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POINT OF VIEW

Climate change and the ecology of *Mycobacterium leprae*: Emerging hypotheses and research gaps from a One Health perspective

Cambio climático y ecología de *Mycobacterium leprae*: hipótesis emergentes y vacíos de investigación desde la perspectiva de Una Salud

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Abstract

Climate change is increasingly driving ecological disruption, potentially affecting neglected tropical diseases such as leprosy. Within a One Health framework, this article examines how environmental changes influence Mycobacterium leprae survival, transmission, and clinical expression. We integrate experimental, ecological, zoonotic, and immunological findings to analyze how rising temperatures, altered precipitation, and extreme weather affect leprosy dynamics. Key factors include M. leprae survival in free-living amoebae, climate-driven shifts in animal reservoirs (such as armadillos), and environmental stressors modulating host immune responses and inflammatory reactions. Potential links to antimicrobial resistance are also cautiously discussed. Rather than establishing causal relationships, this work highlights critical knowledge gaps and outlines research priorities to enhance One Health surveillance and control strategies in a changing climate.

Resumen

El cambio climático impulsa cada vez más la alteración ecológica, lo que podría afectar a enfermedades tropicales desatendidas como la lepra. Desde el enfoque de One Health, este artículo examina cómo los cambios ambientales influyen en la supervivencia, la transmisión y la expresión clínica de Mycobacterium leprae. Integramos hallazgos experimentales, ecológicos, zoonóticos e inmunológicos para analizar cómo el aumento de las temperaturas, la alteración de los patrones de precipitación y los eventos climáticos extremos afectan la dinámica de la lepra. Los factores clave incluyen la supervivencia de M. leprae en amebas de vida libre, los cambios inducidos por el clima en la distribución de los reservorios animales (como los armadillos) y el efecto de los estresores ambientales en la modulación de las respuestas inmunitarias y de las reacciones inflamatorias del huésped. También se discuten con cautela los posibles vínculos con la resistencia a los antimicrobianos. Más que establecer relaciones causales, este trabajo resalta vacíos críticos de conocimiento y plantea prioridades de investigación para fortalecer las estrategias de control y vigilancia orientadas a One Health en un clima cambiante.

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Conflicts of interest

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Declaration of AI use

The authors declare the use of generative AI in the writing process. During the preparation of this manuscript, the authors used ChatGPT 3.5 solely to improve the English language, grammar, and writing style. After using this tool, the authors carefully reviewed and edited the manuscript as needed and take full responsibility for the final content of the publication.

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Introduction

The World Health Organization (WHO) recognizes climate change as one of the major global threats to human, animal, and environmental health [1]. Rising global temperatures, altered precipitation patterns, and increasing frequency of extreme weather events are known to influence the ecology of infectious agents, particularly those involving animal hosts, environmental exposure, or complex transmission pathways [2,3]. These environmental changes may also alter human-wildlife interactions by forcing multiple species into closer contact with human settlements, potentially increasing opportunities for pathogen exposure.

The potential impact of climate change on the transmission dynamics of leprosy remains poorly characterized. While leprosy transmission has traditionally been understood as primarily human-to-human, growing evidence of animal reservoirs and environmental persistence of *Mycobacterium leprae* raises questions about how climate-related ecological changes could influence pathogen circulation and infection patterns, rather than directly causing disease [4-6]. These uncertainties pose challenges to achieving the targets outlined in the WHO global strategy "Towards Zero Leprosy" 2021-2030 [7].

Given the interconnected nature of environmental, animal, and human health, a One Health framework provides a useful lens for examining these emerging questions [8]. Accordingly, this perspective article explores selected hypotheses on how climate change could indirectly influence the persistence, transmission, and clinical expression of leprosy. Rather than establishing causal relationships, the aim is to distinguish existing evidence from theoretical considerations clearly and to identify key research gaps relevant to surveillance, prevention, and control strategies in a changing climate.

Conceptual hypotheses and research questions

Given the multifactorial nature of *M. leprae* infection and the increasing ecological pressures associated with climate change, the following conceptual hypotheses and research questions are proposed. These are not intended to represent established transmission mechanisms, but rather to highlight plausible pathways and critical gaps in current knowledge:

1. Environmental persistence: Could rising global temperatures and changes in soil and water moisture favor the proliferation of free-living amoebae capable of harboring *M. leprae*, thereby enhancing environmental persistence of the pathogen and potentially increasing opportunities for human exposure, without implying direct causation of clinical disease?

