CLIMATE CHANGE AND DISEASE EMERGENCE

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ABSTRACT

The relationship between a warming planet and the rise of infectious disease has moved, over the past two decades, from speculation to one of the defining public-health questions of the century. This review synthesises what is now known about how climate change reshapes the emergence, re-emergence, and geographic redistribution of human pathogens. It begins with the conceptual machinery linking climate to disease: the temperature- and moisture-sensitive biology of vectors, pathogens, hosts, and reservoirs, and the way extreme events and gradual warming alter each link in the transmission chain. The review then traces the evidence across the major disease groups, mosquito- and tick-borne infections, water- and food-borne illness, and wildlife-origin zoonoses, and examines the regional patterns and emerging hotspots that modelling has begun to map. Considerable attention is given to the genuine scientific controversy that surrounds attribution, including the non-linear, hump-shaped response of transmission to temperature, the powerful confounding role of poverty, urbanisation, and control programmes, and well-documented cases in which warming has coincided with disease decline rather than rise. From this evidence, three conclusions emerge. First, climate is one driver among several, and its fingerprint is clearest at the cool and hot margins of a pathogen's range. Second, the dominant near-term effect is redistribution, shifting risk poleward and upward into populations and health systems that may be unprepared, rather than a uniform global increase. Third, the breadth of pathways through which climatic hazards aggravate disease is too large for adaptation alone, underscoring the need to couple resilient surveillance and health systems with reductions in greenhouse-gas emissions. The review closes by setting out practical priorities for early-warning systems, integrated One Health surveillance, and equitable adaptation, alongside the knowledge gaps that most constrain prediction.

Keywords: climate change; emerging infectious disease; vector-borne disease; zoonotic spillover; disease ecology; planetary health; One Health; adaptation and mitigation.

Introduction

Infectious disease has always been a creature of its environment. The seasons of influenza, the monsoon timing of cholera, the altitude ceiling that once held malaria below the East African highlands, all betray an intimate dependence of pathogens, and the organisms that carry them, on heat and water. What has changed in the modern era is that humanity is now altering those very conditions at a planetary scale. The recognition that anthropogenic climate change could therefore rewrite the map of human disease has grown into a substantial scientific field, drawing together epidemiologists, ecologists, climatologists, and public-health practitioners who once worked apart (Patz et al., 2005).

The stakes are not abstract. Early estimates attributed on the order of 150,000 deaths each year to the warming and precipitation shifts of the preceding decades, with the burden falling disproportionately on regions least responsible for the emissions driving the change (Patz et al., 2005). Many common human illnesses, from the cardiovascular toll of heatwaves to malnutrition following crop failure and the altered

transmission of infections, are sensitive to climatic fluctuation (McMichael et al., 2006). A comprehensive synthesis later found that more than half of the infectious diseases known to afflict humanity, some 218 of 375, have at some point been aggravated by climatic hazards sensitive to greenhouse-gas emissions, through more than a thousand distinct pathways (Mora et al., 2022). The sheer number of these connections is itself a central finding: climate does not touch disease through a single mechanism but through a web of them. Reviews spanning the viral, bacterial, and parasitic agents involved reach the same broad conclusion: warming and the extreme weather that accompanies it are expected to drive both the emergence of new infections and the re-emergence of old ones (El-Sayed & Kamel, 2020).

Yet the field has matured beyond simple alarm. A polarising debate ran through its first decade, particularly over human pathogens, where socio-economic drivers and control measures can mask or mimic the signal of climate (Altizer et al., 2013). Some of the most influential work has been openly cautionary, pointing out that although the globe is warmer than a century ago, the evidence that climate change has *already* driven disease increases is more equivocal than headlines suggest, and that responses are frequently non-linear (Lafferty, 2009). This review takes that tension seriously. Its aim is neither to overstate nor to dismiss the climate–disease link but to lay out, as evenly as the literature allows, what is well established, what remains contested, and what can be done. The structure moves from mechanism to evidence to response, beginning with the system through which climate acts (Figure 1).

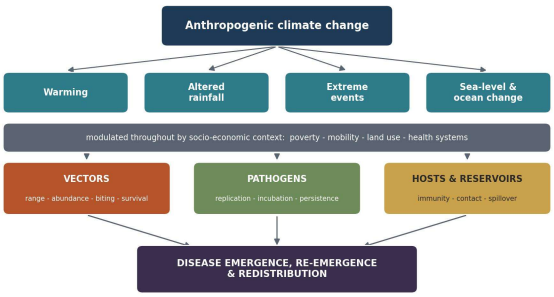

Fig. 1: The climate–host–pathogen–vector system through which a warming climate reshapes the geography and intensity of infectious disease.

