

At the Frontline of Global Health Security: Rethinking Zoonotic and Vector-Borne Diseases at the Climate-Biodiversity-Health Nexus

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ARTICLE INFO

Article history:

Received 21 Jul 2026

Accepted 02 Sep 2026

Published 15 Sep 2026

DOI: <https://doi.org/10.35192/jjoas-n.v20i1.431>

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ABSTRACT

Background: Climate change, biodiversity loss, land-use transformation, urbanization, and human mobility are reshaping the epidemiology of infectious diseases. These interacting forces alter the abundance, distribution, and behaviour of vectors and animal reservoirs, modify pathogen development and transmission, and increase opportunities for spillover into human populations. As a result, climate- and ecology-sensitive infectious diseases are emerging in new locations, extending their transmission seasons, and affecting populations and health systems that may have limited prior experience or preparedness.

Main Findings: The resulting risks are neither uniform nor linear. Temperature, rainfall, humidity, ecosystem disruption, socioeconomic conditions, and population movement act together to create context-specific patterns of disease emergence and spread. In some settings, warming may increase transmission suitability, while in others it may shift risk geographically or favour different pathogens and vectors. These dynamics underscore the limitations of isolated, pathogen-specific approaches to preparedness.

Recommendations: Effective prevention and control therefore require an integrated One Health framework that connects human, animal, and environmental health systems. Climate-informed surveillance, ecological and vector monitoring, interoperable laboratory networks, early-warning systems, clinical readiness, and coordinated multisectoral response should be embedded within routine public health infrastructure. Cross-border data sharing, standardized case definitions, and targeted capacity building are also essential, particularly in regions where changing transmission risks intersect with limited surveillance and response capacity.

Conclusion: Moving from reactive outbreak control to anticipatory health security will enable earlier detection, more timely prevention, and more resilient responses to emerging and re-emerging infectious disease threats. This transformation can also support equitable allocation of preparedness resources according to evolving local vulnerabilities.

Keywords: Zoonoses; Climate-Sensitive; Biodiversity; One Health; Health Security.

Background

This perspective considers the recent hantavirus events as a timely example of the broader challenges associated with relying primarily on reactive, pathogen-specific approaches to emerging infectious disease preparedness. These events, alongside wider patterns observed across zoonotic and vector-borne diseases, highlight the importance of understanding the interconnected ecological, climatic, environmental, and mobility-related factors that can influence pathogen emergence, spillover, and transmission. Within this context, the paper examines the ecological and climatic determinants relevant to hantavirus transmission, explores comparable and overlapping dynamics across zoonotic and vector-borne diseases, and discusses the implications for advancing anticipatory, integrated One Health surveillance and preparedness for emerging infectious disease threats.

The Hantavirus events and what they reveal

The M/V Hondius cruise ship-associated cluster of Andes virus (ANDV), comprising 13 cases, including three deaths (1), has drawn renewed international attention to a group of emerging zoonotic pathogens that impose substantial global morbidity and mortality yet remain without widely available specific therapies or preventive vaccines. ANDV, a member of the genus *Orthohantavirus* within the family *Hantaviridae*, is one of the principal hantaviruses associated with hantavirus cardiopulmonary syndrome (HCPS) in South America.

Transmission to humans for the majority of *orthohantaviruses* occurs through inhalation of aerosolised excreta, urine, faeces, or saliva, from infected rodent reservoir hosts, with direct contact or rodent bites representing uncommon alternative routes (2). ANDV is distinct within the genus *Orthohantavirus* for its docu-

mented ability to sustain person-to-person transmission. Established risk factors for secondary human infection include close intimate contact, sleeping in the same room, and exposure during shared social gatherings with a symptomatic individual in the febrile prodrome (2–4).

