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Research Article

CLIMATE CHANGE, ZOONOTIC SPILLOVER AND THE RISING THREAT OF EMERGING INFECTIOUS DISEASES

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ABSTRACT

The landscape of global public health is being fundamentally reshaped by an accelerating convergence of environmental and anthropogenic factors, leading to an increased frequency and geographic expansion of Emerging Infectious Diseases (EIDs). The majority of these EIDs are zoonotic in origin, underscoring the critical role of spillover events from animal reservoirs to human populations. Climate change supports spillover through a variety of complex and interrelated ways. Primarily, animal reservoirs' behavior and spatial distribution are directly impacted by climate change. New infections are introduced to immunologically naïve human populations as a result of rising temperatures and changing precipitation patterns that extend acceptable habitats for these organisms into new areas. Human-wildlife encounter is more likely as a result of this regional blurring of former biological boundaries. Additionally, as a result of habitat loss and fragmentation brought on by climate change, wildlife are forced to move closer to agricultural regions and human settlements in search of resources, which creates new interfaces for cross-species transmission. Spillover risk is acutely increased by extreme weather events, which are a sign of a warming globe. The climate-driven ecological disruptions are compounded by globalized travel and trade, which can rapidly transport a locally spilled-over pathogen across the world. This complex interplay creates a syndemic of environmental and health crises, challenging existing surveillance and response systems. Thus, the paper is an attempt to explore the evidence demonstrating that anthropogenic climate change acts as a potent catalyst and multiplier of zoonotic spillover risk, thereby elevating the global threat of epidemics and pandemics. In short, preserving ecosystem integrity and stabilizing the climate is not merely an environmental goal but a fundamental cornerstone of pandemic prevention in the 21st century.

Keywords: Climate Change, Zoonotic Spillover, Emerging Infectious Diseases, One Health.

INTRODUCTION

Climate change has emerged as one of the most crucial threats to global public health, with profound implications for the emergence and spread of infectious diseases (Liao *et al.,* 2024). Given that significant and long-lasting changes in temperature, precipitation, humidity and air quality are already having a catastrophic impact on human health globally, the World Health Organization has categorically recognized climate change as an immediate existential danger (Parums, 2024; Bhatnagar, 2026). According to Mora *et al.* (2022), climatic hazards may exacerbate 218 (58%) of the 375 infectious diseases that affect humans, underscoring the pervasive influence of environmental change on disease dynamics. The relationship between climate change and infectious disease emergence is multifaceted and complex. Rising global temperatures, altered precipitation patterns, extreme weather events and ecosystem disruption are creating conditions that favour the transmission of pathogens from animals to humans *via* zoonotic spillover (Gass, 2025; Mahanta and Sulabh, 2025). Majority of emerging infectious diseases are zoonotic in origin and climate change is now recognized as a super-driver of this phenomenon (Hu and Gan, 2026). In comparison to the pre-industrial era, the Earth's mean surface temperature has risen by 1.09°C and by 2100, climatologists predict an unprecedented rise of 2.0°C (Kirchengast and Pichler, 2025). These pose a serious risk to ecosystem stability, animal welfare and human health in addition to being environmental issues. Thus, the paper is an attempt to explore the evidence demonstrating that

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anthropogenic climate change acts as a potent catalyst and multiplier of zoonotic spillover risk, thereby elevating the global threat of epidemics and pandemics

The zoonotic spillover

When a pathogen successfully transfers from an animal host to humans and infects a new host species, this is known as zoonotic spillover. The process is helped by an array of complex interactions between infections, animal hosts, vectors and human populations, with environmental conditions playing a critical mediating role (Ellwanger and Chies, 2021; Ahmed, 2025). Zoonotic spillover episodes with catastrophic outcomes can be seen in recent years. When individuals eat date-palm sap tainted with bat excrement, the Nipah virus, which has caused nearly yearly epidemics in Bangladesh and India with fatality rates surpassing 75%, appears (Khan *et al.,* 2025). Outbreaks of the Ebola virus have been connected to spillover events from wildlife reservoirs, especially bats and primates, which are frequently linked to habitat destruction and increased contact between humans and wildlife (Alexander *et al.,* 2015). The COVID-19 pandemic represents the most consequential zoonotic spillover event in modern history, with scientific evidence strongly supporting its origin through zoonotic transmission, likely from bats (Ruiz-Aravena *et al.,* 2021; Sparrer *et al.,* 2023).