2. Zoonotic interfaces: Might climate-driven habitat disruption and geographic expansion of armadillos and other wildlife increase human-animal contact, potentially amplifying opportunities for zoonotic exposure to *M. leprae* in certain ecological contexts?

3. Antimicrobial resistance: Although *M. leprae* lacks documented horizontal gene transfer, could indirect effects of environmental stress, treatment disruption, or stress-induced mutational selection under rising temperatures influence the emergence or persistence of drug-resistant strains?

4. Host immune modulation: Could prolonged exposure to extreme heat, air pollution, and other climate-related stressors contribute to systemic proinflammatory states that modulate host immunity and potentially influence the frequency or severity of type I leprosy reactions?

5. System-level complexity: Do climate-related interactions among environmental reservoirs, animal hosts, microbial adaptation, and human vulnerability increase the complexity of *M. leprae* transmission beyond traditional human-centered models, thereby challenging existing surveillance and control strategies?

Together, these hypotheses frame climate change as a potential indirect ecological modifier of pathogen persistence, exposure, infection dynamics, and host response, rather than a direct determinant of leprosy as a clinical disease. This perspective emphasizes the need for targeted empirical research grounded in epidemiological distinctions between infection and disease expression within a One Health framework.

Human-wildlife interactions and zoonotic risk of leprosy

This infection has a well-documented zoonotic component, particularly involving armadillos as natural reservoirs of *M. leprae*. Since the 1970s, armadillos have been shown to develop leprosy both naturally and experimentally, providing robust evidence for their role in non-human maintenance of the pathogen [9]. In certain regions of North America, leprosy cases show strong epidemiological and molecular links to exposure to armadillos, especially among individuals with frequent contact with these animals [10]. For example, Sharma *et al.* [11] detected *M. leprae* in 16.4% of armadillos sampled in the southeastern United States, and they demonstrated close genetic concordance between strains isolated from armadillos and humans in the same geographic area, supporting zoonotic transmission in that context.

Beyond armadillos, additional animal hosts have been implicated as potential reservoirs of *M. leprae* and *M. lepromatosis*. In the United Kingdom, strains detected in red squirrels (*Sciurus vulgaris*) are genetically related to medieval human strains, suggesting historical spillover from humans to wildlife with subsequent persistence in animal populations [12,13]. In Latin America, multiple studies have associated hunting, handling, or consumption of armadillo meat with increased markers of *M. leprae* exposure in humans, including elevated anti-PGL-I antibody levels in endemic regions of Brazil and Colombia [14,15]. Evidence of *M. leprae* infection in armadillos has also been reported in areas with lower human leprosy prevalence, underscoring the potential for animal reservoirs to persist independently of current human transmission [16].

While the existence of zoonotic reservoirs is well supported, the extent to which climate change may modify human-animal interfaces relevant to leprosy remains uncertain. Environmental changes such as habitat disruption, land-use modification, and climate-driven shifts in species distribution could theoretically increase human-wildlife contact, including armadillos, rodents, and other mammals known to harbor *M. leprae* across diverse ecosystems [13,17-20]. However, direct evidence linking climate change to increased zoonotic transmission of leprosy is currently lacking. From a One Health perspective, these observations highlight the need for integrated surveillance of human cases, wildlife reservoirs, and environmental drivers to better understand how ecological change might indirectly influence leprosy epidemiology (Table 1).

Table 1. Climate change and its effects on the One Health perspective in leprosy infection

One Health variable	Climate change impact	Potential effects on leprosy transmission
Environmental [19,20]	Increased soil and surface water temperatures. Floods and extreme weather events.	Proliferation of free-living amoebae (FLA). Possible environmental reservoirs for *M. leprae*.
Zoonotic (animal) [13-17]	Habitat alteration and warming. Wildlife displacement toward human settlements.	Increased human contact with armadillos and red squirrels. Higher risk of zoonotic transmission.
Human [25-27]	Chronic heat exposure. Air pollution (e.g., particulate matter, diesel exhaust).	Systemic proinflammatory state. Increase in type I leprosy reactions.
Microbial [23,24]	Thermal stress on bacteria. Increased environmental DNA exchange potential	Potential emergence of multidrug-resistant (MDR) *M. leprae* strains.

