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Opinion

Modeling plant diseases under climate change: evolutionary perspectives

Li-Na Yang,[1] Maozhi Ren,[2,*] and Jiasui Zhan[3,*]

Infectious plant diseases are a major threat to global agricultural productivity, economic development, and ecological integrity. There is widespread concern that these social and natural disasters caused by infectious plant diseases may escalate with climate change and computer modeling offers a unique opportunity to address this concern. Here, we analyze the intrinsic problems associated with current modeling strategies and highlight the need to integrate evolutionary principles into polytrophic, eco-evolutionary frameworks to improve predictions. We particularly discuss how evolutionary shifts in functional trade-offs, relative adaptability between plants and pathogens, ecosystems, and climate preferences induced by climate change may feedback to future plant disease epidemics and how technological advances can facilitate the generation and integration of this relevant knowledge for better modeling predictions.

Highlights

Modeling is a unique and cost-effective approach to predict the long-term impacts of climate change on infectious plant diseases and sustainability.

Model predictions are hindered by a lack of evolutionary understanding of the individual and interactive impacts of climate change on plants, pathogens, and ecosystems.

Climate change creates a series of intra- and inter-specific trade-offs that modulate the entire epidemiological process of infectious plant diseases.

Climatic preferences of plants, pathogens, and their interactions evolve in response to change in local climatic conditions.

Technological advances offer unprecedented opportunities to generate and integrate biological, ecological, and evolutionary knowledge in polytrophic models.

Concerns on plant disease epidemic under climate change

Plant pathogens have a significant impact on agricultural production, causing 13–22% of direct losses annually [1], in addition to indirect costs incurred through control attempts. Economic losses, together with environmental toxicity associated with some control approaches, and biodiversity degradation, greatly threaten socioeconomic and ecological sustainability [2]. The intensity of current global climate change is creating substantial alterations in both magnitude and range of air temperature, rainfall, radiation (UVR) and other climatic events. How these changes and associated secondary and even tertiary environmental and eco-evolutionary events affect epidemics of infectious plant diseases and, through this, primary food production is attracting increasing academic and public attention. As a cost-effective approach to address such complex social and natural scenarios that are not amenable to direct experimentation, computer simulation has become an irreplaceable option to assess these academic and public concerns for better predictions and mitigations [3–5].

Current prediction dilemma and challenges

In recent years, considerable effort has been made to model how infectious plant diseases in agricultural and natural ecosystems may respond to global climate change, with the aim of developing better prevention and mitigation strategies. However, predictions from these theoretical models are generally inconsistent. Some models predict climate change may increase the occurrence and severity of future infectious plant disease, with more impact on northern than lower latitude areas and on developing rather than developed countries [6–9]. Other models suggest that climate change has no major impact on the epidemic risk of infectious plant diseases or may even result in lower epidemic frequency or intensity [10,11]. In addition to the difference in the intrinsic property of particular host–pathogen interactions responding to specific forms of climate change, these inconsistent results may be generated by insufficient knowledge of relevant pathogen and host biology and their interaction with the environment, thereby preventing the development of robust models for confident inference.

[1]Fujian Key Laboratory on Conservation and Sustainable Utilization of Marine Biodiversity, Fuzhou Institute of Oceanography, Minjiang University, Fuzhou, 350108, China
[2]Institute of Urban Agriculture, Chinese Academy of Agricultural Sciences, Chengdu National Agricultural Science and Technology Center, Chengdu, China
[3]Department of Forest Mycology and Plant Pathology, Swedish University of Agricultural Sciences, Uppsala, Sweden

*Correspondence:
renmaozhi01@caas.cn (M. Ren) and
Jiasui.zhan@slu.se (J. Zhan).