A Conceptual Framework: How Climate Acts on Disease

To understand how climate change alters disease, it helps to see transmission as a small ecosystem. A pathogen must complete its life cycle, often inside a cold-blooded vector such as a mosquito or tick, and must pass between hosts whose own susceptibility and behaviour vary. Because arthropod vectors are ectothermic, their survival, reproduction, biting frequency, and geographic range are governed directly by temperature, while rainfall and humidity shape the breeding sites and shelter on which they depend (Githeko et al., 2000). The pathogen itself is equally exposed: warmth typically accelerates its replication and shortens the extrinsic incubation period, the time a virus or parasite needs to mature inside a vector before that vector becomes infectious (Gould & Higgs, 2009).

Warming can therefore act on several links at once. It may speed pathogen development, raise vector survival and transmission rates, and increase host susceptibility, producing synergies capable of reshaping disease risk across both terrestrial and marine systems (Harvell et al., 2002). The classic review of climate and human infections framed these influences as operating through the pathogen, the host, and the transmission process simultaneously, and emphasised that society's capacity to adapt is itself part of the system (Wu et al., 2016). Crucially, the same review and others stress that the outcome is rarely a simple amplification; the direction and magnitude depend on the specific host–pathogen pairing and the form the climatic change takes (Altizer et al., 2013).

Two further features of this framework deserve emphasis. First, the relationships are characteristically non-linear: latitudinal, altitudinal, seasonal, and inter-annual associations between climate and disease, together with historical and experimental evidence, indicate that climate influences infection in a non-linear fashion rather than through smooth proportionality (Lafferty, 2009). Second, the biological signal is never isolated. It is filtered through the socio-economic context, mobility of people and goods, the availability of drugs and vaccines, sanitation, and the strength of public-health systems, all of which can dominate the climatic contribution (Rohr et al., 2011). Any honest account of climate and disease must hold these two truths, mechanistic plausibility and contextual contingency, together.

Mechanisms Linking Warming to Emergence

Temperature and the hump-shaped response of transmission

Perhaps the single most important refinement of recent years is the recognition that transmission does not rise indefinitely with temperature. Trait-based models that assemble the thermal responses of vector and pathasite vital rates show that transmission potential is unimodal, climbing from a lower

threshold, peaking at an intermediate optimum, and falling away again as heat begins to kill vectors and suppress the traits on which transmission depends (Mordecai et al., 2019). Synthesising eleven pathogens carried by fifteen mosquito species, this work placed the optimum for many systems in the range of roughly 23 to 29 degrees Celsius (Mordecai et al., 2019).

For the arboviruses of greatest current concern, the picture is similar. Mechanistic models of Zika, dengue, and chikungunya transmission by *Aedes aegypti* and *Aedes albopictus*, validated against human case data across the Americas, found that transmission occurs between about 18 and 34 degrees Celsius and peaks between 26 and 29 degrees (Mordecai et al., 2017). The implication is profound and is illustrated in Figure 2: in cool places near the lower edge of this curve, warming pushes conditions toward the optimum and can increase risk; in already-hot places near the upper edge, further warming pushes conditions past the optimum and may *reduce* it. A regional analysis reached a complementary conclusion decades earlier, noting that the greatest climate effect on transmission is felt at the temperature extremes of a disease's range, roughly 14 to 18 degrees at the lower bound and 35 to 40 degrees at the upper (Githeko et al., 2000).

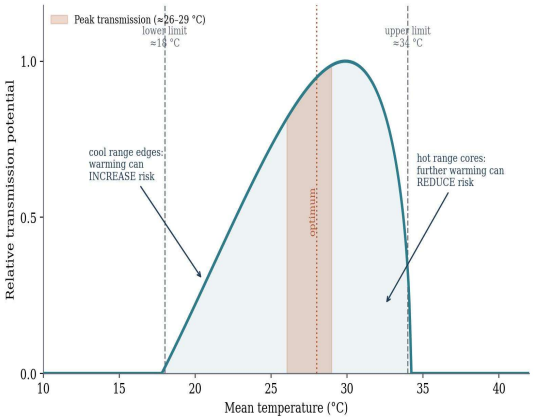

Fig. 2: The temperature dependence of mosquito-borne transmission is hump-shaped: warming raises risk at cool range edges but can lower it where temperatures already exceed the optimum.