Climate change and the potential role of free-living amoebae in the environmental persistence of *M. leprae*

Free-living amoebae (FLA), particularly species of the genera *Acanthamoeba* and *Hartmannella*, are known to phagocytose and harbor a wide range of intracellular microorganisms, acting as environmental shelters rather than transmission vectors. Experimental studies have demonstrated that *M. leprae* can survive within amoebic cysts for extended periods. Notably, Wheat *et al.* [21] showed that *M. leprae* remained viable and infectious within amoebic cysts for at least eight months, suggesting that FLA may protect the bacterium from environmental stressors such as desiccation, ultraviolet radiation, and temperature fluctuations.

Environmental detection studies further support the possibility of transient persistence of *M. leprae* outside the human host. Tió-Coma *et al.*[22] detected *M. leprae* DNA in soil samples from households of leprosy patients in Bangladesh, armadillo burrows in Suriname, and red squirrel habitats in the British Isles. While these findings do not demonstrate environmental transmission, they raise the possibility that soil and water environments potentially mediated by FLA may serve as temporary ecological niches for the bacterium.

In the context of climate change, rising temperatures and altered hydrological patterns could theoretically influence the abundance, distribution, and cyst formation of FLA in soil and surface waters [23]. Extreme weather events such as flooding may further facilitate the dispersal of these protozoa. However, there is currently no epidemiological evidence linking the environmental persistence of *M. leprae* associated with FLA to human infection. From a One Health perspective, this represents an important knowledge gap, highlighting the need for integrated environmental sampling, molecular detection, and epidemiological studies to determine whether environmental exposure plays any role in *M. leprae* transmission under changing climatic conditions.

Climate change and antimicrobial resistance in *M. leprae*

Multidrug therapy (MDT) has markedly reduced the global burden of leprosy. Nevertheless, drug-resistant *Mycobacterium leprae* strains have been documented, primarily through point mutations in the *folP1*, *rpoB*, and *gyrA* genes, conferring resistance to dapsone, rifampicin, and fluoroquinolones, respectively [24]. Although the overall prevalence of resistance remains low, these mutations may compromise treatment efficacy and underscore the importance of sustained molecular surveillance, particularly in endemic regions [25].

The potential relationship between climate change and antimicrobial resistance in leprosy remains largely hypothetical and should be interpreted with caution. *M. leprae* differs fundamentally from free-living bacteria commonly studied in climate-antimicrobial resistance research. Unlike organisms such as *Escherichia coli* or *Staphylococcus aureus*, *M. leprae* is an obligate intracellular pathogen with no documented capacity for horizontal gene transfer. Consequently, mechanisms proposed for other bacteria such as temperature-enhanced gene exchange cannot be directly extrapolated to leprosy.

Any potential influence of climate change on drug resistance in *M. leprae* is therefore likely to be indirect. It has been hypothesized that climate-related disruptions, including extreme weather events, population displacement, and strain on healthcare systems, could affect access to diagnosis and treatment services in endemic settings. In theory, interruptions in treatment delivery or reduced adherence to MDT might create conditions that favor the selection or persistence of resistance-associated mutations. However, direct evidence supporting these pathways in leprosy is currently lacking. Additionally, although climate-related environmental and social stressors have been proposed as factors that may influence healthcare-seeking behavior and disease management, their specific impact on the emergence or spread of drug-resistant *M. leprae* strains has not been demonstrated. These considerations should therefore be regarded as research hypotheses rather than established mechanisms.

From a One Health perspective, these uncertainties highlight the importance of strengthening genomic surveillance, ensuring uninterrupted access to MDT, and improving the resilience of leprosy control programs to environmental and societal disruptions. At present, there is no evidence that climate change directly promotes antimicrobial resistance in *M. leprae*. Further epidemiological, genomic, and implementation research is needed to evaluate whether indirect associations exist.

Effect of climate change on the host immune response in leprosy

Prolonged exposure to extreme heat, together with climate change-intensified environmental stressors such as wildfires, dust storms, and increased air pollution, has been associated with systemic immune activation characterized by elevated levels of proinflammatory cytokines, including IL-6, TNF-α, and IL-1 [26]. Among these factors, particulate matter derived from fossil fuel combustion, such as diesel exhaust, represents a particularly relevant immunomodulatory exposure. Experimental evidence from murine models suggests that such pollutants promote Th17 cell differentiation while suppressing regulatory T cell (Treg) function, thereby shifting immune responses toward a proinflammatory profile [27].

