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Environmental Microbiology Under a Warming Sky: A Critical Review of Climate-Driven Shifts in Microbial Function, Pathogen Ecology, and Antimicrobial Resistance

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Abstract: *Environmental microbiology occupies an unusually exposed position in climate science. Microbial communities regulate the carbon and nitrogen cycles that determine how much warming actually occurs, and at the same time they are reshaped by the warming they help to produce, which makes the relationship circular rather than linear. This review examines three areas where that circularity is best documented: soil and permafrost microbial feedbacks on greenhouse gas emissions, the redistribution of waterborne pathogens such as Vibrio species under rising sea surface temperature, and the emerging evidence linking climate variables to antimicrobial resistance. Drawing on peer-reviewed literature published mainly between 2020 and 2026, including work from Nature Microbiology, PNAS, The Lancet Planetary Health, and regional case data from sub-Saharan Africa, the paper argues that current research has established correlation with more confidence than it has established mechanism, and that policy language has in places run ahead of what the microbiology can support. The Malawi cholera outbreak of 2022–2023, precipitated by Tropical Storm Ana and Cyclone Gombe, is used as an extended case to show how climate hazards convert into microbial risk through infrastructural failure rather than through pathogen biology alone. The review also identifies a structural bias in the literature toward temperate and Arctic systems, leaving tropical and semi-arid microbiomes comparatively under-sampled despite their exposure to some of the more severe projected climate stresses. The paper closes by arguing that environmental microbiology's contribution to climate policy will remain limited until researchers resolve persistent disagreements over whether taxonomic shifts under warming translate into functional consequences, and until surveillance systems in lower-income regions catch up with those already operating in Europe, North America, and China.*

Keywords: environmental microbiology; climate change; soil microbial feedback; waterborne pathogens; antimicrobial resistance

Introduction

There is a particular kind of unease that runs through recent environmental microbiology, one that does not show up clearly if you read only the abstracts. Individually, papers on soil warming experiments, permafrost thaw, or Vibrio range expansion each report findings that are more or less digestible. Read together, though, they describe something harder to summarise cleanly: a set of microbial systems that are simultaneously victims of climate change and, in a meaningful sense, co-authors of it. Soil and permafrost microbes hold a genuinely enormous carbon reservoir, and how that reservoir responds to warming is one of the larger uncertainties in current Earth system modelling (Patoine et al., 2022). At the same time, warmer coastal waters are expanding the geographic and seasonal range in which pathogenic Vibrio species can establish themselves (Baker-Austin et al., 2017; Schneider, 2024), and there is now a body of ecological evidence, still contested in places, suggesting that climate variables interact with the spread of antimicrobial resistance genes (MacFadden et al., 2018). None of this is new in the sense of being unknown to specialists. What has changed over the past five years is the volume and geographic spread of empirical work, and, not always in step with that, the confidence with which findings get translated into policy recommendations.

This review does not attempt to cover the entire field, which would be neither possible nor useful within the space available. It focuses instead on three strands where the climate-microbiology relationship is documented with enough empirical depth to support genuine critical engagement: microbial regulation of soil and permafrost carbon under warming, the climate sensitivity of waterborne and marine pathogens, and the still-emerging evidence base connecting climatic variables to antimicrobial resistance. The sub-Saharan African cholera experience of 2022–2023, is treated at greater length because it illustrates something the other two literatures tend to underplay: that the pathway from a climate hazard to a microbial health outcome usually runs through infrastructure, governance, and poverty before it runs through pathogen physiology. A warming ocean does not cause a cholera outbreak on its own. It creates conditions that, combined with a broken water system and a displaced population, produce one.

The argument developed across the following sections is that environmental microbiology has, over the past half-decade, moved from documenting the existence of climate sensitivity in microbial systems to attempting, with mixed success, to establish direction and magnitude of effect. That is a harder task, and the literature is honest about this difficulty more often than commentary on the literature tends to be. Where this review differs from a conventional synthesis is in treating some of that unevenness as analytically significant rather than as a limitation to be noted and set aside. The uneven geography of the evidence base, concentrated heavily in China, the United States, and Europe, is not incidental; it shapes what counts as an established finding and what remains speculative, and it has consequences for how confidently the field can speak to the regions, largely in the tropics and the Global South, where climate exposure is often highest.