Zoonotic spillover is facilitated by the infections' ability to adapt to new host habitats, their virulence traits and their cellular and tissue tropism. Furthermore, by changing the frequency and character of human-animal interactions, anthropogenic causes including habitat degradation, wildlife trade, agricultural intensification and climate change greatly raise the probability of spillover events (García-Moreno, 2023). It should be noted that not all diseases or environments have the same spillover dynamics. The density and variety of reservoir hosts, the frequency of human-animal contact, the incidence of infection in animal populations and environmental factors that affect pathogen survival and transmission all affect spillover risk. Figure 1 depicts major pathways from climate change to zoonotic spillover.

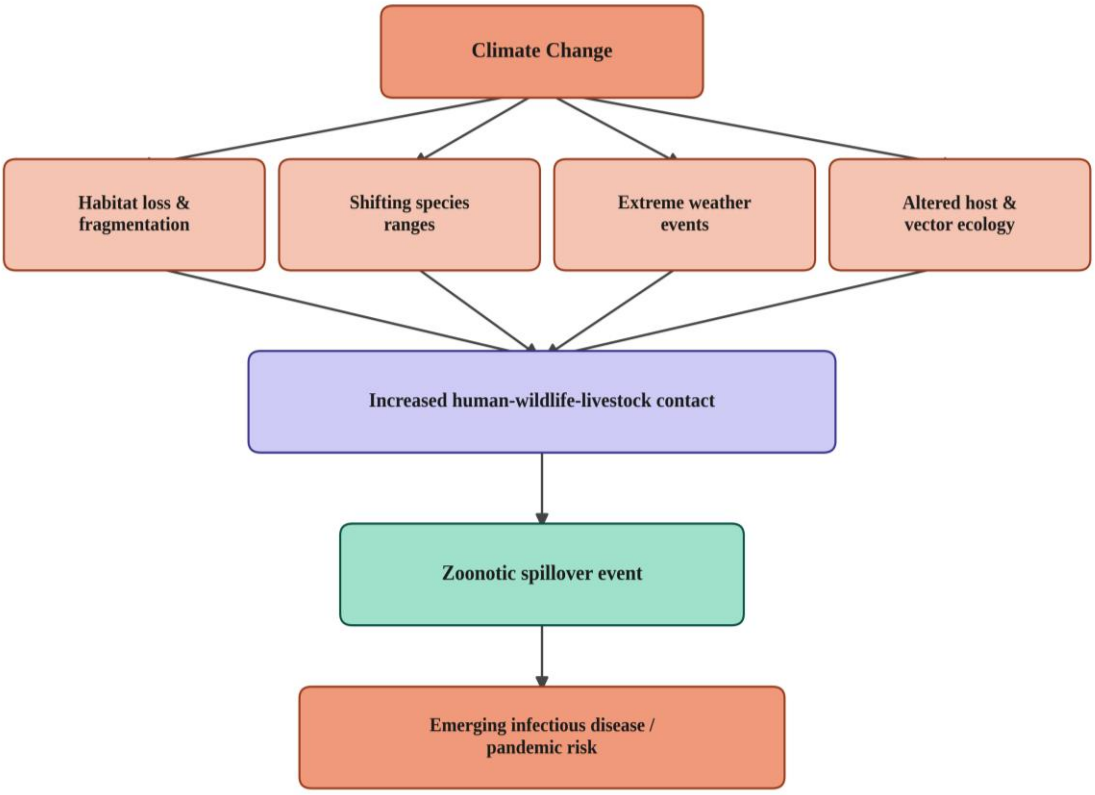


Figure 1. Pathway from climate change to zoonotic spillover.

