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Climate Extremes and the Plant Disease Triangle: A Critical Synthesis of Host, Pathogen and Microbiome Mechanisms and their Implications for Crop Protection

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Review Article

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Abstract

The disease triangle, in which disease arises from the intersection of a susceptible host, a virulent pathogen and a conducive environment, has organised plant pathology for more than half a century. Its environmental apex was conceived as a relatively stable backdrop, yet heatwaves, compound drought and heat episodes, extreme humidity and short-duration flooding now perturb that apex on timescales shorter than a single crop cycle. This review critically evaluates whether the accumulated mechanistic, ecological and agronomic evidence justifies a structural revision of the framework, and what such a revision would mean for crop

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protection. Literature was identified through open scholarly indexes and institutional sources, appraised for design adequacy and translational relevance, and synthesised thematically rather than catalogued. Three conclusions are defensible. Moderately elevated temperature suppresses salicylic-acid-dependent immunity through identifiable molecular nodes, and this suppression has been partially reversed by genetic manipulation, which establishes mechanism rather than mere correlation. Water status functions as an actively contested resource rather than a passive variable, since bacterial effectors manipulate host abscisic acid signalling to hydrate the leaf apoplast. Drought and warming reshape root and leaf microbiota in partially conserved directions, although the inference that such reshaping causes disease remains supported mainly by association, by artificial community reconstitution and by a small number of host-genetic experiments. Confidence is weakest where it matters most for agriculture: almost all mechanistic work uses a narrow set of model pathosystems under constant-temperature growth conditions, field validation under realistic fluctuating extremes is scarce, and microbiome interventions have rarely been tested for efficacy under the stress conditions that justify them. The evidence supports treating the microbiota as a modifier that operates within each apex rather than as a self-contained fourth apex, and supports prioritising thermoresilient resistance, stress-validated biological products and extremes-aware forecasting. Claims that climate extremes uniformly intensify plant disease are not supported; the direction of change is pathosystem-specific and frequently non-monotonic.

Keywords: Climate extremes; disease triangle; plant immunity; phytobiome; heat stress; dysbiosis; crop protection; plant pathology.

1. Introduction

Plant disease has been conceptualised since the middle of the twentieth century as the outcome of a coincidence among three necessary conditions: a susceptible host, a virulent pathogen and an environment conducive to infection. The resulting disease triangle is taught as the organising abstraction of the discipline and underpins the logic of quarantine, resistance breeding, fungicide timing and disease forecasting. The framework achieved its authority partly because the environmental apex behaved, for practical purposes, as a slowly varying boundary condition. Growing seasons recurred within a familiar envelope, and the deviations that mattered to epidemiologists were largely intra-seasonal weather events rather than shifts in the envelope itself.

That assumption no longer holds. Observed increases in the frequency and intensity of hot extremes over most land regions, alongside intensification of heavy precipitation and of agricultural and ecological drought in many regions, are now attributed with high confidence to human influence (Seneviratne *et al*., 2021). Compound events, in which drought and heat coincide or follow one another, are increasing faster than either component alone and are projected to respond steeply to additional warming (Zhang *et al*., 2022; Zscheischler *et al*., 2018, 2020). The environmental apex has therefore acquired properties that the original formulation did not anticipate: rapid excursion beyond historical ranges, correlated rather than independent stressors, and persistence of effects into subsequent seasons through soil and microbial legacies. A recent global meta-analysis further showed that the joint effects of warming or drought and pathogen attack are often non-additive and vary with pathosystem and experimental context, reinforcing the need to evaluate climatic and biotic drivers together rather than as independent components (Gallego-Tévar *et al*., 2024).

The consequences for plant health are not hypothetical. Pathogens and pests already remove an estimated fraction of global production in the five crops that supply most human calories, with the heaviest losses concentrated in regions of high food insecurity (Savary *et al*., 2019). Fungal and oomycete pathogens continue to emerge and spread between continents, and the emergence of wheat blast in South Asia and subsequently in Africa demonstrates that a pathogen restricted to one continent for decades can establish elsewhere within a single season given suitable warm and humid conditions (Ceresini *et al*., 2018; Fones *et al*., 2020; Islam *et al*., 2016; Latorre *et al*., 2023; Ristaino *et al*., 2021). Institutional assessments have reached comparable conclusions about the scale of climate-related plant-pest risk (IPPC Secretariat, 2021).

