Pharmacy Reports

https://doi.org/10.51511/pr.94

REVIEW

Open Access

Climate change and antimicrobial resistance: A global public health crisis at the environmental nexus

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Abstract: Antimicrobial resistance (AMR) and climate change are major global public health challenges, with growing evidence indicating interconnected relationships. This review explores the multifaceted links between climate change and AMR, focusing on how rising temperatures influence bacterial resistance mechanisms, alter pathogen distribution patterns, and affect environmental reservoirs of resistance genes. Evidence synthesized in this review indicates that rising temperatures correlate with increased AMR rates across multiple regions, with each 1°C temperature increase linked to higher resistance prevalence. Climate change affects environmental transmission dynamics via soil ecosystems, aquatic environments, and cryosphere degradation, which can release long-dormant resistance determinants from permafrost and glaciers. Rising temperatures facilitate the geographic spread of resistant pathogens, as observed in *Vibrio* species expanding to higher latitudes and the emergence of *Candida auris* as a clinically significant pathogen from environmental sources. Wildlife and livestock act as potential reservoirs, while climate-driven habitat changes increase human-animal interactions that may facilitate transmission. The bidirectional relationship between these challenges—where rising temperatures contribute to AMR spread and resistant infections may hinder climate resilience—requires integrated One Health approaches. Strategies proposed include enhanced surveillance, climate-informed antimicrobial stewardship, and ecosystem-based interventions to address these interconnected issues. Importantly, integrating climate-informed health policies that align climate adaptation with AMR control should be prioritized globally to safeguard antimicrobial efficacy amid accelerating environmental changes.

Keywords: climate change, antimicrobial resistance, temperature adaptation, environmental reservoirs, One Health

Introduction

Antimicrobial resistance (AMR) is a critical global public health challenge, associated with approximately 1.27 million deaths directly attributed to bacterial drug-resistant infections in 2019 [1]. Concurrently, climate change is accelerating ecosystem transformations and altering environmental conditions worldwide. These crises, previously viewed as distinct challenges, increasingly demonstrate interconnected feedback mechanisms. The convergence of climate change and AMR creates a concerning dynamic where each may exacerbate the other, producing what researchers describe as a "threat multiplier" effect on health systems already strained by addressing these issues separately [2].

The mechanisms linking climate change and AMR span molecular to ecological scales. Temperature directly affects bacterial physiology, modifying membrane permeability, metabolic activity, and resistance gene expression [3]. Such changes can significantly alter microbial interactions with antimicrobial agents, potentially reducing the efficacy of existing treatments. At broader ecological levels, warming reshapes microbial communities, altering competitive dynamics and favoring organisms with resistance traits [4].

Environmental reservoirs—including soil, water, and ice—store and transmit antimicrobial resistance genes (ARGs), with climate change affecting their ability to retain and disperse these genetic determinants. Pathogen distributions are also shifting geographically, introducing new infection risks. For example, *Vibrio* species, particularly human pathogens, have expanded northward as coastal waters warm, dispersing their intrinsic and acquired resistance mechanisms [5]. Fungal pathogens like *Candida auris* have similarly transitioned from environmental niches to human infections, potentially through thermal stress adaptations [6].

This climate-AMR nexus disproportionately affects tropical and low-income nations in the Global South, where limited healthcare infrastructure, high baseline temperatures, and less stringent environmental regulations heighten vulnerabilities. These disparities highlight climate justice concerns, as unequal emissions contributions and healthcare capacities exacerbate AMR risks. Such inequities may hinder sustainable development and overwhelm fragile health systems.

This review examines evidence for climate-associated resistance mechanisms, evaluates temperature-driven changes in ARG transmission, analyzes climate impacts on pathogen distribution and resistance evolution, and explores interventions to address these interconnected issues. By framing AMR through a climate lens, this work provides a framework for researchers, clinicians, and policymakers to safeguard antimicrobial efficacy amid global environmental changes.

Temperature-driven mechanisms of AMR

Direct effects of temperature on AMR

Temperature fluctuations directly influence bacterial physiology and AMR mechanisms. Thermal variations can prompt bacterial adaptations that enhance survival under antibiotic exposure. Studies indicate that temperature shifts alter bacterial growth patterns and resistance profiles. For example, in *Escherichia coli* and *Staphylococcus epidermidis*, resistance evolution to distinct antibiotic classes has been shown to modify optimal growth temperatures. Resistance to nucleic acid and cell wall inhibitors in *E. coli* correlates with lower optimal temperatures, while resistance to protein synthesis inhibitors elevates optimal temperatures in both species [4].

