

Host–Pathogen Coevolution: Emergence of Microbial Virulence in a Changing Global Environment

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Abstract

Host–pathogen interactions represent dynamic evolutionary systems in which hosts continuously develop mechanisms to resist infection while pathogens evolve strategies to colonize, evade immunity, exploit host resources, and transmit successfully. This reciprocal process, commonly described as host–pathogen coevolution, is a major driver of microbial adaptation, pathogen emergence, and variation in disease severity. Virulence is not a fixed property of a microorganism but an evolving phenotype shaped by interactions among pathogen genetics, host susceptibility, ecological conditions, transmission routes, and environmental pressures. Contemporary global environmental change is increasingly modifying these interactions. Rising temperatures, altered precipitation, extreme weather events, habitat fragmentation, biodiversity loss, urbanization, agricultural intensification, pollution, and increased movement of people and animals can change host distributions, pathogen survival, transmission opportunities, and selective pressures. Climate-related environmental changes may therefore influence not only where pathogens occur but also how they evolve. Recent research emphasizes that the effects of climate change on infectious disease are highly pathogen- and context-specific and may involve nonlinear interactions between environmental conditions, host physiology, vectors, microbiomes, and pathogen evolution. Climate extremes can additionally disrupt plant microbiomes and host immunity, increasing disease risks in agricultural ecosystems. At the molecular level, mutation, recombination, horizontal gene transfer, mobile genetic elements, immune selection, and within-host competition contribute to the emergence of novel microbial phenotypes. Antimicrobial resistance further illustrates how environmental and anthropogenic pressures can accelerate pathogen evolution. Understanding these processes requires integration of evolutionary biology, microbiology, ecology, genomics, epidemiology, and climate science.

Keywords: host–pathogen coevolution; microbial virulence; pathogen evolution; emerging infections; antimicrobial resistance; One Health; genomic surveillance.

1. Introduction

Host–pathogen interactions are among the most powerful evolutionary processes shaping biological diversity. Whenever a pathogen infects a host, both organisms experience reciprocal selective pressures. Hosts are selected to detect, restrict, eliminate, or tolerate invading microorganisms, whereas pathogens are selected to overcome host defenses, acquire nutrients, reproduce, and reach new susceptible hosts. Consequently, infection is not simply a biological interaction between two organisms but an evolutionary process occurring simultaneously at molecular, cellular, organismal, population, and ecosystem levels [1]. The traditional view of pathogenicity often treats virulence as an intrinsic characteristic of a microorganism. Contemporary evolutionary biology, however, demonstrates that virulence is highly context dependent.

The outcome of infection depends on the genotype and phenotype of both host and pathogen, the route and intensity of exposure, host immunity, coinfections, microbial community structure, environmental conditions, and opportunities for transmission. Evolutionary theory has consequently shifted from asking whether pathogens become inherently “more virulent” toward understanding how particular ecological and epidemiological conditions select for pathogen traits that maximize fitness. The evolution of virulence is therefore closely associated with trade-offs between pathogen replication, transmission, host damage, and host survival [2]. The global environment in which host–pathogen interactions occur is also changing rapidly. Climate change is modifying temperature regimes, precipitation patterns, humidity, hydrological cycles,

extreme weather events, species distributions, and ecosystem structure. These changes can alter pathogen survival outside hosts, vector abundance, host behavior, migration, nutritional status, immune function, and contact rates [3]. A recent synthesis reported empirical evidence that climatic hazards have aggravated 218 of 375 documented human infectious diseases, illustrating the breadth of possible climate–disease relationships [4]. Importantly, environmental change does not merely affect disease transmission. It can also alter the evolutionary landscape experienced by pathogens. Environmental stress may impose new selection pressures, facilitate geographic expansion into previously unsuitable habitats, modify host–pathogen contact networks, and create opportunities for adaptation. Climate-related changes have also been associated with antimicrobial resistance, although the strength and causality of these relationships differ among systems [5]. The emergence of *Candida auris*, expansion of vector-borne pathogens, changes in waterborne disease risks, and increasing concern regarding climate-associated plant diseases illustrate the complexity of this phenomenon. Human, animal, plant, and environmental health cannot therefore be considered independently. A comprehensive understanding of future infectious disease risks requires an integrated framework that considers the host, pathogen, environment, and their evolutionary interactions.