Two developments complicate the simple expectation that a warmer and more erratic climate will uniformly increase disease. The first is mechanistic. Temperature does not act as a generic accelerator of infection; it acts on specific molecular nodes in host immunity, in pathogen virulence programmes and in the physical environment of the leaf and root. Moderately elevated temperature has been shown to suppress salicylic acid biosynthesis and downstream defence transcription in *Arabidopsis thaliana* while simultaneously promoting the deployment of bacterial virulence factors, producing a dual effect that neither host-focused nor pathogen-focused studies would have revealed alone (Huot *et al*., 2017; Kim *et al*., 2022). Temperature also modulates the function of individual resistance genes in crops, sometimes enhancing and sometimes abolishing their effect (Chen *et al*., 2018; Onaga *et al*., 2017; Yan *et al*., 2021). Generalisation from any single pathosystem is therefore unsafe. A recent systematic review across agriculturally important crops likewise reported that disease responses differ among altered temperature, water availability, humidity and atmospheric CO2 treatments and their combinations, underscoring the context dependence of climate–disease relationships (Méndez *et al*., 2026).

The second development is ecological. Plants are not confronted by pathogens in isolation but within a dense assemblage of resident bacteria, fungi and other microorganisms that occupy the same tissues and compete for the same resources (Trivedi *et al*., 2020). This assemblage is itself strongly restructured by drought and by warming, in directions that appear partially conserved across hosts and soils (Naylor *et al*., 2017; Santos-Medellín *et al*., 2021; Xu *et al*., 2018). Host genetic networks that maintain microbiota composition have been identified, and their disruption produces leaf microbial communities that are abnormal in composition and associated with tissue damage in the absence of a designated pathogen (Chen *et al*., 2020; Paasch *et al*., 2023; Pfeilmeier *et al*., 2021). Proposals to add the microbiota as a fourth apex, converting the triangle into a tetrahedron or pyramid, follow naturally from this evidence, and a formal re-envisioning of the framework around host microbiota and health outcomes has been advanced (Leveau, 2024). Recent experimental work in wheat further showed that drought-induced shifts in microbiome composition and

rhizosphere metabolites can have functional consequences for host drought resistance, supporting the view that stress-driven microbial restructuring is not necessarily a passive ecological response (Li *et al*., 2025).

Existing reviews have addressed components of this problem with authority but have not integrated them. Syntheses of climate change and plant pathogens have concentrated on distributional and epidemiological consequences and on food-security framing (Bebber, 2015; Kumar & Mukhopadhyay, 2025; Singh *et al*., 2023). Molecular reviews have examined temperature and immunity in depth, with the disease triangle used as an organising device rather than as an object of critical scrutiny (Desaint *et al*., 2021; Roussin-Léveillée *et al*., 2024; Velásquez *et al*., 2018; Zarattini & Fagard, 2026). Microbiome reviews have described stress-driven community change and its potential for exploitation, generally without asking whether the causal chain from community change to disease outcome has been demonstrated (Arnault *et al*., 2023; Liu *et al*., 2020; Trivedi *et al*., 2020). The framework itself has been examined conceptually, but largely by proposing extensions rather than by testing whether extension improves explanatory or predictive performance (Chappelka & Grulke, 2016; Grulke, 2011; Leveau, 2024). No recent synthesis has asked, across these literatures at once, whether climate extremes require a structural revision of the disease triangle, which specific evidence would support such a revision, and what the revision would change in practice.

1.1 Scope, Research Gap and Objectives

This review examines how climate extremes, defined as episodic excursions of temperature, atmospheric and soil water status, and their compound occurrence beyond the range to which a cropping system is adapted, alter the relationships among host, pathogen, environment and resident microbiota, and what those alterations imply for crop protection. Coverage is restricted to terrestrial vascular plants, with emphasis on annual field crops and with evidence from model species and from natural plant communities included where it establishes mechanism or provides ecological contrast. Fungal, oomycete, bacterial and viral pathogens are within scope; arthropod pests are considered only as virus vectors and only where climate modifies transmission.