Whether these immunological effects influence the clinical course of leprosy remains uncertain. However, it has been hypothesized that an inflammation-prone immune milieu could contribute to the development or exacerbation of type I (reversal) reactions, which correspond to type IV hypersensitivity responses predominantly mediated by T lymphocytes [28]. These reactions are characterized by the predominance of CD4+ T cells with Th1 and Th17 effector phenotypes, leading to increased production of IFN-γ, TNF-α, and IL-17. The resulting activation of macrophages toward a classically activated (M1) phenotype enhances phagocytic activity and reactive oxygen species production, contributing to local tissue injury [29]. In parallel, cytotoxic CD8+ Tc1 cells have been implicated in damage to peripheral nerves and skin structures [30].

Although reduced Treg activity has been proposed as a potential mechanism linking environmental inflammation and reversal reactions, direct evidence demonstrating this pathway in leprosy is currently lacking.

Together, these observations suggest that climate-related environmental stressors may influence immune regulation in susceptible individuals with leprosy. However, the extent to which these exposures affect the frequency, severity, or neurological consequences of leprosy reactions remains unknown and warrants further investigation (Figure 1).

One Health-based hypotheses on the impact of climate change on *M. leprae* infection

Climate change has the potential to modify the ecological context in which *M. leprae* circulates by altering interactions among environmental, animal, and human systems. From a One Health perspective, these changes may increase contact between humans and animal reservoirs, while also enhancing exposure to environmental niches such as soil and stagnant water, where *M. leprae* DNA and viable bacilli have been detected, potentially within free-living amoebae acting as protective environmental hosts.

Within this conceptual framework, climate change could plausibly contribute to a convergence of environmental persistence, zoonotic exposure, and human infection, without implying direct causality. These processes may be further complicated by the emergence of antimicrobial resistance, a phenomenon well documented in other bacterial pathogens and hypothesized for *M. leprae* but not yet demonstrated. Climate-related environmental stressors may modulate host immune responses, potentially influencing the likelihood or severity of inflammatory reactions among infected individuals.

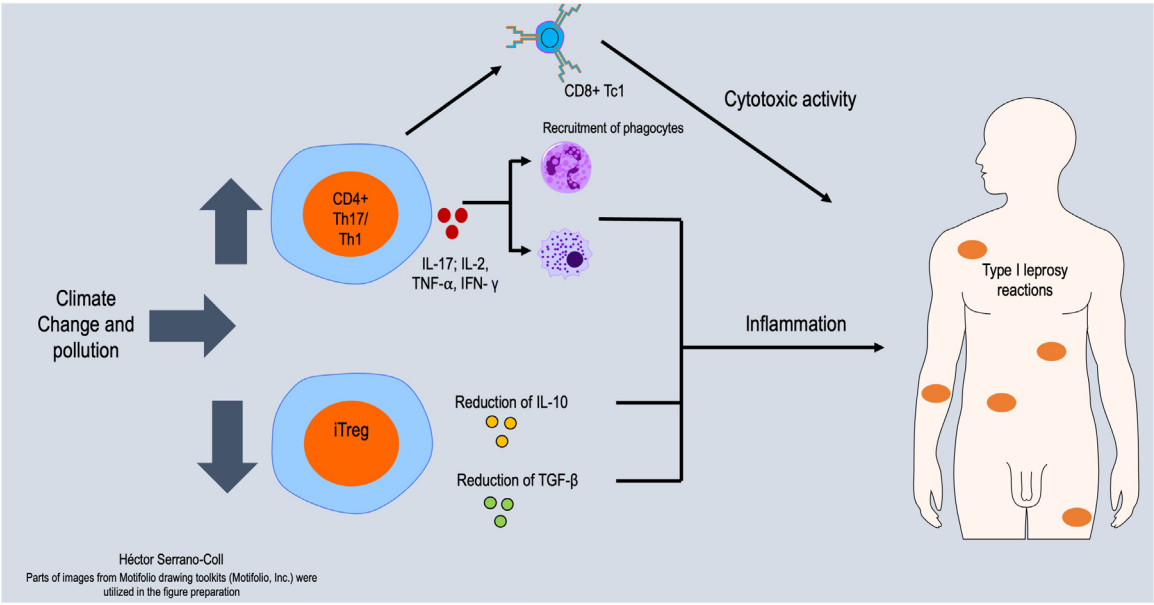


Figure 1. Impact of climate change on leprosy patients: Climate change and pollution may increase Th1 and Th17 effector responses in CD4+ T cells while reducing the immunosuppressive activity of regulatory T cells (Tregs). These immune changes could contribute to a higher frequency of type I leprosy reactions in leprosy patients.