2. Conceptual Basis of Host–Pathogen Coevolution

Coevolution occurs when reciprocal evolutionary changes in interacting species influence the fitness of each other. In host–pathogen systems, the host represents a selective environment for the pathogen, while the pathogen simultaneously constitutes a selective pressure on the host. This reciprocal process can generate continuous evolutionary change resembling an arms race. Hosts possess physical barriers, innate immune mechanisms, adaptive immunity, antimicrobial molecules, microbiomes, and behavioral defenses. Pathogens counter these mechanisms through diverse adaptations, including surface modification, antigenic variation, secretion of effector proteins, immune suppression, intracellular survival, biofilm formation, toxin production, and alteration of host signaling pathways [6]. The evolutionary outcome is rarely a simple progression toward increasing host resistance or pathogen virulence. Instead, it can involve fluctuating selection, balancing selection, specialization, generalization, local adaptation, genetic diversification, and trade-offs among pathogen traits. The same pathogen genotype may produce different disease outcomes in different hosts or environments.

At the population level, coevolution can produce geographic variation in both host resistance and pathogen virulence. Local adaptation occurs when pathogen populations become particularly effective against locally common host genotypes, while hosts may subsequently experience selection favoring resistance mechanisms against prevalent pathogen variants. Such reciprocal selection can maintain genetic diversity in both populations [7]. Coevolution is particularly dynamic for microorganisms because many pathogens have short generation times and large population sizes. Viruses and bacteria can accumulate genetic variation rapidly, while fungi and other microorganisms can evolve through mutation, recombination, gene duplication, genome rearrangement, and horizontal gene transfer.

Within-host evolution can also occur during a single infection, with pathogen populations diversifying under immune, nutritional, and therapeutic selection. Recent genomic studies demonstrate that bacterial pathogens can undergo mutation, clonal succession, convergent evolution, horizontal gene transfer, and selection for immune evasion or antibiotic resistance during infection.

3. Evolutionary Origins of Microbial Virulence

Virulence refers broadly to the degree of harm caused by a pathogen to its host. It should be distinguished from pathogenicity, which describes the capacity to cause disease. Virulence is influenced by multiple microbial traits, including replication rate, tissue invasion, toxin production, immune modulation, nutrient acquisition, dissemination, and transmission [7]. One of the central concepts in virulence evolution is the transmission–virulence trade-off. Excessive pathogen-induced damage may reduce host mobility, survival, or opportunities for transmission, whereas insufficient replication may limit pathogen transmission. Consequently, selection may favor an intermediate phenotype depending on the ecology of transmission. However, there is no universal evolutionary rule that pathogens necessarily evolve toward lower virulence. Changes in transmission opportunities, host population structure, immunity, treatment, and environmental persistence can shift the optimal phenotype [8]. Virulence can emerge through several evolutionary pathways. A previously harmless microorganism may acquire pathogenic traits through horizontal gene transfer, mutation, recombination, or acquisition of plasmids and other mobile genetic elements. Alternatively, an established pathogen may adapt to a new host species. Host switching is particularly important in emerging infectious diseases because pathogen populations entering a novel host encounter unfamiliar immune systems, physiological environments, and transmission conditions [8]. Within-host evolution represents another important route. A pathogen may experience strong selection during infection because of immune responses, antimicrobial treatment, nutrient limitation, spatial compartmentalization, and competition with other microorganisms. Genetic variants that improve survival under these conditions may increase in frequency and subsequently contribute to transmission. Genomic surveillance has increasingly revealed the importance of within-host diversity in antibiotic resistance, immune escape, and adaptation to sustained human transmission.