Several domains are deliberately excluded. Gradual mean warming is treated as context rather than as the subject, because the mechanistic and agronomic consequences of episodic extremes differ from those of slow trends. Post-harvest pathology is excluded except where toxigenic fungi link field water stress to food safety. Economic modelling, trade policy and phytosanitary regulation are outside the analytical remit, as are marine and aquatic plant pathosystems.

The gap this review addresses is specific. Mechanistic plant pathology, microbial ecology and climate-impact assessment have each produced substantial evidence bearing on the disease triangle under extremes, but they employ incompatible units of observation, timescales and standards of causal inference, and no synthesis has evaluated whether their combined weight justifies revising the framework. The objectives follow from that gap. The review first establishes what the conceptual status of the disease triangle is once the environmental apex becomes non-stationary, and whether proposed extensions are analytically productive. It then appraises the mechanistic evidence for temperature and water extremes acting on host immunity and on pathogen virulence, distinguishing demonstrated molecular mechanism from inferred association. It next evaluates the evidence that climate extremes disrupt plant-associated microbial communities and asks whether disruption has been shown to cause altered disease outcomes rather than merely to accompany them. It then examines interactions among apices under compound extremes, where additive reasoning is least reliable. Finally, it assesses which crop protection strategies are supported by the evidence, where the field-efficacy evidence is thinnest, and which research designs would most efficiently resolve the remaining uncertainty. The intended contribution is an integrated and explicitly calibrated account that separates what is established from what is plausible, and that identifies the specific experimental deficits that currently prevent confident prediction.

2. Methods for Literature Selection

2.1 Review Design and Rationale

A critical narrative design was selected because the review question is conceptual and integrative rather than estimative. The governing question asks whether a theoretical framework requires revision and what the practical consequences would be, and it draws on molecular genetics, microbial community ecology, field epidemiology and climate science. These literatures report incommensurable outcomes, including transcript abundance, bacterial colony-forming units, community dissimilarity indices, disease incidence and modelled infection risk, and they operate on timescales ranging from hours to decades. No common effect measure exists across them, so a quantitative synthesis would be statistically inadmissible and a systematic review restricted to one comparable subset would answer a narrower question than the one posed. Critical narrative synthesis is the appropriate instrument when the objective is to interrogate the coherence of an explanatory framework and to grade the strength of competing interpretations. That choice does not license selective citation or the omission of appraisal. Transparency of sources, explicit inclusion reasoning and open statement of where the evidence is weak are applied throughout, in the manner recommended for narrative reviews intended for scholarly rather than expository purposes.

2.2 Information Sources

Searches were conducted in OpenAlex, Crossref, Europe PMC, the Directory of Open Access Journals, Semantic Scholar and Google Scholar. Institutional sources were consulted directly for assessment reports and reviews issued by the Intergovernmental Panel on Climate Change and by the Food and Agriculture Organization of the United Nations acting on behalf of the Secretariat of the International Plant Protection Convention. Database searching was supplemented by backward citation searching from recent reviews and framework papers, by forward citation searching on highly cited mechanistic studies, by related-article retrieval, and by targeted checks for corrections, author corrections and expressions of concern associated with candidate references. Bibliographic

details of every retained source were confirmed against registry metadata, and each digital object identifier was verified to correspond to the cited article.

2.3 Search Strategy and Search Period

Search concepts were constructed from five elements of the review question: the climatic driver, the disease outcome, the host immune or physiological process, the microbial community, and the crop protection response. Within each element, controlled and free-text synonyms were combined with the Boolean operator OR, and elements were intersected with AND. Field restriction to title and abstract was applied where the index permitted it, in order to suppress the large volume of records in which climate terms appear only in introductory framing. Truncation and phrase searching were used where supported. Searches were iteratively refined as terminology gaps became apparent, in particular for water-status vocabulary, where apoplast hydration, water soaking and relative humidity are used inconsistently across subfields.

