as a general and universal phenomenon. Climate change is best understood as a new, partially quantified, context-dependent contributing and amplifying factor that is in addition to the well-established primary driver of antimicrobial use, and causal inference is an ongoing methodological process [7].

Table 2. Key empirical studies and strength/limitations of evidence

Study	Design	Main finding	Key limitation
MacFadden et al. [10]	US ecological analysis	10°C rise in minimum temperature associated with 2.2–4.2% higher resistance across three organisms	Ecological design; residual confounding
McGough et al. [11]	28-country European cross-national analysis	Replicated positive temperature–resistance association	Ecological design
Zeng et al. [12]	Difference-in-differences, 31 Chinese provinces	1°C rise associated with 4.7% (3GC-R*) and 10.7% (CR*) increases in <i>K. pneumoniae</i> resistance	Single-country; quasi-experimental but not fully causal
Zhou et al. [13]	Longitudinal genomic analysis, 488,232 <i>Salmonella</i> genomes, 139 countries	~10% global increase in ARG abundance associated with climate change; nonlinear	ARG abundance ≠ confirmed phenotypic/clinical resistance
Manchal et al. [14]	Distributed lag non-linear model, Queensland, Australia	No significant temperature–resistance association in <i>Salmonella</i> bacteraemia after adjustment	Single organism/setting; null result, not generalizable either direction
Rahman et al. [16]; Cardoso et al. [17]	Metagenomic/qPCR floodwater sampling, Bangladesh and	Detection of MDR organisms and carbapenemase genes (blaKPC, blaNDM) in floodwater following major	Environmental detection only; no linked clinical infection data

	Brazil	flood events	
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3GC-R = *third-generation cephalosporin-resistant*; CR = *carbapenem-resistant*.

Prevention and Intervention Strategies

There are a number of intervention categories that apply to AMR prevention in general, and by plausible extension, to climate-associated AMR. It is important to note that most of the evidence below is for general AMR-reduction interventions, with comparatively little evidence testing interventions specifically designed and evaluated against climate-attributable AMR pathways, and this distinction is kept throughout. The evidence base and its limitations for each of the strategies discussed below is summarized in Table 3.

Antimicrobial stewardship

The evidence base for the effectiveness of antimicrobial stewardship programmes (ASPs) is the strongest of any of the interventions discussed here. A systematic review and meta-analysis [19] showed that ASPs have been effective in reducing antibiotic use in both hospital and non-hospital settings, but the authors specifically highlighted that there is limited evidence for resource-limited settings. This evidence is not specific to climate-attributable AMR, nor was it created to test for effectiveness against climate-attributable AMR, but rather as a proxy for resistance pressure in general.

WASH and environmental controls

Based on the flooding evidence in Section 3.2, the target of interventions is a climate-resilient water, sanitation and hygiene (WASH) infrastructure, which is a mechanistically plausible intervention target. A policy-mapping analysis of 193 countries, however, revealed no single category of WASH-related AMR policy was implemented in all countries, and few national AMR action plans included WASH provisions in enforceable policy [20]—which is not a sign of lack of plausibility of the intervention, but rather a policy implementation gap. A systematic review of biosecurity and WASH interventions in animal-agricultural settings [21] showed that manure-management interventions reduced antimicrobial resistance genes or resistant bacteria in animal waste by up to 99% and farm-management interventions reduced antibiotic use by up to 57%. But, of the 20 studies, only 7 measured resistance outcomes (not antibiotic use or microbial load) and many of the studies included were at high risk of bias. These results are not intended to be interpreted as proof of climate-attributable AMR, but rather as indicative of overall agricultural AMR reduction. The economic impact of AMR is projected to increase by about 30% in the livestock sector by 2040, with total AMR-related economic losses in the sector estimated at US\$318 billion, compared to an estimated US\$53 billion under the most severe scenario for phase-out of antimicrobial growth promoters (AMGs) (FAO 2026 economic assessment [22] – not a climate-specific assessment).

Infection prevention, control, and climate-resilient health systems

Routine infection prevention and control (IPC) capacity and the broader health system could be challenged by climate hazards, either by damage to water/sanitation infrastructure during floods, or by increased health system demand during heatwaves (Section 3.4). The mechanisms in Section 3 suggest that building climate resilience into water, sanitation and hospital infrastructure –

so that these systems continue to function during floods, heatwaves and droughts without becoming exposure pathways – is a logical next step. Conceptually coherent but empirically untested research priority: No study identified for this review directly assessed interventions for IPC or health-system-resilience outcomes attributable to climate change.

Surveillance and One Health integration

The 2024 UN General Assembly Political Declaration on AMR [23] was endorsed by 193 member states, and reiterated by WHO's 2025 Global Call to Action as an operational priority

[24] and the adoption of the Global Action Plan on AMR 2026–2036 by the World Health Assembly through a Quadripartite (FAO/UNEP/WHO/WOAH) process [25]. This architecture does not yet include climate-hazard specific triggers for surveillance or intervention; embedding climate signals into existing One Health infrastructure is still emerging and not yet common practice. AMR priorities differ significantly between higher-income countries, where novel antibiotic research and development is a priority, and the Global South, where clean water, sanitation, vaccine coverage, and regular access to WHO “Access”-category antibiotics are priorities. [26] This equity framing is directly relevant to Pakistan and South Asia: interventions with the highest plausibility for addressing climate-associated AMR pathways (WASH, environmental surveillance, stewardship) are those that the Global-South-priority argument identifies as underfunded compared to high-income country research priorities.