4. Molecular Mechanisms Driving Host–Pathogen Coevolution

4.1 Mutation and Genetic Variation

Mutation generates the raw material for pathogen evolution. RNA viruses often possess particularly high rates of genetic change, although the evolutionary rate of a pathogen depends on replication biology, genome structure, generation time, population size, and selection. Bacteria and fungi can also generate substantial genetic diversity through mutation, recombination, genome rearrangement, and gene duplication [9]. Selection acts on this variation. Beneficial mutations can increase pathogen fitness, whereas deleterious variants are generally eliminated. The consequences of mutation depend strongly on environmental context.

4.2 Horizontal Gene Transfer

Horizontal gene transfer allows microorganisms to acquire genetic material from unrelated cells. Transformation, conjugation, and transduction can transfer genes associated with antibiotic resistance, metabolic capabilities, host colonization, toxin production, and environmental adaptation. Mobile genetic elements therefore provide an important mechanism through which microbial populations can rapidly acquire novel phenotypes. In agricultural and clinical environments, intense antimicrobial selection may increase the frequency of resistant variants and facilitate their persistence.

4.3 Immune Selection

Host immune responses constitute a major selective force. Antibodies, T cells, innate immune receptors, antimicrobial peptides, and other defense mechanisms can eliminate susceptible pathogen variants. Pathogens consequently evolve antigenic variation, immune suppression, intracellular persistence, altered surface structures, and other mechanisms of immune evasion. This evolutionary interaction can create a continuous cycle in which new pathogen variants are favored when they escape existing host defenses, followed by selection for new host immune responses.

4.4 Microbiome-Mediated Selection

The host microbiome represents another component of the selective environment. Commensal microorganisms compete with pathogens for nutrients and attachment sites and can produce antimicrobial metabolites or influence host immune development. Environmental disturbances that alter microbiome composition can therefore modify pathogen establishment and disease severity [10]. This principle extends beyond humans to plants and animals. Climate extremes can disrupt plant microbiomes and weaken microbiome-mediated protection, potentially increasing disease susceptibility.

5. Global Environmental Change as an Evolutionary Force

Climate change should not be viewed solely as a factor affecting disease incidence. It can modify the ecological and evolutionary conditions under which pathogens interact with hosts.

5.1 Rising Temperature

Temperature influences pathogen replication, survival, host physiology, vector competence, and immune responses. Different microorganisms have distinct thermal niches, meaning that warming can favor some pathogens while disadvantaging others [11]. For environmental microorganisms, increasing temperature may also impose selection for thermal tolerance. This has received particular attention in relation to fungal pathogens capable of infecting mammals. The emergence and geographical expansion of human-pathogenic fungi have become increasingly important areas of research under changing climatic conditions.

5.2 Altered Precipitation and Hydrological Cycles

Flooding can mobilize pathogens from soil, sewage, agricultural land, and animal reservoirs into water systems. Heavy rainfall can therefore increase exposure to waterborne pathogens. Conversely, drought can concentrate microorganisms in shrinking water bodies and alter host behavior, potentially increasing transmission [12]. Recent research emphasizes that temperature, heavy rainfall, flooding, drought, and extreme weather can influence pathogen survival, replication, exposure, and transmission through multiple interacting pathways.

5.3 Extreme Weather Events

Heatwaves, floods, cyclones, droughts, and wildfires can disrupt ecosystems and human infrastructure. Such disturbances may produce temporary concentrations of susceptible hosts, alter wildlife movements, increase human-animal contact, and create opportunities for pathogen transmission. Extreme events can also generate strong but transient selection pressures. Pathogens capable of surviving environmental stress may become disproportionately represented after disturbance, potentially altering population composition.

5.4 Habitat Fragmentation and Biodiversity Loss

Habitat destruction changes host community composition and contact patterns. The loss of ecological diversity may alter pathogen reservoirs and transmission networks. Agricultural expansion and urbanization can increase contact between humans, domestic animals, wildlife, and environmental microorganisms. The resulting ecological interfaces create opportunities for host switching and pathogen emergence.