The search period extended from 1 January 2005 to 22 July 2026. The starting year reflects the point at which molecular analysis of temperature-dependent plant immunity and quantitative assessment of climate effects on plant disease had both become established research programmes, providing a coherent body of evidence rather than isolated observations. Foundational publications predating this boundary were included where necessary to establish conceptual origin.

Table 1 sets out the concepts and representative strings actually used. The strings are presented in generic Boolean form rather than in index-specific syntax, since field tags differ among the sources consulted; the concept combinations, not the tags, determine what was retrieved. The table also records the principal retrieval problem encountered for each concept, which is informative in its own right. Concept 3 and Concept 4 proved the most difficult to search reliably, because immune-signalling terminology is gene-specific and because microbiome studies frequently describe stress in titles without naming the community outcome. Retrieval for these concepts depended disproportionately on citation searching rather than on term matching, which is itself a limitation on reproducibility.

Table 1. Search concepts, representative Boolean strings and principal retrieval limitations

Concept	Representative search string (generic Boolean form)	Principal retrieval limitation	Supporting references
1. Climatic driver	("climate extreme*" OR "heat wave" OR heatwave OR "extreme temperature" OR drought OR "compound event*" OR "elevated humidity" OR waterlogging OR flooding)	Climate terms occur in framing text of unrelated studies; title and abstract restriction was essential	Seneviratne *et al*. (2021); Zhang *et al*. (2022); Zscheischler *et al*. (2018, 2020)
2. Disease outcome	("plant disease" OR "plant patho*" OR "disease severity" OR "infection risk" OR "crop loss" OR "disease triangle")	"Disease triangle" retrieves unrelated biomedical usages of the same phrase	Grulke (2011); Leveau (2024); Savary *et al*. (2019)
3. Host process	("plant immunity" OR "effector-triggered immunity" OR "pattern-triggered immunity" OR "salicylic acid" OR "abscisic acid" OR "resistance gene" OR NLR) AND (temperature OR heat OR humidity OR drought)	Gene-specific vocabulary; many relevant studies name only the gene, requiring forward citation searching	Desaint *et al*. (2021); Kim *et al*. (2022); Saijo & Loo (2020)
4. Microbial community	("root microbiome" OR "rhizosphere microbiome" OR phyllosphere OR phytobiome OR microbiota OR dysbiosis OR "synthetic community" OR SynCom) AND (drought OR warming OR "heat stress" OR "stress")	Community outcome frequently absent from title; abstracts emphasise stress treatment rather than microbial result	Arnault *et al*. (2023); Naylor *et al*. (2017); Trivedi *et al*. (2020)
5. Crop protection response	("disease management" OR "biological control" OR "microbiome engineering" OR "resistance breeding" OR "disease forecasting" OR "integrated pest management") AND (climate OR temperature OR drought)	Applied literature is dispersed across crop-specific outlets with heterogeneous indexing	French *et al*. (2021); Garrett *et al*. (2022); Richard *et al*. (2022)

Note. Strings are shown in generic Boolean form because field tags differ among the sources consulted. Truncation is indicated by an asterisk. Searches were restricted to title and abstract where the source permitted. NLR = nucleotide-binding leucine-rich repeat receptor; SynCom = synthetic microbial community

2.4 Eligibility Criteria

Peer-reviewed primary research and reviews in indexed journals were eligible, together with assessment reports and scientific reviews issued by recognised intergovernmental bodies. Studies were included when they reported an effect of temperature, water status or their combination on a host immune or physiological process relevant to infection, on pathogen growth, virulence or distribution, on the composition or function of a plant-associated microbial community, or on disease outcome in a crop or a

comparable wild pathosystem. Conceptual and methodological papers were eligible where they addressed the structure of the disease triangle or the appraisal of narrative reviews.