Climate change not only affects functional traits of pathogens and hosts individually but also their interactions with the demography, genetics, physiology, and biology of other species in the community, creating a **trade-off** (see Glossary) landscape of pathogens and hosts, which may influence the entire epidemiological process of disease occurrence and development [12–14]. Furthermore, theoretical predictions regarding epidemics of infectious plant diseases in changing climates are hindered by a lack of evolutionary understanding of the dynamic responsiveness of host and pathogen adaptive landscapes. Current predictions are almost exclusively built on the assumptions that hosts and pathogens have fixed climatic preferences and ranges for growth, reproduction, transmission, and competition [15]. However, it has been documented that climate preferences and ranges of hosts and pathogens can evolve in response to shifts in local climatic regimes [14,16]. For example, recent works in the wheat pathogen *Zymoseptoria tritici* and potato pathogen *Phytophthora infestans* demonstrate that these pathogens adapt quite well to any changes in climate [17,18] and this adaptation is attributable to changes in both genomic structure and gene expression [19,20]. Models built upon current known climate preferences and ranges of hosts and pathogens may create severe biases in predicting the survival, reproduction, competition, and transmission of pathogens (Box 1) within and among hosts under future climatic conditions. We argue for the urgent need to incorporate these trade-off and adaptation aspects into **polytrophic**, eco-evolutionary frameworks to improve model predictions, as discussed later.

Simple mathematical models and complex reality of the disease triangle

The disease triangle concept implies that plant diseases result from complex interactions among pathogens and hosts in an ecosystem composed of all biotic and abiotic factors surrounding the host–pathogen interaction. Only susceptible plant hosts, virulent pathogens, and conducive environments coexisting in time and space can lead to disease occurrence and epidemics [21]. For example, it has been documented that community structure can prevent, enhance, or alter how hosts and their associated pathogens adapt to changing climatic conditions by affecting population sizes, genetic composition, diversity life history, spatiotemporal dynamics, and/or the fitness landscape of both hosts and pathogens [22–25]. Climatic change affects not only host **susceptibility** and pathogen **pathogenicity** but also the environment that supports the interaction, such as shifts in natural enemies, vector populations, landscape structure, soil characteristics, and community composition (Figure 1). Furthermore, all the individual elements of a climatic system are interconnected (Figure 2), such that change in any one compositional element (e.g., light radiation) can lead to change in another element (e.g., temperature) [26], generating more complicated effects on disease epidemics than expected. Currently, only simple interactions in the disease triangle, such as gradients of climatic factor(s) affecting pathogen

Glossary

Experimental evolution: a system for exploring evolutionary processes, consequences, and mechanisms by manipulating experimental conditions in the laboratory or controlled field.
Heritability: the proportion of phenotypic variation in a population caused by genetic differences among individual members attributable to gene structure alone (narrow-sense heritability) or gene structure together with intra- and intergene interaction (broad-sense heritability). It is a statistical estimate specific to one population in one environment and changes over time and space as circumstances change.
Pathogenicity: the ability of an infectious pathogen to induce disease in a plant host.
Plasticity: the capacity of species to alter their behavior, physiology, and/or morphology in response to environmental changes.
Polytrophic: a community and system biology model approach, considering the mechanisms and dynamic processes of multiple factors involved in host–pathogen–environment interactions.
Susceptibility: the extent to which a plant can be infected by a relevant pathogen.
Trade-off: an evolutionary phenomenon in which an increase in the performance of one functional trait leads to a decrease in the performance of another functional trait.
Virulence: the degree of damage caused to a host by a microbial pathogen.

Box 1. An example showing how the adaptation of pathogen to temperature change affects disease epidemics in the potato–*Phytophthora infestans* interaction