6. Climate Change and the Evolution of Virulence

The relationship between climate change and virulence is complex. Environmental conditions can influence pathogen growth, host susceptibility, and transmission simultaneously, making it difficult to distinguish direct evolutionary effects from ecological changes. For example, warming may increase pathogen replication while simultaneously reducing host immune performance. If increased replication enhances transmission, selection may favor pathogen genotypes with greater replication capacity. However, if high virulence rapidly eliminates transmission opportunities, selection may instead favor lower levels of damage. Environmental change can also modify the spatial structure of pathogen populations. Geographic range expansion exposes pathogens to novel hosts, potentially creating new opportunities for adaptation. Newly introduced pathogens may experience strong selection as they encounter host populations with different genetic backgrounds and immune histories. Plant diseases provide a particularly important example. Climate change can modify pathogen distributions, disease incidence, host physiology, and pathogen-host interactions, thereby creating conditions favorable for emergence of new disease pressures. The relationship is therefore best conceptualized as a climate-host-pathogen evolutionary triangle, in which climate alters both host and pathogen phenotypes while simultaneously modifying their ecological interaction.

7. Emerging Pathogens and Host Switching

Host switching is one of the most important pathways to disease emergence. A microorganism that is well adapted to one host may occasionally encounter another species. Successful establishment requires overcoming physiological barriers, immune defenses, temperature constraints, tissue compatibility, and transmission limitations. Environmental change can increase such opportunities by altering species ranges and contact networks. Agricultural intensification, wildlife trade, urban expansion, deforestation, and climate-driven migration may increase interactions among previously separated populations. Following host switching, pathogens may undergo rapid adaptation. Mutations that enhance receptor binding, tissue invasion, immune evasion, environmental persistence, or transmission can be favored.

The evolutionary trajectory depends on the compatibility between pathogen traits and the new host environment. This process illustrates why emergence should not be understood as a single event. Emergence can involve a sequence of stages: ecological contact, exposure, infection, within-host adaptation, sustained transmission, population expansion, and subsequent evolutionary diversification.

8. Antimicrobial Resistance as a Product of Evolutionary Selection

Antimicrobial resistance is a prominent example of pathogen evolution under anthropogenic selection. Antibiotics, antifungals, antiparasitic drugs, and other antimicrobial agents create selective environments in which resistant variants gain a fitness advantage. Resistance can arise through spontaneous mutation or acquisition of resistance genes. Once selected, resistant microorganisms can spread within and between populations through transmission and horizontal gene transfer. Environmental change may interact with antimicrobial resistance in several ways. Heat, flooding, altered wastewater systems, ecosystem disturbance, and changes in antimicrobial use may influence the abundance, persistence, and transmission of resistant microorganisms. A 2026 review concluded that evidence increasingly links climate change and extreme weather with antimicrobial resistance, while emphasizing that causal relationships remain incompletely resolved. The global AMR problem therefore represents an evolutionary and ecological challenge rather than simply a clinical problem. Effective control requires reduction of unnecessary antimicrobial exposure, improved sanitation, environmental monitoring, responsible agricultural practices, and genomic surveillance.

9. Plant–Pathogen Coevolution under Environmental Stress

Host–pathogen coevolution is not restricted to human disease. Plant pathogens constitute an important component of global environmental change because disease affects food production, ecosystem functioning, and biodiversity. Plants defend themselves through structural barriers, pattern-recognition receptors, antimicrobial compounds, systemic signaling, and resistance genes. Pathogens counter these defenses through effector proteins, toxins, enzymes, immune suppression, and rapid genetic diversification. Climate extremes can disrupt this balance. Heat and drought can alter plant physiology and suppress certain immune processes, while simultaneously changing pathogen growth and distribution. Recent evidence indicates that extreme climatic events can increase the virulence and geographical distribution of some plant pathogens and disrupt protective plant microbiomes. The agricultural implications are substantial. Climate-associated pathogen expansion can reduce crop productivity, increase pesticide dependence, accelerate fungicide resistance, and threaten food security. Evolutionary approaches should therefore become an integral component of climate-resilient crop management.