Temperature also modulates resistance gene expression and functionality. Experimental evidence reveals nonlinear temperature-dependent antibiotic resistance in *E. coli*. At 42°C, the efficacy of antibiotics like gatifloxacin decreases, whereas at 27°C, resistance increases up to 256-fold [2]. These variations correlate with heightened transcription of genes such as *marA*, *ygfA*, and *ibpB*. Thermal stress may also drive mutations at chromosomal sites, contributing to diverse resistance patterns under fluctuating temperatures.

Notably, antibiotics themselves may influence bacterial temperature responses. Systematic studies demonstrate that antibiotic exposure can shift bacterial optimal growth temperatures and broaden the temperature range supporting growth [3]. This suggests antibiotic-induced cellular changes, such as stress pathway activation or structural modifications, may interact with thermal adaptation processes.

Temperature-induced mutagenesis represents a potential pathway for climate-driven AMR acceleration. A notable example involves the fungal pathogen *Rhodosporidiobolus fluvialis*, where mammalian body temperature induces mutagenesis, conferring pan-resistance to three first-line antifungals [7]. Such mutagenesis may enhance both resistance and virulence, enabling pathogens to evade host defenses more effectively.

Physiological mechanisms underlying these effects include altered membrane permeability, protein conformation, and metabolic activity. Elevated temperatures may increase membrane fluidity, potentially enhancing antibiotic uptake or efflux pump efficiency. Temperature shifts can also affect enzyme activity critical to resistance, such as β-lactamase function. For instance, in *Xanthomonas arboricola*, streptomycin's minimum inhibitory concentration decreases at 30°C compared to 28°C, suggesting temperature-dependent resistance modulation [8].

Epidemiological evidence of temperature-resistance relationships

Epidiological studies across diverse regions have identified associations between ambient temperature and AMR, suggesting climate influences on resistance trends at population scales. A cross-national analysis of 28 European countries found that regions with ambient temperatures ~10°C warmer than others experienced faster increases in antibiotic resistance rates between 2000 and 2016, even after controlling for factors like antibiotic use and population density [9].

Nationwide analyses in China have demonstrated temperature-resistance correlations. For carbapenem-resistant *Klebsiella pneumoniae* (CRKP), each 1°C temperature increase was linked to a 1.14-fold rise in prevalence, with similar trends observed for *Acinetobacter baumannii* and *Pseudomonas aeruginosa* [10]. Subsequent research using difference-in-differences models indicated that temperature increases correlated with resistance to multiple antibiotic classes, moderated by socioeconomic variables such as healthcare access [11].

Clinical impacts of temperature-driven resistance have been observed in infection-specific studies. In Israel, a 5.5°C rise in average monthly temperature was associated with a 6.2% increase in community-onset

Escherichia coli bloodstream infections and a 4.9% increase in multidrug-resistant cases [12], highlighting potential needs for seasonally adaptive stewardship programs.

Advanced modeling techniques have refined predictions of resistance patterns. Temperature-resistance relationships often follow non-linear trends, with stronger effects in cooler regions undergoing rapid warming, suggesting disproportionate AMR increases in these areas [13]. Ecological niche modeling (Maxent approach) projects climate-driven shifts in the global distribution of *Staphylococcus aureus*, a key reservoir of resistance genes [14].

These findings have prompted calls to update AMR surveillance frameworks. An ecological study estimated that temperature increases, combined with governance factors (e.g., corruption indices), could explain up to 78% of variance in carbapenem-resistant *Pseudomonas aeruginosa* prevalence across Europe [15]. As temperatures rise, integrating climate variables and human behavioral data into surveillance systems may become critical to track evolving resistance dynamics.

Environmental reservoirs and transmission dynamics of ARGs

Soil and terrestrial environments

Soil ecosystems act as critical reservoirs for ARGs, with climate change influencing their persistence and transmission. Rising soil temperatures have been associated with elevated proportions of ARGs, particularly those conferring resistance to aminoglycosides, beta-lactams, and tetracyclines. Field experiments simulating long-term warming (+4°C over five years) revealed enrichment of 15 ARGs across seasons in both plantation and natural forest soils, indicating temperature-driven shifts in ARG profiles independent of land management practices [2].