Rainfall, humidity, and extreme weather

Moisture is the second great lever. Rainfall creates and destroys the standing water in which mosquitoes breed, humidity governs vector survival and pathogen persistence, and vapour pressure, a measure of atmospheric moisture, proved able to reproduce the global distribution of dengue with striking accuracy (Hales et al., 2002). But the influence of water on disease is double-edged. Drought can concentrate both people and vectors around scarce water sources, while floods can contaminate drinking supplies and displace populations (Wu et al., 2016).

Extreme weather events have a distinctive role because they act as pulses rather than trends. They can create the conditions for clustered outbreaks of insect-, rodent-, and water-borne disease, as ranges shift in altitude alongside retreating glaciers and changing plant communities (Epstein, 2001). The waterborne route is especially climate-sensitive: the dynamics of *Vibrio cholerae* in the environment are controlled by water temperature, salinity, and plankton abundance, which are in turn driven by larger-scale climate variability such as the El Niño–Southern Oscillation, making cholera a model system for studying climate and infection (Lipp et al., 2002). As the burden of these hazards mounts, their convergence on human health has become measurable across a suite of indicators tracking exposure, vulnerability, and the climatic suitability for transmission (Watts et al., 2021).

Range shifts and the redistribution of risk

Where conditions become newly suitable, vectors and pathogens follow. Reviews of recent decades document measurable shifts in vector and pathogen distribution in temperate, peri-Arctic, Arctic, and tropical highland regions, changes that had been anticipated by modellers and are now being observed (Caminade et al., 2019). The spread of the two principal arbovirus vectors illustrates the process: the distributions of *Aedes aegypti* and *Aedes albopictus* are driven jointly by human movement and climatic suitability, and statistical mapping shows their continuing expansion in Europe and the United States, with further spread projected under urbanisation, connectivity, and warming (Kraemer et al., 2019).

Modelling the future burden of *Aedes*-borne viruses suggests that climate change will both add and shift risk. Under more severe scenarios, close to a billion people could face their first exposure to transmission by one of these mosquitoes, concentrated in Europe and in high-elevation tropical and subtropical zones, even as the deep tropics may eventually become too hot for the more heat-limited *Ae. albopictus*, marking a global shift toward seasonal rather than year-round risk (Ryan et al., 2019). Tick-borne disease tells a parallel story in the temperate north: both phenomenological and mechanistic models point to substantial range expansion of *Ixodes* ticks and the pathogens they carry, including the agents of Lyme disease, as the climate warms (Ostfeld & Brunner, 2015). Across these examples the dominant near-term signature of climate change is not uniform escalation but *redistribution*, the movement of risk into new latitudes and altitudes (Caminade et al., 2019).

Spillover and cross-species transmission

The most consequential emergence events of the modern era have come from wildlife. An analysis of 335 emerging-disease events between 1940 and 2004 found that the majority were zoonotic, that most of these originated in wildlife, and that such events have risen over time, even as global surveillance effort remained poorly matched to where new diseases are most likely to arise (Jones et al., 2008). Climate change adds a new dimension to this picture by forcing species into novel contact. As mammals track shifting climate and land use, they are projected to aggregate in new combinations at high elevations, in biodiversity hotspots, and in densely populated parts of Asia and Africa, driving an estimated several thousand first-time instances of cross-species viral sharing by 2070 and creating fresh opportunities for spillover into humans, with bats playing an outsized role (Carlson et al., 2022).

This reframes climate change as a driver not only of where existing diseases occur but of where genuinely new ones may emerge. Set against the broader backdrop of global change, climate acts alongside urbanisation and the doubling of air travel since 2000 to raise the risk of outbreaks, even as improved sanitation and health care have driven progress against many established infections (Baker et al., 2022). The COVID-19 pandemic, while not attributable to climate, sharpened scientific and public attention on the broad environmental drivers of emergence within which climate is embedded (Baker et al., 2022).

Evidence Across the Major Disease Groups

Mosquito-borne disease: malaria and the arboviruses

Malaria has been the central battleground of the climate–disease debate. The intuitive expectation, that warming should extend the disease into cooler highlands and increase its incidence, was endorsed in early assessments and remains biologically plausible at altitude (Githeko et al., 2000). Yet two of the most cited studies in the field complicate that expectation, and they are essential to an honest review. An analysis of long-term meteorological records at four high-altitude East African sites, where malaria had resurged, found no significant change in temperature, rainfall, vapour pressure, or the number of months suitable for transmission over the relevant period, implying that factors other than climate drove the resurgence (Hay et al., 2002).