Studies were excluded when the climatic variable was not manipulated or measured, when disease or microbial outcomes were reported without a stated environmental treatment, when the work concerned post-harvest storage pathology without a field water-stress link, and when it addressed aquatic or marine hosts. Commercial technical literature, non-scholarly commentary, conference abstracts without full reports and sources whose bibliographic identity could not be confirmed were excluded. Preprints were not used as evidence where peer-reviewed work on the same question was available. English-language publications were used, which is a recognised source of bias addressed in the final section. No geographical restriction was applied, and evidence from tropical and subtropical systems was actively sought because the temperate bias of mechanistic plant pathology is itself an analytical problem for this review.

2.5 Screening, Duplicate Handling and Study Selection

Records retrieved from each source were screened first by title and abstract against the eligibility criteria, then by examination of the full report for those that passed. Duplicates arising from overlapping index coverage were identified by digital object identifier where present and by matching of author, title and year where absent, and were resolved to a single record with the most complete metadata retained. Records without a resolvable identifier and without confirmable journal metadata were not carried forward. Where an index returned a preprint and a subsequent journal article describing the same work, the journal article was retained. Corrections and author corrections were checked for every retained source, and the corrected metadata were used. No source was retained when its bibliographic identity remained uncertain after this process. Record counts are not reported, because the selection process was purposive and iterative rather than a single enumerated screening pass, and reporting counts would misrepresent the design as systematic.

2.6 Critical Appraisal and Evidence Synthesis

Appraisal was conducted against criteria appropriate to each study type rather than by a single instrument, and no formal risk-of-bias score was assigned, since no validated tool applies across molecular genetics, community sequencing and epidemiological modelling. Experimental studies were assessed for the realism of the environmental treatment, in particular whether temperature was constant or fluctuating and whether humidity was controlled independently of temperature; for the presence of genetic or chemical complementation establishing mechanism rather than correlation; for replication and independent confirmation; and for whether the reported effect was tested in more than one host genotype or pathogen isolate. Community sequencing studies were assessed for compositional data handling, for the separation of relative from absolute abundance, for whether community change was linked to a functional or disease outcome, and for whether findings were reproduced across soils or sites. Modelling and distributional studies were assessed for the ecological realism of the thermal response functions used, for the treatment of host phenology and management, and for the handling of uncertainty.

Themes were developed inductively from the appraised evidence and then tested against the review objectives, with the disease triangle used as the organising axis so that evidence bearing on each apex, and on the interactions among apices, could be compared directly. Where studies disagreed, the disagreement was retained and its likely origin examined, with particular attention to differences in thermal regime, host genotype, pathogen lifestyle and the distinction between controlled-environment and field observation. Convergence among independent designs was treated as the principal basis for confidence, and single-system findings, however mechanistically elegant, were graded as provisional until reproduced in a second pathosystem or under field conditions.

3. The Disease Triangle under a Non-Stationary Environment

3.1 What the Classical Formulation Assumes

The triangle's analytical power derives from a claim of joint necessity: disease requires all three components, and the area of the triangle scales with the favourability of each. Two assumptions are embedded in this formulation and were rarely problematic while climate remained within its historical envelope. The first is that the apices are separable, so that an environmental effect on the host can be distinguished from an environmental effect on the pathogen. The second is that the environment is exogenous, acting on host and pathogen without itself being modified by them.

Both assumptions fail under climate extremes, and they fail in ways that matter for prediction rather than merely for elegance. Separability fails because the same thermal excursion acts simultaneously and in opposite directions on the two biological apices. Elevated temperature in the range of 28 to 30 degrees Celsius suppresses accumulation of the defence hormone salicylic acid in *Arabidopsis thaliana* while promoting expression of the type III secretion system that *Pseudomonas syringae* requires for virulence, so the net outcome cannot be recovered from host-side or pathogen-side measurements alone (Huot *et al*., 2017). The exogeneity assumption fails because pathogens actively construct the microenvironment on which their success depends. Bacterial effectors hijack host abscisic acid signalling to close stomata and hydrate the leaf apoplast, converting humidity from an external condition into a contested and partly pathogen-determined resource (Hu *et al*., 2022; Roussin-Léveillée *et al*., 2022; Xin *et al*., 2016).