In the 21 *P. infestans* selected for acclimation [19], the mean disease growth rate in a detached leaf assay at 19°C (optimum temperature and assuming to be current temperature) and 22°C (assuming to be post-global warming air temperature) was 0.567/day and 0.402/day, respectively. After 5 consecutive months of acclimation to the elevated temperature, their average disease growth rate at 19°C and 22°C was 0.554/day and 0.518/day, respectively. Using the logistic model for disease epidemics and assuming the initial disease is 0.01 (maximum disease to be 1), the cumulative disease in the first 6 days decreases from 2.04 at 19°C to 1.33 (35%) at 22°C for the pre-acclimated pathogens but only decreases slightly from 1.99 at 19°C to 1.86 at 22°C (7%) for the post-acclimated pathogen. Therefore, the acclimated pathogen causes 40% more disease at 22°C compared with the pre-acclimated parental pathogen. Acclimation also changed the thermal profile of the pathogen. Its maximum, optimum, and minimum temperature for infection before acclimation is 30.3°C, 18.5°C, and 7.6°C, respectively. After acclimation, its maximum, optimum, and minimum temperature for infection were 31.5°C, 19.4°C, and 8.2°C, respectively. Using formula 3 in [20], it is estimated that the latent period in the acclimated pathogen is 4.5% shorter than the pre-acclimated pathogen. When incorporating the changed latent period into the logistic model, the accumulative disease at 22°C in the adapted pathogen is 2.01, which is 52% more than the pre-acclimated pathogen (1.33).

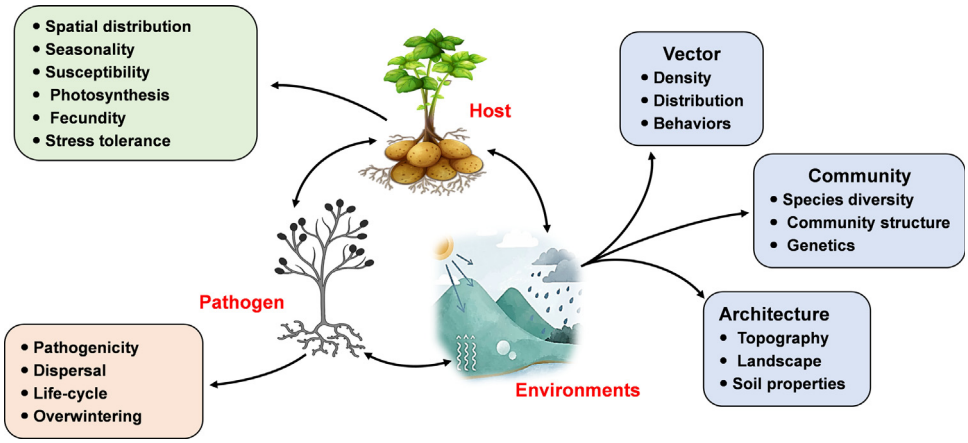

Figure 1. In the disease triangle, climate change can generate complex impacts on plants, pathogens, the environment, and the interactions among the three players. For plants, climate change can modulate their sensitivity and other biological, biochemical, and physiological activities, such as spatial distribution, seasonality, photosynthesis, fecundity, and stress resistance. For pathogens, climate change can affect their **virulence**, dispersal, and overwinter/oversummer ability, and life cycle such as spore production, sexuality, etc. Climate change can also regulate the ecological environment that supports plant–pathogen interactions either directly by regulating the density, distribution, and behavior of vectors and remodeling species diversity, community structure, and microbiome, or indirectly by inducing the changes of landscape and soil properties. Figure created with biorender.com, Adobe Illustrator 2020, and Microsoft PowerPoint.

infection or plant immunity [27], are usually considered when predicting the effects of climate change on future plant diseases. The effects of ecosystem shifts associated with climatic change on future plant diseases are lacking in the disease modeling literature (see Outstanding questions). Habitat reprofiling due to climate change has been documented in many species. For example, global warming has shifted some plants and their associated pathogens into ecosystems that are significantly different from the ones to which they are adapted, either by relocating them to cooler regions