Soil and permafrost microbial carbon under warming

The claim that soil microbial communities mediate the terrestrial carbon cycle is not controversial; it has been established for decades. What has shifted is the granularity with which researchers can now describe how warming alters that mediation, and the honest answer emerging from recent work is: unevenly, and in ways that depend heavily on ecosystem type and experimental duration. Patoine et al. (2022), analysing two decades of global soil microbial carbon data in Nature Communications, found that microbial carbon stocks have generally declined, with implications for the microbial carbon pump and for dark CO2 fixation, both of which contribute to soil carbon sequestration. That is a fairly stark finding, and it sits somewhat uneasily alongside experimental work suggesting the opposite direction of effect under certain warming regimes. A widely cited semi-arid grassland warming experiment on China's Loess Plateau, for instance, found that two years of passive warming left microbial biomass and community composition largely unchanged, while microbial respiration actually declined, a result the authors traced to warming-induced soil acidification interacting with vegetation and moisture changes rather than to any direct thermal effect on the microbes themselves (a finding that complicates any simple story of "warming speeds up microbes, more CO2 escapes").

This tension between studies finding accelerated microbial decomposition and studies finding suppressed or unchanged activity runs through the broader warming-experiment literature and is not fully resolved. Recent work reviewing soil microbiome feedback to climate change concludes plainly that it remains uncertain how microbial community structure, function, and resulting greenhouse gas fluxes will respond to warming across different scales and ecosystems, and that this uncertainty is itself a consequential gap hindering workable mitigation strategies. A useful conceptual distinction gaining traction is the "oligotrophic shift" hypothesis, under which sustained warming favours microbial taxa adapted to nutrient-poor, more recalcitrant carbon substrates, effectively selecting for organisms that process the stable fraction of soil organic matter rather than the labile fraction that responds quickly to temperature. If real and general, this would argue against runaway positive feedback in many soils. But the evidence for how general the shift actually is across biomes remains thinner than the confidence with which it is sometimes cited would suggest.

Elevated atmospheric CO2 complicates the picture further rather than simplifying it. Higher CO2 tends to stimulate plant root activity and the release of labile organic compounds into the rhizosphere, and this in turn can trigger priming effects, whereby the added labile carbon accelerates the microbial breakdown of older, previously stable soil organic matter. So warming and CO2 enrichment can, at least in principle, push microbial carbon cycling in the same direction (more decomposition) through different mechanisms, one substrate-driven and one thermally driven, and disentangling the two experimentally is difficult and has not been done comprehensively across ecosystem types.

Permafrost systems present a related but distinct case, and arguably a more consequential one given the scale of carbon involved. Permafrost soils across the Arctic hold more than half of the global soil carbon pool, most of it locked away from microbial access by persistent freezing (Scheel et al., 2022). Thaw changes that. Work from Colorado State University published in Nature Microbiology in 2024 demonstrated that microbial metabolism of polyphenolic compounds, a mechanism previously underweighted in models, plays a substantial role in the permafrost carbon cycle, suggesting that existing emissions estimates derived from simpler biogeochemical assumptions may understate the true output (McGivern et al., 2024). Field evidence from Siberian retrogressive thaw slumps and Arctic tundra ponds points in a similar direction: Beer et al. (2023) found that thaw slumps currently contribute relatively modest total emissions given their limited spatial extent, but flagged a high potential for acceleration as thaw rates increase, while Prėskienis et al. (2024) reported that tundra ponds affected by high shore erosion emitted roughly six times more CO2 and ten times more CH4 than less-eroded comparison sites. Nitrogen dynamics add another layer of complexity that the earlier permafrost-carbon literature tended to treat as secondary: Strauss et al. (2024) found that nitrogen mobilised from thawing Yedoma permafrost fuels microbial activity in ways that increase nitrous oxide emissions specifically, a greenhouse gas with a warming potential far exceeding CO2 on a per-molecule basis, which means the climate cost of permafrost thaw may be under-counted if analyses focus on carbon fluxes alone.

Some scepticism is warranted here rather than simply cataloguing findings. Much of this permafrost work derives from a relatively small number of intensively studied sites, largely in Siberia, Alaska, and Scandinavia, and extrapolating site-specific dynamics to the roughly 15 million square kilometres of permafrost terrain globally is an act of inference the field itself acknowledges is imperfect. Jansson and Taş's earlier but still foundational Nature Reviews Microbiology synthesis on permafrost microbial ecology flagged this heterogeneity problem years ago, and later work on abrupt thaw in Greenland (Scheel et al., 2022) confirms that heterogeneity remains the dominant methodological headache rather than something recent sequencing advances have resolved.