Hantaviruses cause two principal human clinical syndromes, distinguished primarily by the vascular bed most affected. The first clinical syndrome is haemorrhagic fever with renal syndrome (HFRS) which occurs predominantly in Europe and Asia and is caused by Hantaan virus (HTNV), Dobrava virus (DOBV), Puumala virus (PUUV), and Seoul virus (SEOV) (2). Endothelial infection in the renal medullary capillaries leads to increased vascular permeability, coagulation dysregulation, and acute kidney injury. The clinical course classically progresses through febrile, hypotensive, oliguric, diuretic, and convalescent phases; with case-fatality rates ranging from less than 0.1% for PUUV up to 12.0% for DOBV (2). The second major clinical syndrome is HCPS, caused by several hantaviruses, including ANDV, and characterized primarily by pulmonary and cardiovascular endothelial involvement. After a febrile prodrome of 2–7 days; characterised by myalgia, headache, abdominal pain, and gastrointestinal symptoms; patients enter the cardiopulmonary phase, in which non-cardiogenic pulmonary oedema, respiratory failure, and cardiogenic shock may develop within hours (2). Most deaths occur within the first 24 hours of hospital admission in the cardiopulmonary phase. Case-fatality rates are substantially higher than for HFRS; 21.4–35.9% for ANDV, and approximately 35% for Sin Nombre virus (SNV) (2). See Figure 1.

For ANDV, the incubation period is usually estimated to range from 1 to 8 weeks. Unlike other *orthohantaviruses*, ANDV has a well-documented capacity for interhuman transmission, usu-

ally after close and prolonged exposure to symptomatic individuals, a feature that distinguishes it within the genus and has important implications for public health risk assessment. The 2018–2019 Epuy  n outbreak in Argentina underscored the capacity of ANDV to generate clustered human infections beyond the initial zoonotic spillover event. Following a single rodent-to-human transmission event, subsequent spread was amplified by three symptomatic individuals who attended crowded social gatherings, resulting in 34 confirmed infections and 11 deaths (4).

Ecological and climatic drivers of hantavirus spillover

The spatial and temporal distribution of human hantavirus infection is determined by a convergence of ecological, climatic, and socioeconomic factors that modulate reservoir population dynamics and human-rodent contact frequency. Hantavirus spillover risk is shaped by climate-sensitive ecological processes that influence rodent reservoir populations and human exposure. Ferro et al. showed that lagged rainfall and temperature were significantly associated with HCPS incidence, supporting the use of climate-informed early warning approaches, while highlighting the need for integrated rodent surveillance to clarify reservoir-specific mechanisms (5). At a continental scale, a pan-European analysis of human hantavirus infections from 2011 to 2021 identified late-year maximum temperature as the strongest predictor of infection risk, with temperature-related bioclimatic features consistently outperforming precipitation (6). These findings suggest that, across hantavirus–host systems, climatic conditions that promote rodent population growth and overwinter survival may prolong and intensify periods of human exposure to infected reservoirs.

The mechanistic chain linking climate to human infection nevertheless remains incompletely specified, and its intermediate