Interactions between environmental contaminants and climate factors may amplify resistance evolution. For example, mercury pollution combined with higher temperatures enhances antimicrobial resistance by activating bacterial efflux pumps—membrane transporters that expel antibiotics and other toxins [16]. This co-selection mechanism represents a potential pathway for climate-driven resistance spread, as persistent metals maintain selective pressure on ARGs even without direct antibiotic exposure.

Soil composition and moisture further mediate climate impacts on ARG transmission. Phenotype-based single-cell Raman spectroscopy combined with metagenomic sequencing has shown that soil warming increases the activity of antibiotic-resistant bacteria within ecological networks [17]. Similarly, studies of collembolan gut microbiomes revealed that warming intensifies pesticide-driven increases in ARG abundance and diversity [18], highlighting how climate change alters microbial communities to favor resistance proliferation.

Atmospheric greenhouse gases may have context-dependent effects on ARG dissemination. A free-air CO_2 enrichment experiment (+200 ppm) demonstrated that elevated CO_2 reduced the abundance of sulfadiazine-associated ARGs and mobile genetic elements at low antibiotic concentrations (0.5 mg/kg) [19]. This unexpected finding suggests that, while climate change broadly exacerbates resistance spread, specific greenhouse gas interactions could inform targeted mitigation strategies.

Aquatic systems and hydrological influences

Aquatic environments, particularly river systems, act as dynamic pathways for ARG dissemination under climate change. Studies of river biofilms—microbial communities attached to submerged surfaces—highlight their role in ARG ecology. Contrary to initial assumptions, experimental warming (30°C) showed divergent effects: naturally occurring ARGs increased in abundance, while wastewater-derived ARGs were lost more rapidly [20]. These findings challenge the notion of uniform climate-driven ARG amplification and underscore the complexity of aquatic resistance transmission.

Hydrological shifts linked to climate change also influence ARG spread. Extreme precipitation events and altered water cycles have been associated with degraded groundwater quality and modified ARG profiles [21]. In urban settings, higher rainfall intensity—a projected climate change outcome—correlates with elevated ARG concentrations in stormwater runoff [22], suggesting altered precipitation patterns may create new resistance transmission routes.

Managed aquatic systems, such as drinking water networks, face distinct climate-related AMR challenges. Temperature increases and shifting precipitation may reduce water treatment efficacy, while recreational water use poses emerging risks. For example, technical

snow produced for skiing in mountainous regions has been found to contain antimicrobial agents, ARGs, and resistant bacteria, though upstream reservoir construction reduced ARG transfer [23]. As warming expands snow tourism to marginally cold regions, monitoring recreational water systems for resistance dissemination becomes increasingly critical.

Reclaimed wastewater, increasingly used in water-scarce regions due to climate pressures, introduces new ARG transmission risks. Submerged macrophyte systems show promise for mitigating this issue: *Myriophyllum spicatum* achieved greater ARG reductions in treated effluent compared to other species [24]. However, treatment efficacy depends on wastewater loading levels, which may fluctuate with climate-driven rainfall variability. Lower loading conditions improved ARG removal, highlighting the need for adaptive wastewater management strategies in a changing climate.

Marine environments as resistance reservoirs

Coastal and estuarine ecosystems act as critical interfaces where terrestrial and marine systems interact, fostering conditions conducive to AMR development and transmission. The distribution of resistant *Vibrio* species in these regions has drawn attention due to their public health implications and sensitivity to climate variables. A decade-long surveillance study (2013–2022) in southern Chinese coastal waters documented shifts in *Vibrio* spp. resistance patterns, including increased detection of extended-spectrum β-lactamase genes and carbapenemases such as *bla*NDM-1, alongside rising sea surface temperatures [25]. These temporal trends align with climate-driven environmental changes, though causal relationships require further investigation.

Marine resistomes—the collective ARGs in ocean ecosystems—exhibit global distribution patterns influenced by climate factors. Metagenomic analyses reveal latitudinal and depth-dependent trends in ARG abundance, with tetracycline, bacitracin, macrolide, and fluoroquinolone resistance genes dominating marine resistomes [26]. Climate projections suggest ARG distributions will shift by 2100, with higher-risk genes potentially concentrating in lower latitudes and regions of intensified human activity, particularly in the Pacific and Atlantic Oceans.