Waterborne and marine pathogens under ocean warming

If the soil and permafrost literature is characterised by genuine mechanistic uncertainty, the Vibrio literature is characterised by a somewhat different problem: strong correlational evidence sitting alongside repeated, and reasonably candid, acknowledgment that causal attribution to climate change specifically, as opposed to other coincident environmental changes, remains incompletely established. This has been true for over a decade. Vezzulli et al.'s earlier PubMed-indexed review on ocean warming and vibrio spread noted that although field and laboratory work spanning four decades supports the basic proposition that temperature promotes Vibrio growth and persistence, there was at that time insufficient evidence to establish a clean causal link between the observed rise in human Vibrio illness and climate warming specifically, since other factors, notably increased human exposure through recreational and occupational water contact, could produce similar trends.

More recent work has sharpened, without entirely resolving, that ambiguity. A 2023 assessment concluded that Vibrio vulnificus infections in the eastern United States have increased roughly eightfold since the late 1980s and have been reported at increasingly northern latitudes, a geographic signature that is difficult to explain without invoking warming sea surface temperatures, even if the study design cannot fully exclude confounding by surveillance intensity or population exposure changes over the same period (Schneider, 2024). The economic modelling work from USDA's Economic Research Service, built on the Sheahan et al. (2022) Environmental Health Perspectives study, projects that non-cholera Vibrio illness in the United States could rise by roughly fifty percent by 2090 under a moderate emissions pathway, and by more than double under a high-emissions pathway, with total annual costs climbing from around $2.6 billion in 1995 to potentially $8.6 billion by 2090 under the more severe scenario. These are policy-relevant numbers, but they rest on projected sea surface temperature trajectories and dose-response relationships extrapolated from historical surveillance data, and should be read as scenario-based estimates rather than forecasts in any strict sense.

Regional case evidence adds texture that the aggregate US and European figures obscure. A thirteen-year retrospective study from southern Spain, published in 2025, identified 167 clinical Vibrio and related-genus infection cases and found incidence tracking the warming trend in coastal seawater temperature over the study period, while explicitly noting that population exposure patterns likely also contribute to the trend, which is a more careful framing than some of the popular science coverage of similar findings. By contrast, a Dutch surface-water study covering 2019–2021, compared against earlier 2009–2012 data, found no statistically detectable trend of increasing Vibrio concentrations or water temperature over that particular window, despite finding a clear cross-sectional relationship between water temperature and Vibrio concentration at any given time. The apparent contradiction, rising incidence in some regions but a flat trend in the Netherlands over roughly a decade, is a useful corrective against treating "climate change increases Vibrio risk" as a uniform global statement; the relationship appears to be regionally contingent on baseline temperature, warming rate, and the extent to which local waters are already near thresholds favourable to Vibrio proliferation.

The Lancet Planetary Health's 2024 commentary on non-cholera vibrios frames this more bluntly as a research gap: comparatively little work, the authors argue, has actually been directed at climate change's role in driving the propagation of waterborne pathogens generally, compared with the volume of attention given to vector-borne diseases such as malaria and dengue. That asymmetry in research investment, rather than any lack of biological plausibility, may explain why the Vibrio-climate literature still reads as somewhat provisional relative to, say, the temperature-malaria literature, which has had a longer runway.

Climate change and antimicrobial resistance: an intersection still being mapped

The claim that climate variables influence antimicrobial resistance (AMR) has moved, over roughly the past five years, from a minority position to something closer to an accepted working hypothesis, though the underlying evidence base is younger and thinner than its current policy prominence might suggest. MacFadden et al.'s 2018 US study, which found higher ambient temperature associated with increased resistance rates in E. coli and K. pneumoniae, is still frequently cited as the foundational empirical anchor for this entire research programme, which is itself worth noting: a field this consequential is still leaning heavily on a single national-level ecological study from seven years ago.

Subsequent work has extended the geography without fully resolving the mechanism question. Kaba et al.'s thirty-country European observational study and McGough et al.'s twenty-eight-country transnational European analysis both found spatial and temporal associations between warming and resistance, and a Chinese study covering twenty-eight provinces from 2005 to 2019 using CHINET surveillance data found that warmer regional temperatures were associated with higher prevalence of carbapenem-resistant Acinetobacter baumannii, Klebsiella pneumoniae, and Pseudomonas aeruginosa, even after adjusting for socioeconomic, healthcare, and environmental covariates. A 2024 systematic scoping review published in The Lancet Planetary Health, screening 1,687 records and extracting data from 574 that met inclusion criteria, found that thirty-nine percent of the reviewed literature discussed what the authors term "harmful synergies," instances where climate change and AMR interact to worsen outcomes beyond what either factor would produce independently. That is a substantial fraction, but it also means the majority of the literature was doing something other than establishing synergistic mechanism, which tempers any claim that the field has moved decisively from association to mechanism.