10. Environmental Change, Fungal Pathogens and Thermal Adaptation

Fungi provide a particularly informative example of environmental selection and pathogen emergence. Most environmental fungi do not readily infect humans because mammalian body temperature represents a physiological barrier.

Adaptation to warmer environments may reduce this barrier for some fungal populations. The emergence of *Candida auris* has generated substantial interest in the possibility that environmental change can contribute to the evolution of human pathogenicity. However, causal attribution to climate change remains an active area of investigation, and multiple ecological, medical, and evolutionary factors must be considered. More broadly, climate variability can alter fungal distributions and facilitate the establishment of pathogenic species in previously unsuitable regions. Recent assessments emphasize the importance of surveillance of fungal dispersal, adaptation, population immunity, diagnostics, and antifungal stewardship under a changing climate.

11. The Role of Human Activity

Human activities are increasingly integrated into pathogen evolutionary ecology. Global transportation allows microorganisms to cross geographical barriers rapidly. Intensive livestock production can increase pathogen population density and create repeated opportunities for transmission and adaptation. Agricultural monocultures may provide extensive genetically similar host populations, facilitating pathogen spread. Urbanization creates dense networks of human hosts and extensive infrastructure for pathogen movement. Wastewater systems, healthcare facilities, food-processing environments, and animal-production systems can function as reservoirs in which microorganisms experience strong selective pressures. International travel and migration further increase connectivity among pathogen populations. Consequently, the modern pathogen is evolving not only within natural ecosystems but also within highly modified anthropogenic environments.

12. Genomic Surveillance and Predicting Pathogen Evolution

The rapid development of genomic sequencing has transformed the study of host–pathogen coevolution. Whole-genome sequencing can identify mutations, transmission chains, resistance determinants, population structure, and evidence of adaptation. Metagenomic sequencing additionally enables surveillance of microbial communities without requiring isolation of individual organisms. Longitudinal genomic datasets can reveal how pathogen populations change over time and how environmental conditions influence their evolution. Climate-informed genomic surveillance represents an important emerging approach. Environmental variables can be integrated with pathogen genomic data, epidemiological records, host distributions, and ecological information to identify conditions associated with pathogen emergence. Sequencing-based approaches are increasingly being used to monitor pathogen evolution associated with climate change, providing opportunities to identify emerging lineages before they become widely established.

14. Strategies for Managing Evolutionary Disease Risk

Traditional disease management frequently focuses on reducing pathogen abundance. Evolutionary theory suggests that interventions should also consider how management practices alter selection.

Table 1: Major drivers of host–pathogen coevolution under global environmental change

Driver	Effect on host	Effect on pathogen	Potential evolutionary consequence
Rising temperature	Altered physiology and immunity	Changes in growth and replication	Selection for thermal tolerance
Drought	Physiological stress	Environmental concentration and stress selection	Changes in transmission and persistence
Flooding	Increased exposure	Mobilization and dispersal	Geographic expansion
Habitat fragmentation	Altered host contact	New host opportunities	Host switching
Biodiversity loss	Changed community structure	Modified transmission networks	Reservoir shifts
Antimicrobial use	Selective pressure on host-associated microbiota	Selection for resistance	Resistant lineages
Agricultural intensification	Reduced host diversity	Large susceptible host populations	Rapid pathogen spread
Urbanization	High host density	Increased transmission opportunities	Adaptation to urban environments
Extreme weather	Host stress and displacement	Environmental stress	Selection for environmental tolerance
Global transportation	Increased exposure	Long-distance dispersal	Rapid geographic emergence

Evolution-informed interventions include maintaining genetic diversity in crops, rotating antimicrobial modes of action where appropriate, reducing unnecessary antimicrobial exposure, strengthening vaccination programmes, protecting ecosystem diversity, improving sanitation, and using targeted surveillance. For plant disease management, breeding for durable resistance rather than reliance on single resistance genes may reduce the probability of rapid pathogen adaptation. Similarly, microbiome-based approaches could strengthen host resilience while reducing pathogen establishment.