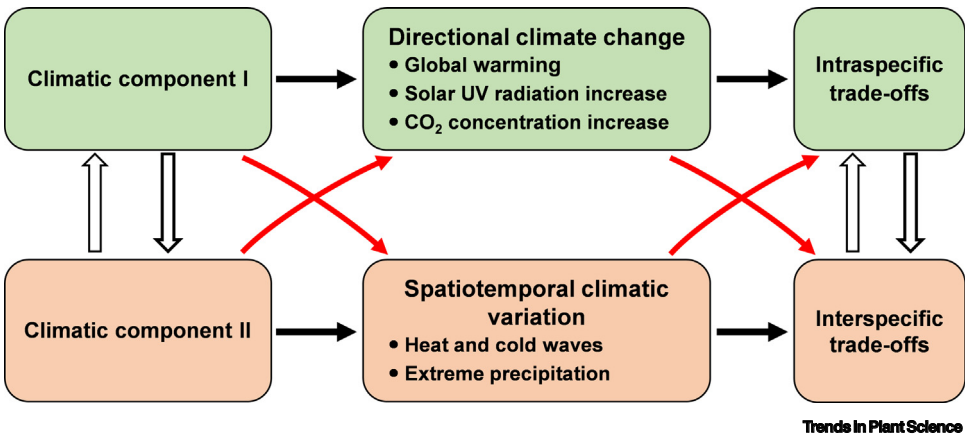

Figure 2. Impact of climate change on the development of trade-offs of plants, pathogens, and their interactions. Climate change is a phenomenon associated with changes in both mean and variability of the interconnected weather elements, creating a landscape of intra- and inter-specific trade-offs. Within species, functional trade-offs arise when an increase in the fitness of plant or pathogen trait associated with climate change leads to a decrease in the fitness of another plant or pathogen trait. Between species, functional trade-offs may emerge when plants and pathogens exhibit differential responses to climate change, where benefits associated with climate change in one species may adversely affect the performance of the counterpart species. Additionally, functional trade-offs can also be developed due to genetic and evolutionary differences in species adaptation to changes in the mean and variability of different climatic factors. Figure created with biorender.com, Adobe Illustrator 2020, and Microsoft PowerPoint.

and/or allowing them to thrive early in the season [28–30]. To ensure robust predictions, these demographical, genetic, physiological, and ecological elements should be integrated into future modeling frameworks.

Trade-offs in plant–pathogen interactions under climate change

Trade-offs are ubiquitous mechanisms that regulate biological and ecological processes in nature. They occur when the provision of one functional service decreases due to an increase of another functional service. In pathogens, greater pathogenicity often impedes other biological and ecological functions, such as intrinsic metabolic rate, spore production [31], transmission [32], stress tolerance [33], and competitive capacity [34]. In plants, higher disease resistance is usually negatively associated with germination, cellular growth, photosynthetic rates, leaf area, and seed production [35].

The impact of climate change on infectious plant diseases involves multiple biological and ecological trade-offs (Figure 2). Although pathogen pathogenicity and plant susceptibility play key roles in the outcome of host–pathogen interactions, disease development is also affected by a range of other functional traits each regulated by biological and biochemical processes that differ in their climatic preferences, such as optimum temperature and humidity [36]. How climate change may affect the development and epidemics of infectious plant diseases depends on both the intraspecies and interspecies trade-offs among pathogenicity, susceptibility, and other functional traits of plants and pathogens [37]. The differential effects of climatic change on trade-offs among these functional traits may have additive, multiplicative, antagonistic, or no effects on epidemics (see Outstanding questions). For example, if increased humidity associated with climate change enhances pathogen infection while suppressing plant immunity in a plant–pathogen interaction, there could be additive or even multiplicative effects on future disease epidemics. However, no epidemiological effects may be observed in another plant–pathogen interaction if temperature changes have opposite intraspecific effects on the development of functional traits such as pathogen infection or host susceptibility.