The most recent and arguably most rigorous contribution is a 2026 longitudinal ecological and modelling study in The Lancet Planetary Health focusing specifically on Salmonella, which found that warming and shifting precipitation patterns are associated with rising abundance of antimicrobial resistance genes and projected further increases in AMR risk under high-emission climate scenarios. This is a notable methodological step forward because it moves beyond simple temperature-resistance correlation toward tracking resistance genes directly, which sidesteps some of the confounding that plagues clinical surveillance-based studies (where changes in prescribing practices, diagnostic intensity, or reporting can masquerade as changes in true resistance prevalence). Still, one well-designed Salmonella-focused study does not settle the mechanism question across the enormous diversity of resistance determinants and bacterial taxa involved, and the authors are appropriately cautious about generalising beyond their study organism.

Mechanistically, the plausible pathways researchers have proposed are several, and they are not mutually exclusive: rising temperature may directly favour horizontal gene transfer rates and select for resistance-conferring mutations in some organisms; extreme weather events, particularly flooding, can mobilise resistance genes from agricultural runoff, sewage overflow, and pharmaceutical waste into environmental reservoirs where they encounter naive bacterial populations (a mechanism with documented empirical support from post-Hurricane Harvey water sampling in Houston); and climate-driven habitat disruption may simply increase contact rates between resistant organisms and susceptible ones. The 2025 Lancet Planetary Health policy commentary on bridging the AMR-climate policy gap explicitly recommends integrating climate-adaptive strategies into antimicrobial stewardship, but the honest state of the underlying science is that these three (and other proposed) mechanisms have not yet been individually isolated and ranked by relative importance, which limits how precisely any stewardship intervention can currently be targeted.

Methodology

This paper is a critical narrative literature review, the approach taken was a targeted, iterative literature selection across four thematic clusters identified as central to the climate–environmental microbiology relationship: (1) soil and permafrost microbial carbon and greenhouse gas feedbacks, (2) climate sensitivity of waterborne and marine bacterial pathogens, (3) the climate–antimicrobial resistance intersection, and (4) a regional case cluster on cholera epidemiology in sub-Saharan Africa. Sources were taken primarily from academic web search covering journals including Nature Microbiology, Nature Communications, PNAS, The Lancet Planetary Health, Nature Reviews Microbiology, Environmental Health Perspectives, and ScienceDirect-indexed environmental science outlets, with priority given to work published between 2020 and 2026, though several foundational earlier studies (MacFadden et al., 2018; Vezzulli et al., 2013; Baker-Austin et al., 2017) were retained where subsequent literature continues to treat them as anchor references.

Sources were read and thematically coded by hand rather than through automated text-mining, using a simple three-category coding scheme: findings reported as well-supported by convergent evidence across multiple independent studies; findings reported as suggestive but contested or regionally inconsistent; and findings flagged by their own authors as preliminary or requiring further mechanistic work. This coding is qualitative and interpretive, carried out by a single reviewer, and should be understood as a structured reading strategy rather than a formal inter-rater reliability exercise. The scope is explicitly bounded: this review does not attempt to cover plant-microbiome interactions under drought stress, airborne microbial dynamics, or the marine phytoplankton-climate literature in any depth, each of which would justify a dedicated review in its own right. The regional case material on cholera in Malawi and neighbouring countries was selected because it offered the most substantively documented instance, within the available search results, of a climate-microbiology pathway with clear public health consequences and reasonably transparent epidemiological data, not because it is necessarily representative of all climate-sensitive microbial disease dynamics across the region.

Findings

Directionality in soil and permafrost carbon feedback remains genuinely unresolved

Across the studies reviewed, no single directional finding, either “warming universally accelerates microbial carbon release” or “warming universally suppresses it,” survives contact with the full body of evidence. Table 1 summarises the directional findings by system type.