Climate change as a super-driver of disease emergence

Climate change is increasingly recognised as a 'super-driver' of disease emergence, accelerating the appearance, spread and severity of infectious diseases by reshaping environmental conditions, ecological interactions and human vulnerability (Paul *et al.,* 2025; PREZODE, 2025). The survival, reproduction and dispersion of infections, vectors and animal reservoirs are altered by rising temperatures, changing rainfall patterns and an increase in the frequency of extreme weather events. This increases the geographic and temporal windows for transmission. In this sense, climate change actively modifies the biological and social environments in which diseases arise and spread rather than just acting as a background stressor (Liu *et al.,* 2025; Borhani *et al.,* 2026). One key pathway is through

vector-borne diseases. The distribution of dengue, chikungunya, malaria, Lyme disease and other illnesses are expanded by warmer weather, which allow mosquitoes like Aedes and Anopheles and ticks like Ixodes to live and procreate in areas and at elevations where they were previously absent or uncommon. Even in temperate regions, higher temperatures increase the danger of outbreaks by shortening pathogen incubation times inside vectors and extending transmission seasons. Concurrently, shifting precipitation intensifies transmission hotspots by concentrating vectors around diminishing water sources during droughts and creating new breeding grounds during floods (Ali *et al.,* 2025; Apetrei-Pandrea and Dascalu, 2026). The rise of food-borne and water-borne illnesses is also fuelled by climate change. Particularly in low- and middle-income nations with inadequate health systems, excessive rains and flooding overwhelm sanitation facilities, contaminate drinking water, and cause outbreaks of cholera, typhoid, and diarrheal illnesses. On the other hand, droughts cause starvation, limit access to clean water, and impair hygiene, all of which increase vulnerability to illnesses like tuberculosis. Additionally, heat waves and changes in humidity can have an impact on food safety by encouraging the growth of germs and the creation of toxins in foods that are inadequately cooled or kept (Zhang and Liu, 2025; Chidubem, 2025). In addition, the 'dilution effect,' which buffers transmission through varied host populations, is weakened by biodiversity loss; as ecosystems become simpler, competent reservoir hosts frequently take over, increasing the probability of spillover (Keesing and Ostfeld, 2021; Chen and McFarlane, 2025). The introduction or reemergence of illnesses like Nipah, Ebola and other hantaviruses, where land-use change and climate variability cross, are clear examples of these dynamics.

Climate change also exacerbates social determinants of disease in addition to its direct biological impacts (Ragavan *et al.,* 2020; Allen *et al.,* 2025). When people are displaced by floods, cyclones or slow-onset degradation, health services are disrupted, people are forced into informal settlements, their livelihoods and nutrition are compromised and epidemics are encouraged. A poverty-disease trap exists in low- and middle-income countries and other vulnerable areas, where socioeconomic deprivation and climate-sensitive illnesses compound one another (Barbier, 2025). Climate change is a threat multiplier in these situations, exacerbating already-existing disparities in exposure, susceptibility and care access. The idea that climate change is a super-driver also describes how it helps diseases get past biological boundaries. For instance, mammalian thermal defenses that have traditionally prevented systemic fungal infections may be compromised by the ability of some fungal pathogens to adapt to higher temperatures due to warming (George *et al.,* 2025; Jiang *et al.,* 2026). Similarly, changing climate regimes can select for more heat-tolerant or desiccation-resistant strains of bacteria and viruses, subtly shifting pathogen traits in ways that favour persistence and transmission under new conditions.

Ecosystem disruption and the human-wildlife interface

One of the major anthropogenic factors contributing to the rise of zoonotic diseases is deforestation and habitat fragmentation. Deforestation in tropical countries was substantially associated with increases in zoonotic and vector-borne disease epidemics between 1990 and 2016 (Morand and Lajaunie, 2021). There are several interrelated pathways that link the emergence of diseases to forest loss. The edge effects of forest fragmentation raise the possibility of infection transmission between humans, domestic animals, and wildlife as well as the movement of organisms across ecotones and interaction with new species (Hending *et al.,* 2024). In South America and Southeast Asia, where forest removal encourages malaria mosquito vectors, deforestation has been directly linked to malaria outbreaks. In a similar vein, the resurgence of leishmaniasis has been connected to deforestation because the loss of biological management of reservoir hosts results from forest fragmentation and biodiversity loss.

Wildlife is forced to seek food and shelter in human-dominated landscapes due to habitat degradation, which increases the likelihood of pathogen spillover (Jackson *et al.,* 2023). After habitat loss from drought, wildfires or deforestation, wildlife has been linked to the spread of viruses like Nipah and Ebola into human regions. For instance, several human viruses, such as the Australian bat lyssavirus, Hendra virus, Nipah virus and rabies virus, are found in bat species that are endangered by habitat degradation. Grillet and Vincenti-González (2024) noted that in severely fragmented forests, deforestation promotes the introduction and reemergence of rabies, yellow fever and hantavirus viruses. The significance of intact ecosystems in lowering viral zoonotic risk is shown by the protective function of host and vector diversity in less degraded forests (Martin and Watson, 2016).