Climate change refers to long-term shifts in multiple weather patterns and is a phenomenon with many interconnected components. For example, an increase in air temperature can lead to a change in precipitation, ice duration, wind speed, etc., which in turn affects evaporation and soil properties, such as salinity (Figure 1). Due to trade-offs in the functional adaptation of hosts and pathogens to different climatic events, such as adaptation to heat stress associated with maladaptation to UV radiation [38], changes in these climatic events can have differential effects on pathogen pathogenicity, plant susceptibility, and other functional traits associated with host pathogen interactions. In addition, climate change is expected to be accompanied by more frequent and severe weather extremes, such as heat and cold, floods and droughts, as well as escalated temporal and spatial variation in climatic conditions [39]. Species have evolved different mechanisms for adapting to long-term, directional changes and immediate, stochastic fluctuations in climate events (Figure 3). Under directional changes of climatic magnitudes, adaptive mutations in genomes are often selected to maximize the fitness of plants and pathogens [40,41]. However, **plasticity** regulated by gene expression and enzymatic activity is expected to be more suitable for adapting to random fluctuation in climate events [42–44]. These trade-offs further complicate the impact of climate change on disease epidemics and may explain inconsistencies between laboratory predictions and field observations of infectious plant diseases.

Relative adaptability of plants and pathogens

Relative evolvability is an important factor that must be considered when modeling the impact of climate change on infectious plant diseases. Plants and pathogens differ remarkably in generating genetic variation and regulating gene expression for environmental adaptation. The relative contribution of these two events to species adaptation to climate change can be measured by

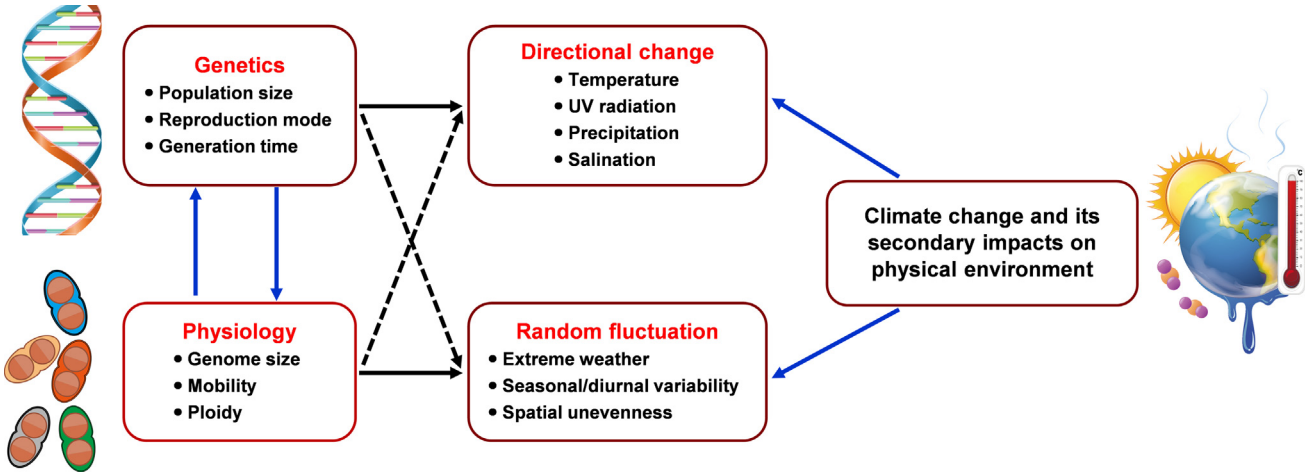

Figure 3. Mechanisms of adaptation to different forms of climate change. Climate change can be directional or increase spatial temporal fluctuations of weather events. The adaptation of species to climate change can be achieved both genetically through changes in genome structure to form new adaptive traits and physiologically through changes in gene expression and enzymatic activity (unbroken and broken black arrow). These two events are intertwined and can influence each other (blue arrows), with the physiological event acting as the initiation of species adaptation, which is then reinforced by the genetic event. In addition to genome characteristics, factors that regulate the formation of genetic adaptation include the mating system, generation time, and population size of species, while those affecting physiological adaptation include their genome size, mobility, and ploidy. Due to its immediate effects, physiological adaptation is expected to be a favored mechanism for adapting to fluctuating environmental stresses such as elevated spatiotemporal climate variability caused by climate change (unbroken black arrow). However, genetic adaptation should be a preferential mechanism selected for the adaptation of species to directional shifts in climatic means (unbroken black arrow). Figure created with biorender.com, Adobe Illustrator 2020, and Microsoft PowerPoint.