The challenge to a simple warming-equals-more-malaria narrative went further still. By comparing a modern map of *Plasmodium falciparum* with one from around 1900, before major control efforts, researchers quantified a dramatic century-long contraction of malaria's range and intensity during a period of unequivocal global warming, and concluded that the effect of rising temperature is at least an order of magnitude weaker than that of control measures and economic development (Gething et al., 2010). The lesson is not that temperature is irrelevant but that it operates within a far more powerful set of social and programmatic forces.

For the *Aedes*-borne arboviruses, dengue, Zika, and chikungunya, the climate signal is clearer, in part because these viruses have expanded rapidly in recent decades. Empirical modelling of dengue based on humidity reproduced its current limits well and projected a substantial increase in the population at risk under climate and population change (Hales et al., 2002). More recent mechanistic and multi-model work refines this, showing temperature-bounded transmission windows and projecting that warming will lengthen the transmission season and enlarge the population at risk of both dengue and malaria, particularly in densely populated urban areas of Africa, South-East Asia, and the Americas, and at higher altitudes where populations may be immunologically naive (Colón-González et al., 2021).

Tick-borne and other vector-borne disease

In the temperate world, tick-borne infections are among the clearest illustrations of climate-driven redistribution. The evidence that warming is changing the distribution of *Ixodes* ticks and their pathogens has been carefully reviewed; while laboratory thresholds for tick survival rarely translate cleanly to the field, both modelling approaches converge on the expectation of marked range expansion of ticks and tick-borne disease, including Lyme disease, the most common vector-borne infection of the United States and Europe (Ostfeld & Brunner, 2015). More broadly, the European experience shows infections arriving through multiple routes, arthropod, rodent, water, food, and air, with incidence, prevalence, and distribution all projected to shift in a changing climate (Semenza & Menne, 2009).

The arboviruses beyond the *Aedes* group reinforce the theme. Patterns of emerging arbovirus disease have changed markedly over the past half-century, and climate is a major determinant of the distribution of arthropods, the characteristics of their life cycles, the dispersal and even the evolution of the viruses they carry, and the efficiency of transmission to vertebrate hosts (Gould & Higgs, 2009). Many of these conditions fall among the neglected tropical diseases, whose life cycles are sensitive to environmental factors across air, water, and soil, making the future geography of dozens of infections likely to respond to climate change over seasonal,

annual, and decadal horizons, though decadal projections remain comparatively under-studied (Booth, 2018).

Water-, food-, and air-borne disease

Not all climate-sensitive infections travel by vector. Cholera remains the archetype of a directly climate-coupled disease: the abundance of *Vibrio cholerae* in coastal and estuarine waters tracks temperature, salinity, and plankton blooms, themselves tied to regional climate cycles, so that the disease has a historical association with particular seasons and biogeographic zones (Lipp et al., 2002). Beyond cholera, climate influences the survival and transmission of many water- and food-borne pathogens through its effects on temperature and the hydrological cycle, with flooding and drought both capable of raising risk (Wu et al., 2016).

Air-borne and seasonal infections add a further layer. Temperature and humidity shape the survival and transmission of respiratory pathogens, and shifting climatic conditions can alter their seasonal timing and reach (Semenza & Menne, 2009). Taken together with the vector-borne and zoonotic evidence, the breadth of transmission routes affected explains why a systematic synthesis could implicate climatic hazards in the majority of human pathogenic diseases through such a large number of distinct pathways (Figure 3) (Mora et al., 2022).

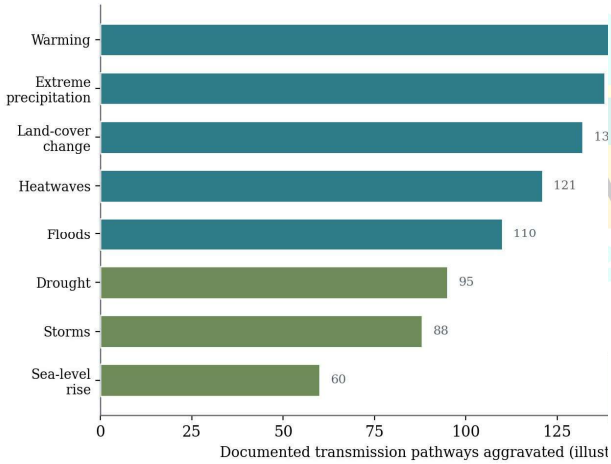

Fig. 3. Multiple greenhouse-gas-sensitive hazards converge on infectious disease; the schematic follows the structure of a large systematic synthesis in which more than half of human pathogenic diseases were aggravated through roughly a thousand pathways.