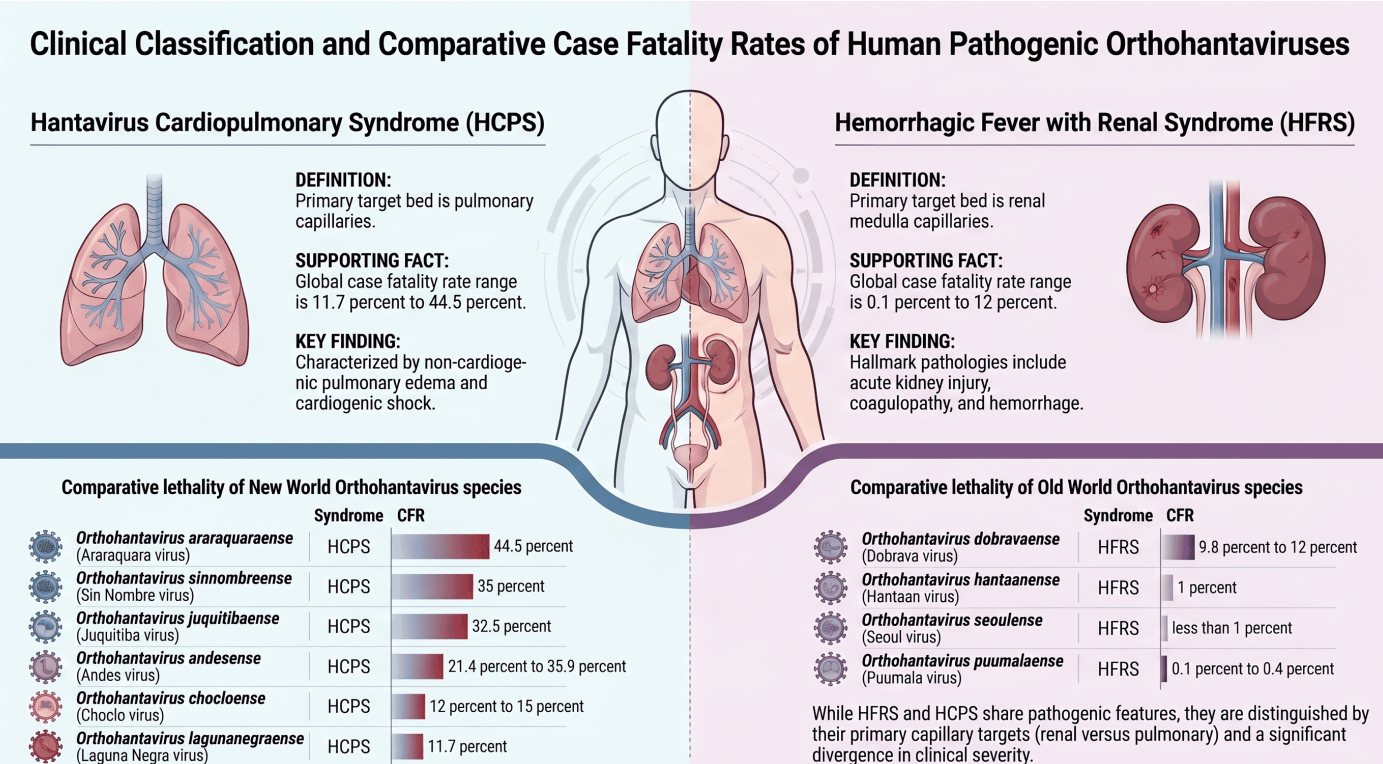


Figure 1. An overview of hantavirus clinical syndromes, the associated viral species, and case-fatality rates. Based on Vial PA, Ferr  s M, Vial C, et al. Hantavirus in humans: a review of clinical aspects and management. Lancet Infect Dis. 2023;23(9):e371-e382. doi:10.1016/S1473-3099(23)00128-7.

steps warrant explicit acknowledgement. First, reservoir abundance is an imperfect proxy for the abundance of infected hosts as rapid host population growth may initially reduce infection prevalence, with elevated prevalence emerging only later as contact rates rise, generating predicted lags between peaks in host density and peaks in prevalence (7). Empirical support for this delayed density-dependent relationship has been reported for Sin Nombre virus in deer mice, in which autumn host density predicted antibody prevalence the following spring (8), although the relationship was detectable at regional but not consistently at local scale, indicating that host abundance alone is an unreliable predictor of infection risk at the spatial resolution at which public health decisions are made (9). Comparable decoupling has been observed in European systems, where *Puumala* virus prevalence in bank voles was similar during increase and peak phases of the density cycle despite a twofold difference in host abundance (10), and the broader literature indicates that prevalence is shaped by demographic structure, host behaviour, small mammal community composition, and habitat characteristics in addition to density (11). Second, the density of infected hosts does not translate directly into human exposure, which is additionally mediated by housing construction and peridomestic food storage, occupational activity, and seasonal recreational patterns; in a 22-year United States case series, exposures were reported in the home, at work, and in recreational settings in differing proportions, and the contribution of recreational exposure differed substantially between eastern and western regions despite broadly comparable reservoir ecology (12). Third, in the specific case of ANDV, person-to-person transmission decouples a proportion of the case burden from reservoir dynamics entirely, as illustrated both by the Epuy n outbreak (4), and by the maritime cluster described earlier (1).

Biodiversity and landscape structure may act as modifiers of transmission risk, although the responsible mechanisms remain contested. The pan-European assessment found that human hantavirus infection risk varied non-linearly with habitat richness, peaking at intermediate values of the habitat richness index (HRI) (6). This pattern is consistent with a dilution effect, in which diverse host assemblages reduce the relative abundance of competent reservoirs; but it is equally consistent with an edge or intermediate-disturbance mechanism, in which fragmented landscape mosaics maximise the reservoir-human interface irrespective of host community composition; and with a primarily anthropogenic explanation, in which landscapes of intermediate heterogeneity correspond to rural-residential settlement patterns that place dwellings in close proximity to reservoir habitat.

Limitations of current preparedness systems

In the absence of an approved specific antiviral therapy, clinical management of hantavirus disease relies largely on early recognition and supportive care (2). Current preventive strategies rely primarily on reducing human-rodent contact through environmental management, rodent population control, and targeted public health education in endemic regions and high-risk occupational and recreational settings (2,6). Although inactivated Hantaan virus vaccines are licensed in the Republic of Korea and bivalent Hantaan-Seoul vaccines in China (13), no hantavirus vaccine is licensed in Europe or the Americas, and none exists for ANDV or HCPS in any setting (14).