heritability and plasticity, respectively. Genome size, independent of its genetic information, can profoundly affect the development of functional traits in species from the subcellular to the organismal level [45–48] and interacts with climate change to influence plant–pathogen interactions and disease epidemics [49–51]. It is hypothesized that evolution and ecological adaptation of species can be constrained by large genomes, with accumulated empirical supports [45–48]. Pathogen genomes are often smaller than the corresponding plant hosts. Shorter generation time, and smaller genome size together with larger population size [52] and high reproduction rate [53], enable pathogens to accumulate more genetic variation than their plant partners in a short period of time, making them quicker to adapt to directional change in climate means. For example, pathogenicity of *P. infestans* and *Colletotrichum gloeosporioides* rapidly recovered or increased after several months of serial passaging under heat stress [19] and elevated CO_2 [54]. Such a rate of adaptation has not been documented yet in agricultural, forest, and wild plant immunity. Furthermore, haploidy in many pathogens ensures that new genetic variants generated can be rapidly selected by climatic stress and favorable mutants can come to dominate pathogen populations quickly. The epidemic consequences of this evolutionary gap are particularly worrying in the early stages of climate change and may contribute to the increased vulnerability of forest ecosystems to root rot caused by *Phytophthora citrophthora* and canker caused by *Phytophthora cinnamomi* [55]. The phenomenon is expected to gradually fade out as plant susceptibility and pathogen pathogenicity reach a new equilibrium.

Evolutionary shifts of climatic preferences in plants and pathogens

Species vary widely in their climate preferences, with some adapting to hot and humid climates, while others only thrive in cold, dry environments. Likewise, some species are restricted to small geographic areas due to their extreme sensitivity to climate fluctuations, while others can survive in many parts of the planet. However, climate preferences of a species are not homogeneous and constant. They may vary among intraspecies members, depending on their geographic origins

[56] and evolutionary signatures [57], and can evolve in response to shifts in local climatic regimes [58,59]. Taking the thermal reaction norm of pathogens as an example, it can coevolve with local climate (Figure 4). Indeed, experimental data have detected large variation in growth and infection temperatures (optima, boundary, and breadth) and adaptive potential to thermal stress among intraspecies members of plant pathogens from different thermal zones and altitudes [19,60]. Intraspecies members from warmer regions tend to have narrower temperature ranges suitable for growth and infection and require higher temperatures to achieve the same growth and infection rate [6] than those from cooler regions. Similarly, there are significant differences in UV tolerance and adaptive potential among intraspecies members from different altitudes [61–63]. Unfortunately, knowledge of intraspecies heterogeneity and evolutionary adaptation to climate preferences are largely missing from many plants and their associated pathogens, greatly hindering the robustness of current modeling predictions.

Infectious plant diseases in agricultural and natural ecosystems

Climate change may have a greater impact on infectious plant diseases in natural ecosystems than in agricultural ecosystems [64]. Although genetic variation in natural plants is generally higher compared with their counterparts in agricultural ecosystems [53], natural plants lack artificial evolution, like agricultural plants in which the adaptation of plants to climatic change can be greatly improved by continuous and timely integration of adaptive traits into crop varieties through breeding, making natural plants more vulnerable to climate change and pathogen infection [65]. Furthermore, unlike agricultural ecosystems, the effects of climate change on host–pathogen interactions in natural ecosystems is generally not suitable for mitigation through field practices and the implementation of plant protection systems. For example, the influence of short-term precipitation reductions associated with climate change on the growth and physiology of agricultural plants, and thus their susceptibility to pathogens, can be greatly reduced by irrigation systems, but similar practices cannot be easily implemented in natural ecosystems. To evaluate sustainability, these such mitigations should be considered when simulating the impact of climate change on future plant diseases.