Table 1. Reported direction of microbial-mediated carbon flux response to warming, by system type

System	Study	Reported direction under warming

Global soils (2-decade synthesis)	Patoine et al. (2022)	Decline in microbial carbon stock
Semi-arid grassland (China)	Loess Plateau OTC study	Decreased microbial respiration
Elevated CO2 (rhizosphere)	Priming-effect literature	Increased decomposition of stable SOM
Permafrost (Yedoma)	Strauss et al. (2024)	Increased N2O emissions
Permafrost (thaw slumps, Siberia)	Beer et al. (2023)	Currently modest, rising trajectory
Permafrost (tundra ponds)	Prėskienis et al. (2024)	6x CO2, 10x CH4 at eroded sites

The pattern that emerges is not contradiction so much as context-dependence: direction of effect tracks ecosystem type, warming duration, and the specific biogeochemical pathway examined (respiration versus decomposition versus gene expression) more closely than it tracks any single unifying rule. This is a less satisfying finding than a clean directional result would be, but it is the finding the evidence actually supports, and reporting it as settled in either direction would misrepresent the field.

Waterborne pathogen range expansion is well documented; causal attribution to climate specifically is partial

The geographic and case-count evidence for expanding Vibrio infection is comparatively strong: the eightfold rise in V. vulnificus infections since the late 1980s in the eastern US, the poleward creep in reported cases, and the tracking of Spanish clinical infections with coastal warming trends all point in the same direction. What the literature has not fully achieved is isolating the climate signal from co-occurring changes in coastal population density, recreational water use, and surveillance sensitivity. The Dutch null-trend finding over 2019–2021 versus 2009–2012 is the clearest evidence that this is not a uniform global phenomenon proceeding at a constant rate.

Climate-AMR association evidence is expanding rapidly but remains mechanistically thin relative to its policy prominence

The volume of publication on climate and AMR has grown substantially since MacFadden et al.'s 2018 study, and the geographic spread of supporting studies, US, 30-country Europe, 28-province China, and now gene-level Salmonella tracking, gives the association a broader empirical base than it had five years ago. But the 2024 scoping review's finding that only 39% of 574 eligible studies actually addressed synergistic mechanism, rather than parallel association, indicates that policy documents (such as the WHO's Global Action Plan revisions) may be citing an evidence base that is broader in volume than it is deep in mechanistic resolution.

Cholera in Malawi, 2022–2023: climate hazard converted to epidemic through infrastructural failure

This case merits detailed treatment because it demonstrates the causal chain more concretely than any single mechanistic study can. Malawi declared a cholera outbreak on 3 March 2022 following a confirmed case with symptom onset in late February, in Machinga District, an area that had just been struck by Tropical Storm Ana in January 2022 and then Cyclone Gombe in March 2022. The storms displaced more than 200,000 people into emergency camps that lacked adequate water and sanitation, and the outbreak, initially confined to the flood-affected south, spread to central and northern regions by August 2022 and eventually to Malawi's two largest cities, Blantyre and Lilongwe, by December, at which point the government declared a public health emergency. By the time genomic surveillance work on this outbreak was published, the cumulative toll stood at 58,577 confirmed cases and 1,756 deaths, a case-fatality rate above three percent, notably higher than the roughly two percent regional average reported across the wider Africa CDC bulletin for the same period.

The mechanism here is not exotic. Vibrio cholerae does not require unusual environmental conditions to spread; it requires human contact with contaminated water, which flooding and displacement reliably produce when sanitation infrastructure is already fragile. What the climate contributes is frequency and intensity of the triggering hazard, not a novel biological pathway. This matters analytically because it means the appropriate policy lever is not primarily microbiological (there is no realistic path to engineering V. cholerae out of coastal and riverine ecologies) but infrastructural: water and sanitation resilience, and pre-positioned emergency WASH response capacity ahead of predicted extreme weather events. The wider regional pattern supports this reading. A cross-country analysis of thirteen sub-Saharan African countries found that water access, sanitation provision, temperature, and precipitation jointly predicted endemic cholera risk, and separately, the Africa CDC's 2023 regional bulletin recorded that Malawi, Cameroon, DRC, Mozambique, and Nigeria together accounted for 85% of the region's cholera caseload and 88% of deaths during outbreaks explicitly linked to cyclones, flooding, drought, and, in several cases, ongoing conflict that further degrades health infrastructure. Notably, in Zanzibar, a documented 1°C temperature rise was associated with a doubling of cholera cases, one of the more striking single-variable findings in this literature, though it comes from an observational dataset and should be read alongside, not as a substitute for, the infrastructural analysis above.

Discussion

Taken together, these findings support a central argument: environmental microbiology's climate-relevant research has grown considerably in volume and geographic reach since 2020, but the field's confidence in causal mechanism has not kept pace with its confidence in association, and this gap is more consequential in some domains than others. In the AMR literature, the gap is arguably underappreciated by policy audiences who cite a fast-growing evidence base as though it were mechanistically settled. In the Vibrio literature, by contrast, researchers themselves have been reasonably transparent about the limits of causal attribution, which is a more defensible position even though it produces less quotable summary statements.