Toward anticipatory One Health surveillance

Effective hantavirus surveillance requires integrated platforms linking ecological, climatic, and epidemiological data streams. The analytical framework demonstrated in the pan-European modelling study; combining high-resolution administrative case data with temperature, habitat richness, and socioeconomic variables; illustrates the predictive potential of multi-sector surveillance, accurately reproducing known high-risk areas and temporal epidemic patterns across the continent (6).

The translation of such frameworks to the Americas, Asia, and emerging risk zones requires investment in standardised rodent population monitoring, expanded diagnostic laboratory networks, and longitudinal integration of environmental and human health data. A One Health approach; simultaneously monitoring reservoir populations, climatic indicators, and human incidence; provides the most scientifically coherent basis for early warning systems capable of identifying periods of elevated spillover risk in advance of case clusters (6). Internationally standardised case definitions, cross-border data sharing agreements, and capacity-building for serological surveillance in regions with limited current data, including parts of the Middle East and Central Asia, where hantavirus circulation may be underdetected (15), are essential components of a globally coordinated preparedness framework.

Dynamics across ecology- and climate-sensitive infectious diseases

Ecology- and climate-sensitive infectious diseases are undergoing a broad epidemiological shift, most prominently the vector-borne diseases, which constitute a substantial and growing share of the global infectious disease burden. Climate change is already reshaping their transmission and geographical distribution, and its effects are widely projected to intensify as warming continues (16). The underlying mechanisms are principally biological. Because arthropod vectors are ectothermic, their development, survival, biting rates, and the extrinsic incubation of pathogens respond strongly and non-linearly to ambient temperature. A trait-based synthesis across eleven mosquito-borne pathogens demonstrated that transmission varies unimodally with temperature, peaking near 23–29 °C and falling to negligible levels below 9–23 °C and above 32–38 °C, so that warming amplifies transmission below the thermal optimum but suppresses it beyond (17). This non-linearity implies that a warming climate will not uniformly increase risk but will instead redistribute it, expanding suitability across cooler high-latitude and high-altitude regions while potentially rendering some already-hot areas less hospitable to transmission.

Empirical surveillance corroborates these mechanistic predictions. The Lancet Countdown on health and climate change has estimated that the climatic suitability for dengue transmission by *Aedes albopictus* and *Aedes aegypti* rose by 46.3% and 10.7%, respectively, between 1951–1960 and 2014–2023 (18). The same assessment documented increasingly favourable conditions for other climate-sensitive infections, including malaria in some settings and pathogenic *Vibrio* in coastal waters (18). However, these indices describe modelled transmission potential derived from vector and pathogen thermal biology; they quantify neither incidence nor realised transmission, and are not adjusted for population immunity, vector control, housing quality, water and sanitation infrastructure, or access to care.

The historical elimination of malaria from much of Europe and North America under conditions that remain climatically suitable illustrates how substantially realised transmission may diverge from modelled suitability. These changes are already becoming epidemiologically visible. West Nile virus risk has expanded across parts of Europe, while locally acquired dengue and chikungunya transmission has occurred in several southern European settings, demonstrating that climate-related changes in suitability are no longer purely theoretical (18, 19).

Climate-associated changes have also been observed in tick distributions, including northward expansion of *Ixodes* species in parts of North America and Europe. Temperature-driven models project further poleward and, in some settings, altitudinal expansion of suitable habitats, while milder winters and longer periods of seasonal activity may facilitate the persistence and transmission of Lyme borreliosis, tick-borne encephalitis, and other tick-borne infections (16,20–24). Similar projections have been reported for mosquito-borne diseases. Across the model and scenario combinations examined, the population living in areas climatically suitable for malaria and dengue could increase by up to 4.7 billion people by 2070 relative to 1970–1999. Climatic suitability was projected to extend toward temperate regions and higher elevations, including areas where populations may have limited immunity and public

health systems may be insufficiently prepared (25). Warming may also reconfigure the disease landscape qualitatively rather than merely enlarging it; modelling for sub-Saharan Africa indicates that continued temperature rise could shift the dominant burden from malaria toward arboviral infections in some regions as conditions exceed the thermal optimum for malaria while remaining favourable for Aedes-borne viruses (26).