Urbanization: Concentrating risk in dense populations

Infectious diseases can spread more quickly and impact a greater number of people in shorter amounts of time following spillover occurrences because urbanization concentrates people, activities and dangers in densely populated areas. High population density shortens the transmission chains for respiratory infections by increasing the frequency and intensity of human encounters. Infectious droplets and aerosols can build up in crowded, poorly ventilated interior places, increasing the likelihood that a single case will result in numerous secondary infections which was particularly evident during the COVID-19 pandemic (Azuma *et al.,* 2020). This concentration of risk is compounded by the spatial organisation of cities. Dense urban cores and peri-urban fringes often exhibit heterogeneous patterns of infrastructure, governance and socio-economic vulnerability, producing hotspots where transmission is amplified.

Urban density also interacts with mobility and connectivity to magnify risk. Cities function as hubs in regional and global transport networks, where spillover pathogens introduced into dense populations can disseminate quickly along commuting routes, intercity corridors and air travel links. Larger, denser urban regions see earlier, sharper peaks for acute respiratory infections than smaller towns and rural areas, according to modelling studies that show how city size and structure impact epidemic curves. Because of this amplifier effect, when seeded in large metropolitan centers, even slight increases in transmission potential can result in significant absolute case numbers (Wang *et al.,* 2025; Kimball *et al.,* 2025). Additionally, urban growth creates new interfaces for the spread of zoonotic diseases by bringing human settlements closer to peri-urban wildlife. The effects of urbanization are especially worse in developing countries, where the fast expansion of informal settlements results in inadequate access to healthcare, clean water and sanitary facilities. These settings turn into hotspots for respiratory ailments, vector-borne infections and diarrheal disorders, which also contributes to the global spread of diseases.

Zoonotic spillover and rising threat of emerging infectious diseases

Zoonotic spillover is widely acknowledged as the primary source of newly emerging infectious diseases with wildlife or domestic animal reservoirs accounting for around 75% of recent emergences. Events like HIV, Ebola, SARS, MERS, Nipah, avian influenza and SARS-CoV-2 show how viruses that circulate silently in animal hosts can cross species boundaries and if they adapt to human transmission, cause local outbreaks or worldwide pandemics (Banisetti, 2025; Verma *et al.,* 2025). The increasing frequency and intensity of these occurrences are indicative of systematic shifts in human interactions with animals and ecosystems under growing anthropogenic pressure rather than haphazard biological mishaps. Bats, rodents and monkeys are examples of reservoir hosts that are forced into greater and more regular contact with human populations as a result of deforestation, habitat fragmentation, agricultural development and urban encroachment. For instance, increased urban land use along forest frontiers and forest fragmentation in the Amazon have increased the danger of yellow fever viral spillover, and similar processes are responsible for the rise of Nipah in South and Southeast Asia, where bat habitats are intersected by pig farms and orchards (Chen, 2025; Fell *et al.,* 2026). By concentrating a large number of genetically similar animals in high-density environments that promote pathogen amplification and development, agricultural intensification and livestock expansion further increase the danger of spillover. In particular, the production of industrial pigs and poultry can serve as mixing vessels for influenza viruses, allowing strains from domestic animals, wild birds, and humans to reassort before spreading to farm workers and nearby towns. However, smallholder systems that incorporate homesteads, livestock, and crops in quickly shifting landscapes can provide numerous, recurring human–animal interfaces where low-level spillover events build up over time (Verma *et al.,* 2025; Hamilton-West *et al.,* 2026).