Complexity, Controversy, and Confounding

A review that presented only the case for climate-driven disease increase would misrepresent the science. The field is marked by genuine and productive disagreement, and three sources of complexity recur. The first is non-linearity. Because transmission peaks at intermediate temperatures, the same degree of warming can raise risk in one place and lower it in another, so that regional predictions cannot be read off the global trend (Mordecai et al., 2019). The malaria record makes this concrete: warming did not prevent a century-long retreat of the disease, and over-attributing its dynamics to climate risks misdirecting control resources (Gething et al., 2010).

The second is confounding by social and economic change. Detecting a climate signal in human disease is hard precisely because socio-economic drivers and deliberate control measures can both obscure and counterfeit it, a difficulty at the heart of the field's polarised debate (Altizer et al., 2013). The early caution that there was, at the time, little firm evidence that climate change had already favoured infectious disease, despite clear warming, was a deliberate corrective to overconfident claims (Lafferty, 2009). Resolving such controversies, this line of argument holds, requires better integration of data and models, closer attention to confounders and context dependence, and the application of metabolic theory to host–parasite systems, not louder assertion (Rohr et al., 2011).

The third is the seeming paradox of parasite extinction. If warming pushes some systems beyond their thermal optimum, climate change should in places *reduce* disease even as it increases it elsewhere, an expectation that sits uneasily with blanket predictions of rising infection and that the field has had to confront directly (Rohr et al., 2011). The synthesis showing that most human pathogenic diseases can be aggravated by climatic hazards also found that a meaningful minority were at times diminished, underlining that the net effect is a balance of opposing tendencies rather than a single direction (Mora et al., 2022). None of this dissolves the climate–disease link; it disciplines it, locating the clearest effects at range margins and insisting that climate be weighed alongside the other drivers of emergence (Jones et al., 2008).

Modelling, Prediction, and Early Warning

If the relationships are complex, prediction becomes both harder and more valuable. The trajectory of the field has been toward mechanistic, trait-based models that derive transmission from the measured thermal responses of vectors and pathogens, rather than from correlation alone, because such models can be transported to new conditions and tested against independent case data (Mordecai et al., 2017). The call for a predictive framework that integrates ecophysiology and community ecology with

modelling, and that anticipates and monitors disease trends rather than merely reacting to them, has become a unifying goal of the field (Altizer et al., 2013).

Multi-model, multi-scenario approaches now provide the most credible projections by spanning several disease models, climate models, and emissions pathways, allowing the uncertainty itself to be characterised; such an intercomparison projected lengthening transmission seasons and growing populations at risk of malaria and dengue, with notable expansion toward higher altitudes and temperate regions (Colón-González et al., 2021). Mapping the future spread of the *Aedes* vectors similarly combines climatic suitability with human mobility and urbanisation to forecast distributions under alternative scenarios (Kraemer et al., 2019). The accumulating output of these efforts now feeds annual indicators that translate climatic suitability for transmission into a form usable by policy-makers (Watts et al., 2021). The consistent recommendation that follows is the construction of proactive, integrated early-warning systems that link meteorological, entomological, and epidemiological data, rather than passive surveillance after outbreaks occur (Semenza & Menne, 2009).

Practical Applications: From Evidence to Action

The practical value of this science lies in what it allows societies to do. Because climate change is already affecting the transmission and spread of vector-borne disease and its impacts are expected to worsen, the operative conclusion is that prevention and control efforts must be intensified now rather than deferred (Rocklöv & Dubrow, 2020). Four lines of action follow from the evidence, and they are summarised in Figure 4.

Surveillance and early warning: The recurring recommendation is to build integrated networks that connect epidemic intelligence with meteorological, entomological, water-quality, and remote-sensing data, enabling outbreaks to be anticipated and pre-empted (Semenza & Menne, 2009). Because risk is shifting into new latitudes and altitudes, surveillance must extend to places with no recent history of a given disease, where both populations and clinicians may be unprepared (Ryan et al., 2019).