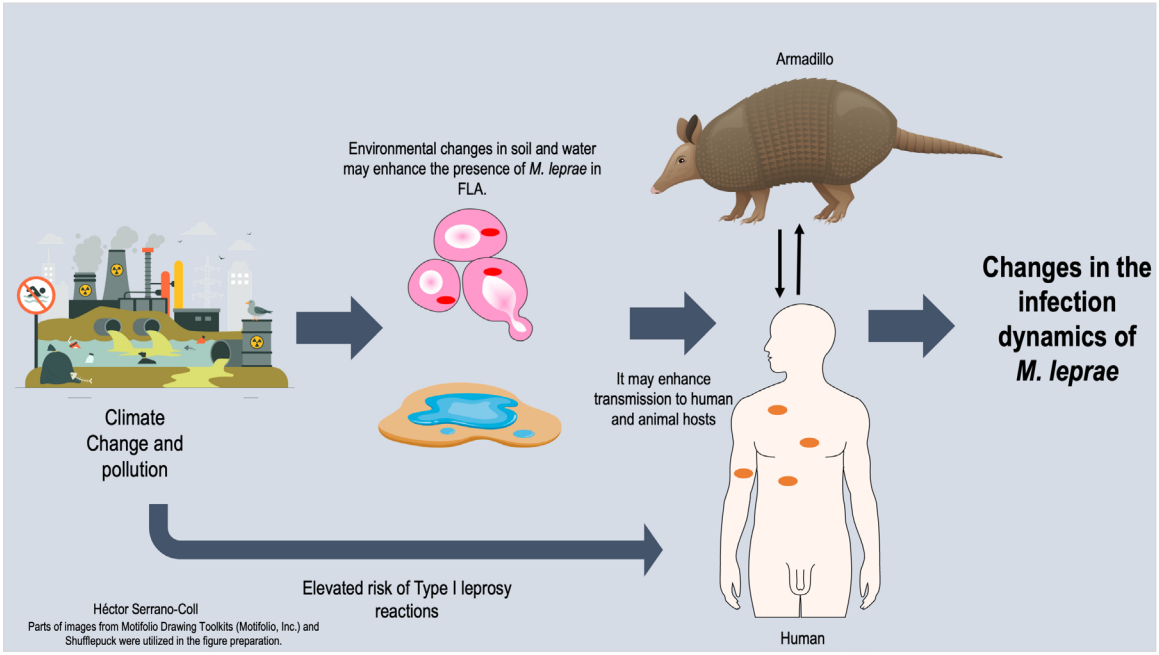
In parallel, chronic exposure to climate-related environmental stressors, including extreme heat, wildfires, and air pollution, may promote a systemic proinflammatory immune milieu. In susceptible individuals, this immune imbalance could lower the threshold for inflammatory complications such as type I leprosy reactions, potentially increasing their frequency or severity. Together, these interconnected hypotheses highlight the need for integrated surveillance and research strategies grounded in the One Health approach (Figure 2).

Operationalizing a One Health approach for sustainable leprosy control

Currently, strategies targeting household contacts of patients with leprosy primarily focus on early clinical detection and preventive interventions such as chemoprophylaxis with rifampicin and immunoprophylaxis with BCG. Although these measures have demonstrated effectiveness, they remain largely centered on a biomedical model and often overlook essential components of the One Health approach, including community empowerment for early recognition of suspicious signs, reduction of disease-related stigma, understanding of potential animal reservoirs, and improvement of the social and environmental conditions that favor the persistence of *M. leprae*.

A successful example of operationalizing the One Health approach is the SAFE strategy (Surgery, Antibiotics, Facial cleanliness, and Environmental improvement), promoted by the World Health Organization for trachoma control [31]. This model integrates clinical interventions, behavioral change, and environmental improvements to interrupt pathogen transmission. Similarly, leprosy infection control could benefit from integrated strategies that move beyond individual patient management and incorporate social, environmental, and community determinants that influence exposure and transmission dynamics.