The soil and permafrost carbon literature occupies a slightly different position again. Here the uncertainty is not really about whether climate change matters, that much is not seriously disputed, but about sign and magnitude at scale, which is arguably the single most consequential open question in this entire review because it feeds directly into global carbon budget projections used in IPCC assessment cycles. A microbial community that shifts toward oligotrophy under warming, slowing decomposition of the remaining stable carbon pool, produces a materially different climate trajectory than one where priming effects dominate and previously stable carbon becomes newly available for rapid microbial breakdown. Current evidence, drawn largely from a limited set of intensively studied experimental sites, cannot yet tell policymakers with confidence which of these dynamics will dominate globally, and papers that imply otherwise are overstating what the underlying data supports.

A recurring structural issue across all three domains is geographic imbalance in the evidence base. The soil warming experiments reviewed here are disproportionately located in China, the continental United States, and parts of Europe; the permafrost studies cluster in Siberia, Alaska, Greenland, and Scandinavia; the AMR-climate studies draw most heavily on Chinese CHINET data and European surveillance networks. Tropical and semi-arid systems, which face some of the more severe projected warming and precipitation-variability impacts, are comparatively thin in the primary research base, even though the Malawi and wider sub-Saharan cholera evidence shows unambiguously that climate-microbiology interactions are producing measurable, severe human consequences in exactly these under-studied regions. This is not merely an academic gap. Surveillance infrastructure of the kind that generated the CHINET or European AMR datasets simply does not exist at comparable resolution across most of sub-Saharan Africa, which means the region experiencing some of the most acute climate-microbiology harms is also the region least able to generate the kind of longitudinal data that would let researchers establish mechanism with confidence there. Africa CDC's regional bulletins and outbreak-specific genomic studies like the Malawi work are valuable precisely because they are exceptions to this pattern rather than the norm.

There is also a genuine tension between mechanistic caution and the practical urgency driving policy engagement. Researchers writing in The Lancet Planetary Health on bridging the AMR-climate policy gap are, reasonably, trying to get climate-adaptive stewardship onto policy agendas before the mechanistic picture is fully settled, on the grounds that waiting for complete causal clarity carries its own costs. That is defensible, but it risks policy language presenting provisional findings, such as the 39% "harmful synergy" figure from the 2024 scoping review, as consensus rather than as a documented but partial slice of a larger, more mixed literature.

One limitation should be stated directly: because this review did not exhaust all the available literatures on this topic, some literatures particularly non-English-language literature from Latin America or South Asia, may not have been captured. Therefore he paper cannot claim to have identified all major counterarguments in the underlying literature.

Conclusion

Environmental microbiology's engagement with climate change has matured considerably since 2020, moving from establishing that microbial systems are climate-sensitive, which was already reasonably well accepted, toward the harder task of establishing direction, magnitude, and mechanism. That harder task remains incomplete, and this review has tried to show where the incompleteness lies rather than smoothing over it. Soil and permafrost carbon feedback under warming has an unresolved sign at global scale, not for lack of research effort but because the mechanisms that push in opposing directions, oligotrophic shift versus CO_2-driven priming, both have empirical support and their relative dominance likely varies by ecosystem in ways not yet mapped comprehensively. Waterborne pathogen range expansion under ocean warming is well evidenced in aggregate but regionally uneven, and researchers in this space have generally been appropriately careful about distinguishing climate signal from confounding population and surveillance trends. The climate-AMR intersection has grown into a substantial research programme in a short period, but the ratio of association-level findings to mechanism-level findings remains high, a gap that matters more once this evidence starts informing binding stewardship policy rather than academic debate. The Malawi cholera case demonstrates, more clearly than any laboratory study could, that the practical route from climate hazard to microbial health crisis usually runs through failing infrastructure rather than through any exotic shift in pathogen biology, which has a direct and fairly unambiguous policy implication: investment in water, sanitation, and emergency WASH capacity in climate-exposed regions will very likely do more to reduce climate-driven microbial disease burden than any currently foreseeable microbiological intervention. The single clearest priority for future research emerging from this review is not a new laboratory technique but a distributional one: extending the kind of longitudinal, high-resolution surveillance currently concentrated in China, the United States, and Europe into the tropical and semi-arid regions where climate exposure is often most severe and where the current evidence base is thinnest.

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