Warming coastal ecosystems may facilitate *Vibrio* proliferation in marine food chains, raising questions about AMR transmission via seafood. Aquaculture studies have identified associations between rising water temperatures and increased prevalence of resistant *Vibrio parahaemolyticus* strains, a pathogen linked to seafood contamination [27]. Such findings highlight the need for enhanced monitoring of AMR in seafood systems to mitigate human exposure risks.

Microplastic pollution in marine environments may further amplify AMR dissemination. Bacterial communities on microplastics exhibit resistance profiles comparable to those on natural organic particles, suggesting both substrates could serve as ARG vectors [28]. Climate-driven changes in ocean circulation may alter the distribution of microplastic-associated ARGs, complicating marine resistance mitigation efforts.

Extreme weather events, intensified by climate change, can rapidly modify marine AMR dynamics. Post-Hurricane Ian genomic studies in Florida coastal waters detected shifts in *Vibrio* populations and ARGs, with storm-induced water parameter changes creating favorable conditions for bacterial proliferation and genetic exchange [29]. Similar observations following mucilage events in the Marmara Sea revealed elevated levels of antimicrobial-resistant *Escherichia coli* and *Clostridium perfringens* [30].

In aquaculture systems, critical for global food security under climate change, AMR management challenges are pronounced. Genomic analyses of *Vibrio parahaemolyticus* in southern Korean waters identified widespread resistance linked to seafood consumption risks [27]. European assessments project that coastal warming and extreme weather may increase *Vibrio* abundance in seafood, particularly in brackish waters [31], underscoring the need for climate-adaptive aquaculture policies.

Cold environments and permafrost: reservoirs and release of ancient resistance

Permafrost and glacial resistomes

Permafrost regions, historically viewed as microbial archives, are now acknowledged as significant reservoirs of ancient antibiotic resistance genes and associated mechanisms. Thawing of these permanently frozen soils due to rising global temperatures facilitates the release of microorganisms and genetic material preserved for millennia. Metagenomic studies of Arctic permafrost have identified a diverse array of ARGs. For example, one large-scale analysis detected 70 ARGs targeting 18

antimicrobial drug classes and nearly 600 virulence factors categorized into 38 groups [32].

Genomic analyses of permafrost-preserved bacteria provide evidence for the long-term persistence of resistance traits. Whole genome sequencing of *Acinetobacter lwoffii* strains isolated from permafrost aged 15,000 to 1.8 million years revealed genetic parallels with modern clinical and environmental counterparts [33]. Notably, chromosomal determinants in ancient permafrost strains showed no distinct differences from those in contemporary clinical isolates. Phylogenetic comparisons further indicated that permafrost strains did not cluster separately, with some ancient strains closely related to modern clinical variants. These findings imply that antibiotic resistance and virulence mechanisms existed in permafrost bacteria long before human antibiotic use.

Functional studies confirm that permafrost-associated resistance extends beyond genetic signatures to active mechanisms. Research on Arctic soil-derived haemolytic bacteria evaluated both AMR levels (via minimum inhibitory concentrations) and underlying genetic factors [34]. *Micromonospora* isolates displayed non-wild-type resistance to erythromycin, penicillin, and tetracycline, while *Pseudomonas* isolates exhibited elevated resistance to nalidixic acid, streptomycin, and colistin. This demonstrates that functional resistance persists in permafrost microbes despite prolonged frozen states.

High-altitude glaciers and ice sheets also serve as reservoirs of resistance elements, with climate change driving both the release of ancient genetic material and the deposition of modern contaminants. A study of Tibetan glaciers across 21 sites identified distinct ARG distribution patterns linked to atmospheric processes [35]. Monsoon-influenced glaciers showed higher ARG abundance than westerly-dominated ones, likely reflecting antibiotic use trends in source regions such as the Indian subcontinent. This suggests glaciers accumulate contemporary resistance determinants alongside ancient ones.

Glacier-fed streams further enable ARG transfer from ice masses to downstream ecosystems. Research on Central Asian glaciers revealed shared resistance genes across surface ice, cryoconite (microbial sediment), and meltwater streams [36]. Of 944 ARGs (spanning 22 antibiotic classes) detected, 633 were common to all three environments. Similarities in ARG profiles between surface ice, cryoconite, and stream biofilms highlight how glacial melt facilitates the dispersal of resistance genes into aquatic systems.