For this reason, we propose that operationalizing a One Health framework for leprosy should be structured around four interconnected strategic lines. First, community integration and stigma reduction through training local leaders as early detection agents, strengthening health education to reduce diagnostic delays, and involving previously treated individuals as community health promoters. Second, strengthening environmental and ecological surveillance, including systematic investigation of potential animal reservoirs and environmental niches, while consistently promoting



Hypothetical effects of climate change on M. leprae infection dynamics: Climate change and pollution may lead to rising temperatures in surface water and changes in soil conditions, promoting the proliferation of free-living amoebae (FLA) capable of maintaining the viability of M. leprae. These amoebae, interacting with both animal and human hosts, could increase the risk of interspecies transmission. This ecological shift may ultimately contribute to a rise in leprosy cases due to altered infection dynamics.

animal welfare; conducting eco-epidemiological studies linking housing conditions, overcrowding, and social vulnerability with *M. leprae* transmission; and incorporating environmental sciences into national leprosy control programs. Third, implementing a truly interdisciplinary approach based on integrated teams composed of physicians, veterinarians, epidemiologists, social scientists, and anthropologists, supported by shared surveillance systems across human, animal, and environmental health sectors and the development of predictive risk models grounded in social and ecological determinants. Finally, advancing sustainable prevention beyond the individual level by complementing chemoprophylaxis and immunoprophylaxis with structural interventions that improve living conditions, access to water and sanitation, and community resilience among populations affected by neglected tropical diseases.

The persistence and reemergence of infections previously considered controlled highlight the need to move beyond fragmented approaches. Fully adopting a One Health perspective requires shifting from patient-centered interventions to coordinated strategies that recognize that *M. leprae* transmission arises from the continuous interaction among humans, animals, environmental conditions, and social determinants. Only through this integrated vision will sustainable and equitable leprosy control become achievable.

Conclusions

Leprosy control programs continue to face important challenges, particularly in the early detection of new cases, which is hampered by delayed diagnosis and the lack of widely available tools to detect infection at its earliest stages. Therefore, strengthening early diagnostic strategies and surveillance systems remains a central priority for disease control. In this context, the development of novel diagnostic strategies to detect subclinical or early disease stages is a priority, particularly in endemic regions where transmission persists despite ongoing control efforts, in light of potential environmental and immunological changes associated with climate variability.

Beyond a human-centered framework, expanding surveillance to include genomic, environmental, and zoonotic components may contribute to a more comprehensive

Table 2. Research priorities and One Health applications for investigating the impact of climate change on *Mycobacterium leprae*.

Research domain	Key hypothesis/question	Current evidence	Research priority	Potential One Health application
Environmental persistence	Could free-living amoebae and environmental niches contribute to *M. leprae* persistence?	Moderate experimental evidence	High	Environmental surveillance
Zoonotic interfaces	Does climate-driven habitat change increase human exposure to infected wildlife?	Moderate ecological evidence	High	Wildlife monitoring and reservoir studies
Host immune modulation	Do climate-related stressors influence inflammatory leprosy reactions?	Limited indirect evidence	High	Risk prediction and patient monitoring
Antimicrobial resistance	Could climate-related disruptions indirectly affect resistance emergence or persistence?	Low evidence	Moderate	Genomic surveillance
System-level complexity	How do environmental, social, and biological determinants interact to shape transmission?	Conceptual evidence	High	Integrated One Health surveillance

understanding of *M. leprae* transmission dynamics. Factors such as environmental persistence of *M. leprae*, contact with animal reservoirs, and immunological responses potentially influenced by climate-related stressors should be considered plausible contributing factors. However, direct causal links remain to be established. From a One Health perspective, integrating human, animal, and environmental data provides a useful conceptual framework for identifying knowledge gaps, guiding future research, and strengthening surveillance strategies in regions where leprosy remains endemic. To facilitate future research efforts, the main hypotheses discussed in this perspective, together with their current level of supporting evidence, epidemiological support, and research priority, are summarized in Table 2. As climate change continues to reshape ecological and social systems, such integrative approaches may support adaptive public health strategies to sustain progress in leprosy control and prevention.

Limitations

This perspective has several limitations. The hypotheses presented are primarily based on indirect evidence derived from ecological observations, experimental studies, animal models, and findings from other infectious diseases. At present, there is no direct epidemiological evidence demonstrating that climate change influences *Mycobacterium leprae* transmission, environmental persistence, or leprosy incidence. Therefore, the proposed associations should be interpreted as conceptual frameworks intended to guide future research rather than as established causal relationships.

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