Concluding remarks: opportunities and technologies for developing sophisticated models based on eco-evolutionary knowledge

Current modeling attempts provide a useful starting point for inferring future infectious plant diseases but may be far from reliable due to a lack of evolutionary understanding of the complex

Outstanding questions

Which functional traits (e.g., seasonality, sexuality, ecological heterogenicity, see Figure 1 for more information) in addition to plant susceptibility and pathogen pathogenicity are particularly important for species adaptation to climate change?

How do the ecosystems created by climate change affect plant–pathogen interactions and how can this be incorporated into modeling frameworks?

How does climate change reformulate intra- and inter-specific trade-offs of functional traits and pass the adaptation events sequentially to the disease triangle?

What are the mechanisms and relative adaptability of plants and pathogens to different types of climate change in agricultural and natural ecosystems?

How have climate preferences of species developed and how may they coevolve with climate change?

What are the roles of human society in mitigating climate change and, consequently, the management of future plant disease epidemics?

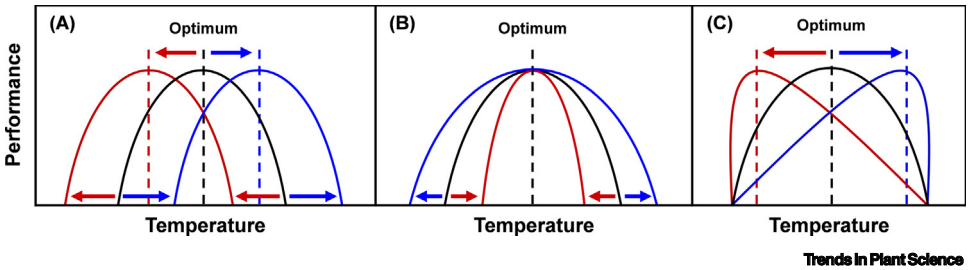

Figure 4. Hypothetical chart showing the evolutionary modifications of thermal reaction norms in response to climate change. Four scenarios could be observed. (A) In scenario 1, the optimum temperature of the species decreases or increases but thermal breadth (maximum–minimum temperature) does not change, resulting in a horizontal movement of the thermal reaction norm (black line) to the left (red line) or right (blue line), respectively. (B) In scenario 2, species thermal boundaries (maximum and minimum temperature) and breadth are altered but optimum temperature does not, shrinking (red line) or widening (blue line) the thermal reaction norm from the original (black line). (C) In scenario 3, the optimal temperature for the species changes but the thermal boundary and breadth do not, skewing the original thermal norm (black line) to the left (red line) or right (blue line). Scenario 4 will change all temperature parameters (boundaries, optima, and breadth) of the species (combination of A, B, and C). Scenario 4 is the most likely expectation and scenario 3 is the least likely expectation. Figure created with biorender.com, Adobe Illustrator 2020, and Microsoft PowerPoint.

impacts of climate change on interplays among pathogens, hosts, and ecosystems. To mitigate this modeling problem, knowledge of adaptive responses in the climatic preferences of hosts and pathogens to climate changes, and of intra- and inter-species functional trade-offs associated with such climate changes, is urgently needed (see Outstanding questions). Such adaptive data can be gradually accumulated through multidisciplinary collaboration with advances in technology and experimental approaches. For example, genes that contribute to pathogen pathogenicity, plant susceptibility, and other important traits can be effectively characterized by molecular, functional, and genomic analyses. Trade-offs and climate preferences in functional traits within and among plant and pathogen species can arise through population genetics and eco-evolutionary studies of relevant species [66]. The scale of climate change can be better projected due to advances in meteorological studies associated with the development of highly sensitive instruments and use of satellite-based measurements. The mutual influences and impacts of climate change on trait development, evolution of climate preference, and ecosystem can be evaluated by **experimental evolution** approaches, while advances in modeling technology allow the integration of these demographical, genetic, physiological, and eco-evolutionary elements into polytrophic modeling frameworks to ensure robust predictions.

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Declaration of interests

The authors have no interests to declare.

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