Climate-driven release mechanisms

Thermokarst lakes, formed by thawing permafrost, are significant yet understudied reservoirs of ARGs in polar and high-altitude regions. Metagenomic analyses of these lakes in the Qinghai-Tibet Plateau's Yellow River Source Area identified diverse ARG profiles in sediments and water, with rifamycin resistance genes being particularly abundant [37]. Comparative studies across permafrost regions further indicate that thermokarst lakes contain higher ARG concentrations than adjacent permafrost soils, particularly when considering both sediment and water components [36]. As climate warming accelerates permafrost thaw, these lakes create new aquatic environments that accumulate resistance determinants.

Snow and ice in alpine regions act as reservoirs and transport pathways for resistance genes. A study of Antarctic green and red snow—colored by algal blooms—detected 525 ARGs conferring resistance to 30 antibiotic classes [36]. Green snow exhibited higher ARG diversity than red snow, suggesting that algal-influenced snow may serve as unrecognized reservoirs for resistance genes. With climate change increasing the frequency of colored snow globally, these environments could contribute to the dispersal of antibiotic resistance.

Thawing permafrost drives ecological shifts in microbial communities that affect resistance dynamics. A metagenomic comparison of ancient Siberian permafrost and modern Kamchatkan frozen soils revealed taxonomically diverse communities with no clear correlation to sampling location, depth, or age [38]. Despite this variability, β-lactamase genes were consistently prevalent, averaging 0.9 copies per bacterial genome—levels comparable to heavily contaminated modern environments. The unexpected abundance of β-lactamases in pristine Arctic soils suggests these enzymes may have ecological roles beyond antibiotic resistance, such as in microbial competition or biofilm formation.

Atmospheric circulation patterns, altered by climate change, also influence ARG distribution in glacial systems. Research on Tibetan glaciers found higher ARG abundance in monsoon-dominated glaciers than in westerly-dominated ones, likely due to monsoons transporting air from regions with high

antibiotic use, such as the Indian subcontinent [35]. As monsoon-influenced glaciers melt rapidly, they may release substantial ARG quantities into downstream ecosystems. This link between shifting atmospheric patterns and resistance gene dispersal highlights an emerging mechanism through which climate change could redistribute resistance determinants across regions.

Emerging and re-emerging pathogens under climate change

Human pathogen dynamics

The geographic distributions of pathogens are shifting in response to climate warming, with species historically restricted to tropical and subtropical regions now detected in temperate zones. *Vibrio* species illustrate this pattern: non-O1/non-O139 *V. cholerae* isolates from German and European coastal waters show increasing prevalence in northern latitudes [5]. These isolates exhibited widespread AMR, particularly to beta-lactams, chloramphenicol, and tetracycline, with the carbapenemase gene *VCC-1* identified in multiple samples from German surface waters. These observations align with evidence that rising sea surface temperatures enable *Vibrio* proliferation in previously inhospitable habitats.

Fungal pathogens demonstrate adaptive responses to temperature changes, raising concerns that climate shifts may facilitate environmental fungi transitioning to human pathogens. A study isolating the multidrug-resistant yeast *Candida auris* from Andaman Islands coastal wetlands provided insights into this process [6]. The pathogen, which emerged globally as a human health concern in the past decade, was detected in natural salt marshes and sandy beaches, with both drug-susceptible and multidrug-resistant strains coexisting. These findings support hypotheses linking *C. auris*' emergence as a human pathogen to climate-driven ecological changes in wetland ecosystems.

Clinical reports and surveillance data highlight the health impacts of climate-related pathogen spread. For example, *Shewanella putrefaciens*—a gram-negative bacterium typically aquatic—has recently caused human infections associated with environmental exposure [39]. One case involved a soft tissue infection following coastal water exposure. Similarly, hospital-acquired infections caused by carbapenemase-producing *Aeromonas hydrophila* were linked to plumbing system issues during heatwaves [40]. Researchers identified strains carrying resistance genes such as *KPC-3, VIM-2, OXA-232*, and chromosomal *CphA*-like carbapenemases, suggesting warmer temperatures may enhance pathogen persistence in water systems and promote resistance development.

Airborne transmission pathways are also evolving under climate change. A study of dust storms in the Eastern Mediterranean demonstrated how atmospheric transport redistributes pathogens and resistance genes [41]. Air samples collected during Middle Eastern dust storms contained facultative pathogens like *Klebsiella pneumoniae*, *Stenotrophomonas maltophilia*, and *Aspergillus fumigatus*, with pathogen abundance increasing during storms and at higher temperatures. As climate change intensifies drought and alters precipitation patterns, dust-mediated dispersal of resistant pathogens may become a more prominent mechanism for long-distance spread.