Synthesis.

None of the interventions reviewed in this section have been assessed in a study that was specifically designed to test the intervention for effectiveness in reducing climate-attributable AMR as a specific outcome. The evidence available supports the plausibility of stewardship, climate-resilient WASH, agricultural biosecurity, infection prevention and control, and integrated One Health surveillance as prevention strategies that are consistent with the mechanistic pathways outlined in Section 3, but not as being equivalent to proven effectiveness against climate-associated AMR specifically.

Table 3. Prevention and intervention strategies, evidence and limitations

Strategy	Evidence for general AMR reduction	Evidence specific to climate-associated AMR
Antimicrobial stewardship (5.1)	Systematic review/meta-analysis [19]: effective in reducing antibiotic consumption, hospital and non-hospital settings	None identified
WASH & environmental controls (5.2)	Policy-mapping across 193 countries [20]; biosecurity/WASH review [21]: up to 99% reduction in agricultural ARGs, 57% reduction in antibiotic use; FAO livestock economic case [22]	Mechanistically plausible given flooding pathway (3.2); not directly tested
Infection prevention, control, and climate-resilient health systems (5.3)	Established general principles; not separately reviewed here	Conceptually coherent (health-system disruption pathway, 3.4); empirically untested
Surveillance & One Health integration	UN Political Declaration [23]; WHO Global Call to Action [24]; GAP-	Climate-hazard-specific triggers not yet embedded in governance

(5.4)	AMR 2026–2036 [25]; equity analysis [26]	architecture
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Research and Policy Priorities

Methodological priorities.

Future studies should focus on quasi-experimental and longitudinal designs, rather than cross-sectional ecological analyses, and should build on the difference-in-differences design employed by Zeng et al. [12]. Studies linking environmental resistance-gene detection (as in the Bangladesh and Brazil flood studies) to downstream, confirmed clinical infection outcome are needed to close the gap identified in Sections 3.2 and 4. The study of AMR in fungi, viruses, protists and helminths in relation to climate hazards is virtually absent, and is identified as understudied by van Bavel et al. [5].

Geographic and data priorities.

Although South Asia has been repeatedly highlighted as having one of the highest projected future AMR burden [8], primary studies directly measuring climate-hazard–AMR associations with Pakistani or South Asian data are limited. This is a priority gap for national research investment, especially in the context of Pakistan's documented exposure to both extreme heat and flooding, and should be coordinated through existing AMR National Action Plan and climate-adaptation structures.

Intervention-evaluation priorities. As described in Section 5, there was no identified intervention study that was designed to test effectiveness against climate-attributable AMR. Trials should prioritize those that explicitly include climate as a study variable (e.g., post-flood WASH restoration compared to downstream AMR surveillance; heatwave-triggered stewardship intensification) rather than as background context.

Policy integration priorities. In most countries, including Pakistan, climate adaptation planning and AMR National Action Plans are largely parallel and siloed policy tracks. The Quadripartite One Health governance architecture and GAP-AMR 2026–2036 provide a structural opportunity for integration, but this is not yet systematically operationalised and should be an explicit priority for the public health and environmental policy communities in Pakistan.

Review limitations. This is a narrative review, which was performed by one reviewer without independent dual extraction, and did not follow a registered protocol with complete screening records, which may introduce selection bias compared to a systematic review. Sources were primarily limited to English language, peer-reviewed literature and major intergovernmental reports, which may under-represent Pakistani, South Asian, or non-English regional evidence.

Conclusion

Climate change and antimicrobial resistance are now understood to be interlinked and not separate threats to global health. Based on current evidence, climate change is a likely and partially quantifiable contributing and amplifying factor for AMR, most clearly for increased temperatures, and most directly for environmental mobilization of resistant organisms during flooding, in addition to the well-established primary driver of antimicrobial use. There is limited evidence assessing interventions specifically developed to prevent AMR associated with climate change, especially in LMICs and South Asia. The need to incorporate climate-informed surveillance and prevention strategies into current One Health and AMR National Action Plan frameworks is a pressing research and policy

priority in Pakistan, where there is documented exposure to both extreme heat and flooding, and the region is expected to have a high burden of AMR.

Declarations

Author Contributions: Maham Saeed conceived and coordinated the review, and contributed to the literature synthesis and manuscript preparation. Maha Saeed helped with the literature search,

evidence synthesis, and manuscript writing. Bisma Imtiaz participated in literature review, critical revision, and refinement of the manuscript. Muqadas Shehzadi contributed to the epidemiological framing of the review, data verification, and critical review of the manuscript for important public-health content. All authors read and approved the final manuscript and are accountable for the manuscript's content. The use of generative AI assistance in the preparation of the manuscript is recognized here and explained in the AI-Use Disclosure below.

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