Crucially, temperature operates in concert with precipitation, humidity, extreme weather events, land-use change, urbanisation, and human mobility, generating context-dependent and often time-lagged effects that complicate both prediction and control (16). These interacting determinants mirror the ecological and socio-economic drivers already described for hantaviruses, and they reinforce that no single climatic variable adequately captures transmission risk (See Figure 2). Because the largest increases in suitability frequently coincide with regions of constrained health-system capacity, these shifts also carry pronounced equity implications, concentrating emerging risk among populations least able to absorb it (25). The shared implication across pathogen systems is that resilient preparedness depends on integrated, climate-informed surveillance that couples environmental and meteorological monitoring with epidemiological data to anticipate shifting risk in space and time rather than reacting to outbreaks once established (16, 25).

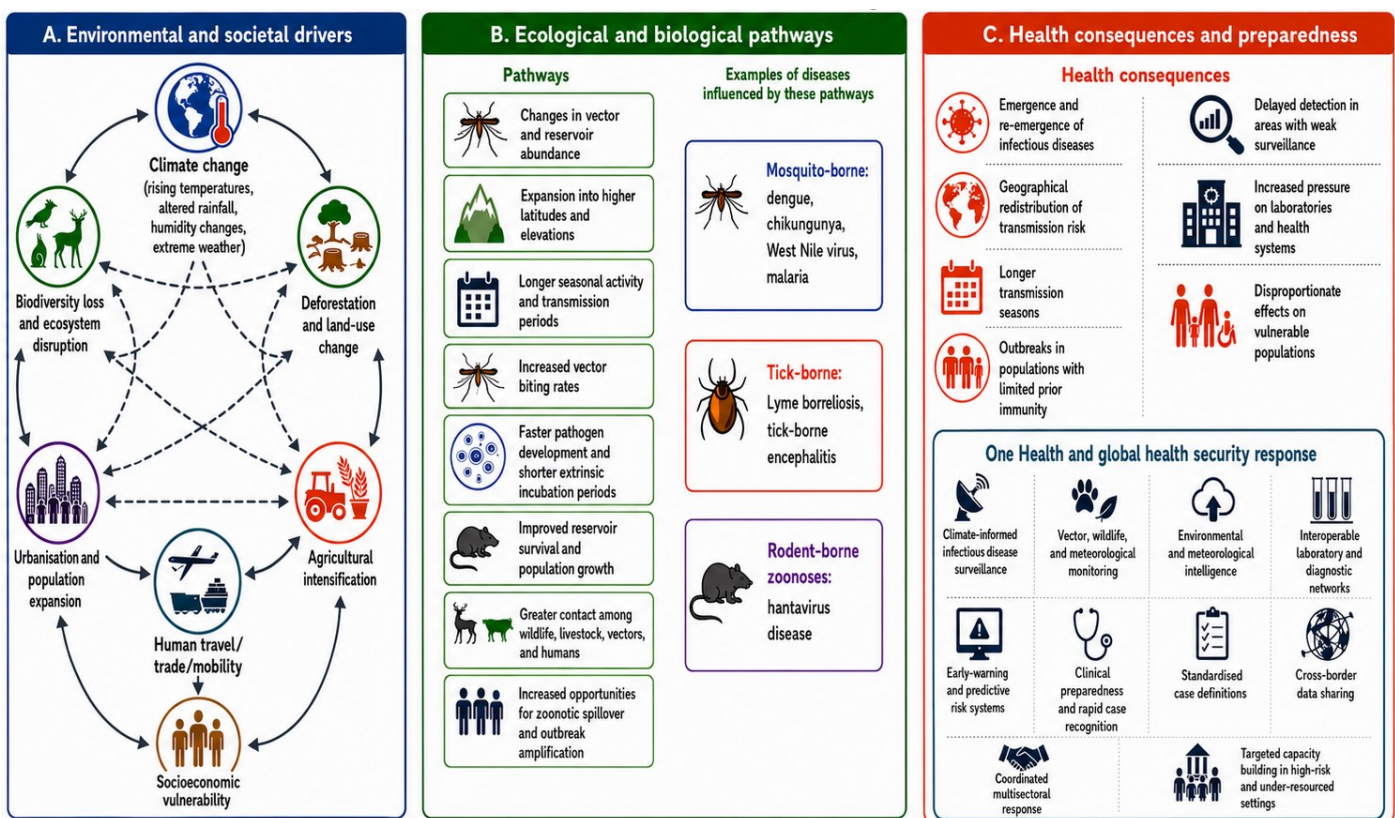


Figure 2. Conceptual pathways linking climate change, biodiversity disruption, and societal drivers to changing infectious disease risks and climate-informed One Health preparedness.