Another crucial route is the trading and consumption of wildlife. People are exposed to blood, tissues and excrement that are high in infectious agents due to bushmeat hunting, wet markets, exotic pet industries and wildlife-based tourism, which increase the direct handling, slaughter and ingestion of wild animals (Mpakosi *et al.,* 2026). These activities are particularly dangerous when they include high-risk species like rats, bats and non-human primates, which have a disproportionate amount of zoonotic viruses. Trade and travel globalization then makes it possible for locally limited spillovers to spread quickly across national boundaries, turning local occurrences into global crises. By changing seasonal transmission windows, rearranging host and vector distributions and stressing ecosystems in ways that modify host behavior and immunity, climate change interacts with these causes. As temperatures rise, vectors like ticks and mosquitoes can spread to higher latitudes and elevations, bringing diseases like dengue, chikungunya, Lyme disease and different arboviruses to new human populations (Areda and Edao, 2025). Additionally, wildlife movement, breeding cycles and stress physiology are all impacted by climate variability, which may increase viral shedding and contact rates at human–wildlife interactions. In this way, rather of functioning independently, climate change multiplies the threat by intensifying already-existing spillover pathways (Hu and Gan, 2026). Biodiversity loss further complicates the picture. The need for preventive, upstream techniques rather than reactive outbreak control alone is highlighted by the growing threat of new infectious illnesses originating from zoonotic spillover. Given that the causes of spillover are mostly ecological and socioeconomic, it is crucial to incorporate preventive strategies into One Health frameworks that integrate human, animal and environmental health. The world will continue to be susceptible to an accelerating series of zoonotic emergences with potentially ever-higher health, economic and social costs if the structural issues that force humans and animals into hazardous proximity are not addressed.

CONCLUSION

Rapid urbanization, environmental disturbance, zoonotic spillover and climate change are all aspects of a single global catastrophe rather than distinct public health issues. The environmental conditions that control pathogens, vectors and animal reservoirs are changing due to anthropogenic climate change, while deforestation, habitat fragmentation, agricultural expansion, wildlife trade and urban growth are increasing the frequency of interactions between domestic animals, wildlife and human populations. The likelihood that diseases will overcome species barriers, adapt to new hosts and establish long-term transmission in human societies is increased by these processes taken together. The potentially catastrophic effects of these ecological and social changes are demonstrated by the recent rise in emerging diseases. As it simultaneously

intensifies multiple known mechanisms of disease genesis, climate change acts as a super-driver. In addition to changing pathogen development, extending transmission seasons and exposing immunologically naïve populations to diseases in newly favourable conditions, rising temperatures also increase the geographic range of vectors. Ecosystems are disrupted, wildlife is displaced and water, sanitation and healthcare systems are harmed by extreme weather events. These disruptions worsen poverty, displacement, starvation and unequal access to healthcare while simultaneously increasing human-animal interactions. As a result, communities that have made the least contribution to global greenhouse gas emissions and have the fewest means for adaptation are likely to bear the brunt of developing infectious diseases.

Intact forests, wetlands and other diverse ecosystems provide ecological barriers that regulate host populations, preserve biodiversity and reduce unnecessary contact between wildlife and people. Conversely, habitat destruction and simplification can favour competent reservoir hosts and increase pathogen transmission at forest edges, farms and expanding settlements. Protecting biodiversity is consequently not merely a conservation objective; it is an investment in public health and pandemic prevention. Sustainable land-use planning, responsible agricultural development, protection of wildlife habitats and strict regulation of high-risk wildlife trade should be treated as core health interventions. Urbanization further magnifies the consequences of spillover by concentrating susceptible populations in crowded and highly connected environments. Once a pathogen enters a densely populated city, inadequate housing, poor ventilation, insufficient sanitation and intense mobility can accelerate transmission and enable rapid national or international dissemination.

A meaningful response must move beyond emergency reaction towards prevention at the human–animal–environment interface. One Health provides the most appropriate framework because it connects public health, veterinary science, ecology, climate science, agriculture and social development. Ultimately, preventing emerging infectious diseases depends on changing the conditions that produce them. Stabilizing the climate, conserving ecosystems, improving urban living conditions and promoting responsible relationships with animals are mutually reinforcing goals. Pandemic preparedness remains necessary, but preparedness alone cannot substitute for prevention. Protecting planetary health is therefore inseparable from protecting human health and the preservation of ecological balance must become a central foundation of public-health policy in the twenty-first century.

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CONFLICT OF INTERESTS

The authors declare no conflict of interest

ETHICS APPROVAL

Not applicable

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AI TOOL DECLARATION

The authors declare that no AI and related tools are used to write the scientific content of this manuscript.

DATA AVAILABILITY

Data will be available on request

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