Vector and environmental management: Managing the breeding habitat created and destroyed by rainfall and flooding, and adapting vector-control programmes to shifting seasons and ranges, remains a frontline defence; the persistence of strong control effects in the malaria record is a reminder that such interventions can outweigh the climatic signal (Gething et al., 2010). Water and sanitation infrastructure is the corresponding defence against the climate-coupled waterborne diseases (Lipp et al., 2002).

Health-system strengthening and One Health integration: Because the burden falls hardest on regions with the least capacity to respond, and because so many emerging threats arise at the interface of wildlife, livestock, and people, resilient health systems and integrated One Health surveillance that spans human, animal, and environmental health are essential (Jones et al., 2008). Adaptation must be matched to the social factors, mobility, land use, and poverty, that govern vulnerability (Wu et al., 2016).

Mitigation at the source: The most distinctive practical conclusion of the recent literature is that the diversity of pathways through which climatic hazards aggravate disease is simply too large for comprehensive societal adaptation, which points to the need to work at the source of the problem by reducing greenhouse-gas emissions (Mora et al., 2022). Framed as converging crises, the climate and health agendas are increasingly understood as one, and many responses to climate change carry direct co-benefits for health (Watts et al., 2021).

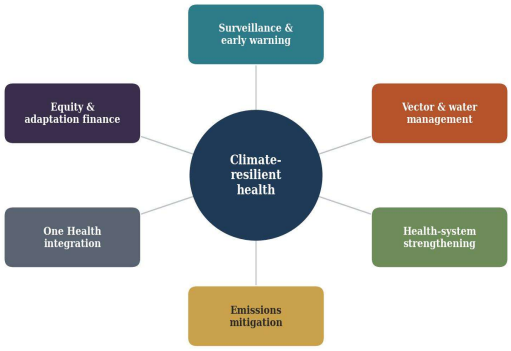

Fig. 4: A One Health response combines proactive adaptation, surveillance, vector and water management, health-system strengthening, and equitable finance, with greenhouse-gas mitigation that addresses the problem at its source.

Knowledge Gaps and Future Directions

Several gaps continue to constrain the field. The first is attribution. Disentangling the climatic contribution from the powerful influence of socio-economic change and control programmes remains difficult, and progress depends on longer, higher-quality data sets and on study designs capable of separating these drivers (Patz et al., 2005). The second is the integration of data and models: closer linkage of observational and mechanistic approaches, better treatment of confounders and context dependence, and the application of ecological theory to host–parasite

systems are needed to sharpen prediction (Rohr et al., 2011).

The third gap concerns the long view. While seasonal and inter-annual relationships are comparatively well characterised, the decadal-scale projections most relevant to planning, especially for the neglected tropical diseases, remain sparse and uncertain (Booth, 2018). The fourth is the frontier of emergence itself: predicting where climate- and land-use-driven changes in wildlife communities will create new opportunities for viral spillover is still in its early stages, yet it may prove the most consequential question of all (Carlson et al., 2022). Finally, the field continues to call for a unifying predictive framework that anticipates and monitors pathogen biodiversity and disease trends across natural and human systems, rather than treating each outbreak in isolation (Altizer et al., 2013).

Conclusion

Climate change and disease emergence are bound together, but not in the simple way that early rhetoric implied. Warming, shifting rainfall, extreme weather, and rising seas act on every link of the transmission chain, on the vectors that carry pathogens, on the pathogens themselves, and on the hosts and wildlife reservoirs that sustain them. The weight of evidence shows that these forces are already reshaping where and when many infections occur, and that they are opening new frontiers for spillover from animals to people. At the same time, the science has matured into something more careful than alarm. Transmission responds to temperature in a hump-shaped way, so that the same warming can raise risk at the cool margins of a disease and lower it where conditions are already hot. The climatic signal is filtered through poverty, mobility, urbanisation, and the strength of control programmes, which can dominate the outcome and have, in the case of malaria, driven decline even as the planet warmed.

Three conclusions stand out. The clearest effects of climate change appear at the edges of a pathogen's range, where it expands into newly suitable territory or retreats from territory grown too hostile. The dominant near-term consequence is therefore redistribution, the movement of risk poleward and upward into populations and health systems that are often unprepared, rather than a uniform global rise. And because climatic hazards aggravate disease through so many independent pathways, adaptation alone cannot keep pace; the breadth of the threat is itself an argument for cutting greenhouse-gas emissions at the source. The most effective response couples proactive, integrated surveillance and resilient health systems with that mitigation. The task ahead is to predict, to prepare, and to act before the map is redrawn, rather than after.

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