Recommendations and way forward

Moving from reactive outbreak response toward anticipatory preparedness for ecology- and climate-sensitive infectious diseases requires priorities that are operationally feasible, institutionally anchored, and adaptable to different epidemiological and resource settings. Strengthening routine detection, standardized noti-

fication, and agencies can contribute data on temperature, precipitation, drought, flooding, humidity, land cover, and other locally relevant variables. These indicators should be analysed alongside human, animal, vector, and laboratory surveillance data, with particular attention to deviations from established seasonal and historical patterns. Their value lies not in assuming that a single climatic variable can predict disease emergence, but in identifying combina-

tions of environmental and epidemiological conditions that may increase the probability of reservoir expansion, vector proliferation, pathogen spillover, or human exposure.

Population mobility should represent an additional dimension of preparedness. Ecological suitability determines where transmission opportunities may emerge, but human movement can influence where exposure occurs, where cases subsequently present, and how rapidly public health signals cross administrative or national boundaries. Migration, displacement, tourism, maritime and air travel, occupational mobility, and settlement expansion can all reshape patterns of exposure and disease detection. Integrating mobility information with epidemiological and ecological intelligence can therefore help distinguish areas where environmental conditions favour transmission from locations where populations are most likely to encounter those risks or subsequently introduce infections into other settings.

Furthermore, early warning should consequently be conceptualized as a multisource decision-support process rather than as a single predictive model. Countries should identify a manageable set of locally informative epidemiological, laboratory, ecological, climatic, environmental, and mobility indicators and determine in advance which combinations of signals warrant enhanced surveillance, field investigation, targeted testing, clinical preparedness, vector or reservoir assessment, or risk communication. Thresholds should be locally calibrated and periodically reviewed, as relationships between climate, ecology, host or vector dynamics, and human disease differ across pathogens and geographical settings.

This perspective provides a narrative rather than systematic synthesis. The infectious disease examples and geographical settings were selected to illustrate relevant mechanisms and patterns rather than through a predefined search strategy. The underlying evidence base also has important limitations, as it relies substantially on ecological-level association studies and process-based models that are sensitive to structural assumptions, parameterization, and scenario selection. These considerations do not diminish the overall consistency of the evidence, but they warrant cautious interpretation of the projections discussed here as conditional, scenario-dependent estimates rather than deterministic forecasts.

Conclusions

In conclusion, hantaviruses and other climate- and ecology-sensitive infectious diseases demonstrate that preparedness can no longer remain pathogen-specific, sectorally fragmented, or dependent on detecting outbreaks only after human cases emerge. Effective health security requires One Health systems capable of identifying ecological change before it develops into a clinical and public health crisis. This entails connecting wildlife, reservoir, and vector surveillance with climate and land-use intelligence; strengthening interoperable laboratory and epidemiological networks; maintaining risk-based clinical readiness; and enabling rapid information sharing and coordinated action across sectors and borders. These capacities should be embedded within routine public health infrastructure, with particular attention to populations and regions where shifting transmission suitability is outpacing diagnostic, surveillance, and response capacity. The essential policy shift is therefore from reacting to spillover to anticipating where and when transmission conditions are emerging—and translating that intelligence into timely

prevention, detection, and coordinated response.

Author Contributions

ABA was responsible for conceptualization, data curation, visualization, supervision, and writing—original draft preparation, review, and editing.

Declaration of Generative AI and AI-Assisted Technologies in the Manuscript Preparation Process

During the preparation of this work, the author used ChatGPT (OpenAI) solely for language refinement and editorial assistance. The tool was not used to generate scientific content, analyze data, interpret findings, or formulate conclusions. Figures 1 and 2 were created using Gemini (Google) and ChatGPT (OpenAI), respectively, based on technical prompts and scientific content provided by the author. The author reviewed and verified all AI-assisted outputs and takes full responsibility for the accuracy, integrity, and scientific content of the manuscript.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflict of Interest

The author declares no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Ethical Approval

Not required.

Acknowledgments

None.

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