Animal and wildlife reservoirs

Wild animals act as reservoirs and vectors for antibiotic-resistant bacteria, with climate change potentially influencing transmission risks through shifts in migration and habitat use. A study of urban birds in Central Spain identified antimicrobial-resistant *Salmonella* strains in 12.3% of sampled individuals, with the highest prevalence in white storks foraging in landfills [42]. Researchers observed that climate-driven food scarcity and habitat changes have led many species to adapt feeding behaviors, increasing their interaction with urban environments. This creates opportunities for resistance transmission between wildlife and human populations.

Marine ecosystems are emerging as reservoirs for resistant bacteria as ocean temperatures rise. A novel pathogenic strain of *Kocuria kristinae*, isolated from the marine fish *Larimichthys crocea*, was found to carry multiple virulence factors and ARGs [43]. Genomic analysis revealed genes associated with drug resistance and regulatory systems. Notably, *K. kristinae* has been reported in humans and livestock, indicating possible cross-species transmission pathways. Warmer coastal waters may accelerate such adaptations, fostering new resistance reservoirs in marine species.

Wildlife rehabilitation centers provide insights into resistance patterns in animals affected by climate-related stressors. A study of *Campylobacter* in wild birds admitted to a rescue center detected the bacterium in 24.88% of individuals across 33 species, with widespread resistance to trimethoprim/

sulfamethoxazole, ciprofloxacin, and enrofloxacin [44]. The authors suggest that climate change, habitat loss, and agricultural pressures may compromise wildlife health, increasing interactions between resistant bacteria from diverse environments during rehabilitation.

Livestock systems face growing challenges from climate-driven AMR. A simulation model of equine cyathostomin parasites projected faster anthelmintic resistance development under future climate conditions, driven by extended periods favorable for free-living larval stages [45]. These results indicate that warming temperatures could undermine parasite control strategies in agriculture.

Heat-tolerant livestock, such as camelids, are becoming more prevalent in arid regions under climate change, but their resistance profiles require attention. A genomic study of Staphylococcaceae from African dromedaries found non-wild-type antibiotic resistance in one-third of isolates [46]. As desertification increases reliance on such species, understanding their resistance dynamics will be critical for managing antimicrobial use in warming climates.

Conclusion

The intersection of climate change and AMR presents a complex challenge to global public health that requires coordinated, multidisciplinary strategies. This analysis highlights how these issues are interconnected through mechanisms operating across molecular, ecological, and geographical scales. Rising temperatures may influence the evolution and spread of resistance, while environmental changes can create new pathways for the transmission of resistance genes and the emergence of pathogens.

Key points from this synthesis include: (i) the relationship between temperature and resistance involves non-linear dynamics, which could disproportionately affect specific regions as global temperatures rise. (ii) environmental reservoirs—particularly in rapidly changing ecosystems such as permafrost and coastal areas—serve as repositories of historical resistance traits and potential sites for new resistance development. (iii) expanding pathogen ranges and adaptations to new hosts introduce risks that may not be fully captured by existing surveillance systems.

These findings underscore the need to reassess approaches to AMR in the context of climate change. Current stewardship programs could benefit from integrating climate variables and seasonal patterns into their frameworks. Expanding environmental monitoring to track resistance determinants across ecosystems (terrestrial, aquatic, and atmospheric) is also critical. Additionally, the One Health framework should be strengthened with metrics and interventions that explicitly address the climate-AMR relationship.

Integrating climate-informed policies into national and global AMR strategies is increasingly important. Climate adaptation measures should be incorporated into AMR mitigation efforts, particularly in regions vulnerable to both environmental shifts and resistance challenges. As climate change progresses, interactions between AMR and ecosystem changes may intensify, necessitating a unified One Health approach to safeguard antimicrobial efficacy and public health.

Delays in addressing these interconnected challenges could lead to compounded effects, potentially undermining progress in both AMR management and climate resilience. While opportunities for intervention exist, they are narrowing. Evidence-based, collaborative strategies that recognize the links between climate change and AMR are essential to preserving antimicrobial effectiveness and enhancing adaptive capacity in a changing environment.

Acknowledgment

None.

Declaration of interest

The authors declare there are not competing interests.

Author contributions

DP: investigation, visualization, original draft writing; AP: editing, draft writing editing, finalization.

Received: December 3, 2024
Revised: April 26, 2025
Accepted: April 26, 2025
Published: April 28, 2025

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