16. Conclusion

Host–pathogen coevolution is a continuous evolutionary process in which microbial populations and their hosts respond reciprocally to one another. Virulence emerges from this interaction rather than representing a fixed characteristic of a pathogen. Mutation, recombination, horizontal gene transfer, immune selection, within-host competition, ecological specialization, and transmission dynamics collectively shape pathogen phenotypes. Global environmental change is adding new dimensions to this evolutionary process. Rising temperatures, altered precipitation, extreme weather, habitat fragmentation, biodiversity loss, urbanization, agricultural intensification, and global connectivity can modify pathogen distributions, host susceptibility, transmission opportunities, and selective pressures. Climate change therefore has the potential to influence not only the epidemiology of infectious diseases but also the evolutionary trajectories of microbial populations. Recent research across human, animal, plant, fungal, and environmental systems demonstrates that these relationships are complex, pathogen-specific, and strongly influenced by ecological context. The central challenge is consequently to move from reactive disease control toward evolution-aware and climate-informed disease preparedness. Integrating genomics, microbiology, evolutionary biology, ecology, epidemiology, environmental science, and One Health surveillance will be essential for anticipating emerging microbial threats. Understanding how environmental change reshapes the evolutionary landscape of host–pathogen interactions may ultimately provide the scientific foundation for more durable strategies to protect human health, animal populations, agricultural productivity, and ecosystem stability.

References

1. Bote, L., & Maes, M. (2024). Tracking pathogen evolution through climate change. *Nature Reviews Microbiology*, 22, 390. <https://doi.org/10.1038/s41579-024-01057-7>

2. Charnley, G. E. C., & Kelman, I. (2024). Perspectives on climate change and infectious disease outbreaks: Is the evidence there? *npj Climate Action*, 3, 61. <https://doi.org/10.1038/s44168-024-00115-3>

3. Anikeeva, O., Hansen, A., Varghese, B., Borg, M., Zhang, Y., Xiang, J., & Bi, P. (2024). The impact of increasing temperatures due to climate change on infectious diseases. *BMJ*, 387, e079343. <https://doi.org/10.1136/bmj-2024-079343>

4. Roussin-Léveillé, C., Rossi, C. A. M., Castroverde, C. D. M., & Moffett, P. (2024). The plant disease triangle facing climate change: A molecular perspective. *Trends in Plant Science*, 29, 895–914.

5. Singh, B. K., Delgado-Baquerizo, M., Egidi, E., Guirado, E., Leach, J. E., Liu, H., & Trivedi, P. (2023). Climate change impacts on plant pathogens, food security and paths forward. *Nature Reviews Microbiology*, 21, 640–656. <https://doi.org/10.1038/s41579-023-00900-7>

6. Baldrian, P., López-Mondéjar, R., & Kohout, P. (2023). Forest microbiome and global change. *Nature Reviews Microbiology*, 21, 487–501. <https://doi.org/10.1038/s41579-023-00876-4>

7. Jansson, J. K., & Wu, R. (2023). Soil viral diversity, ecology and climate change. *Nature Reviews Microbiology*, 21, 296–311. <https://doi.org/10.1038/s41579-022-00811-z>

8. Trivedi, P., Batista, B. D., Bazany, K. E., & Singh, B. K. (2022). Plant–microbiome interactions under a changing world: Responses, consequences and perspectives. *New Phytologist*, 234, 1951–1959.

9. Raza, M. M., & Bebbler, D. P. (2022). Climate change and plant pathogens. *Current Opinion in Microbiology*, 70, 102233.

10. Velásquez, A. C., Castroverde, C. D. M., & He, S. Y. (2018). Plant–pathogen warfare under changing climate conditions. *Current Biology*, 28, R619–R634.

11. Nnadi, N. E., & Carter, D. A. (2021). Climate change and the emergence of fungal pathogens. *PLoS Pathogens*, 17, e1009503.

12. Desaint, H., Aoun, N., Deslandes, L., & Vailleau, F. (2021). Fight hard or die trying: When plants face pathogens under heat stress. *New Phytologist*, 229, 712–734.