A third difficulty arises from timescale. The classical triangle is evaluated at the moment of infection, whereas extremes exert effects that persist well beyond that moment. Prolonged drought imparts compositional changes to the rice root microbiome that remain detectable after rewatering and into subsequent growth, indicating that the environmental apex carries a memory not represented in the instantaneous formulation (Santos-Medellín *et al*., 2021). Soil-borne legacies of aboveground infection operate on

the same principle in reverse, with root exudate changes during infection altering the microbial community encountered by the following crop (Yuan *et al*., 2018).

3.2 Proposed Extensions and their Analytical Cost

Several extensions of the framework have been proposed. The most common adds human management or time as a fourth vertex, generating a disease tetrahedron. A more recent and more substantial proposal integrates the host microbiota fully into the framework and shifts the focal outcome from disease to plant health, on the argument that the resident community determines whether a pathogen encounter results in disease at all (Leveau, 2024). Other treatments have retained three apices but redefined the environmental apex to include chemical and physical anthropogenic change, arguing that pollutants and altered atmospheric composition disrupt the triangle rather than merely shifting it (Chappelka & Grulke, 2016; Grulke, 2011).

These proposals are not equivalent in what they cost. Adding a vertex increases descriptive completeness but reduces the framework's discriminating power, because a model in which every relevant factor occupies its own apex cannot be falsified by any observed outcome. The microbiota proposal is the strongest of the three on empirical grounds, since host genetic control of microbiota composition has been demonstrated and its disruption produces measurable tissue damage (Chen *et al*., 2020; Paasch *et al*., 2023; Pfeilmeier *et al*., 2021). It is also the most conceptually awkward, because the microbiota is not a separable factor of the same logical type as host, pathogen and environment. Resident microorganisms are simultaneously part of the host phenotype, competitors of the pathogen and a component of the environment the pathogen experiences. Placing them at a fourth apex implies an independence they do not possess.

A more defensible reading treats the microbiota as a modifier operating within each existing apex rather than as a new apex. Under this reading, resident communities alter host susceptibility by priming or suppressing immune signalling, alter pathogen success through resource competition and antibiosis, and alter the effective environment by changing the moisture, nutrient and chemical conditions at the infection court. The framework is then retained in its three-apex form, with the explicit acknowledgement that each apex is biologically composite. This interpretation accommodates the same evidence as the tetrahedral proposal without asserting a separability the data do not support, and it generates a sharper research question: not whether the microbiota belongs in the framework, which is not seriously disputed, but through which apex a given microbial effect is transmitted, and whether that pathway is measurable.

3.3 Consequences for Prediction Rather than for Teaching

The practical question is whether any revision improves prediction. Here the evidence is discouraging in an instructive way. Global assessments that combine pathogen thermal response functions with crop distribution project increased infection risk at higher latitudes and in some cases decreased risk in low-latitude regions where temperatures exceed pathogen optima, and this pattern tracks yield projections closely enough to suggest that thermal matching captures a real first-order signal (Chaloner *et al*., 2021). The same approach cannot capture the mechanisms described above, because it represents neither host immune suppression nor pathogen manipulation of apoplast water nor microbial community state. Distributional projections and mechanistic studies therefore currently occupy separate epistemic compartments, and the framework revision debate has not yet produced a formalism that bridges them. Any claim that the triangle has been successfully redrawn should be judged against that standard rather than against diagrammatic completeness.

Table 2 compares the principal formulations on the criteria that bear on this judgement. The comparison shows that no current formulation combines mechanistic fidelity with predictive tractability, and that the microbiota-integrated formulation gains explanatory reach at the cost of parameter identifiability. This is the central conceptual finding of the present synthesis, and it frames the mechanistic sections that follow: the value of the mechanistic evidence lies not in decorating the framework with additional vertices but in specifying which apex-level parameters actually change under extremes, and by how much.

Table 2. Comparison of formulations of the plant disease framework against criteria relevant to prediction under climate extremes

Formulation	Core claim	What it captures well	What it fails to capture	Parameter identifiability	Supporting references
Classical three-apex triangle	Disease requires simultaneous sufficiency of host susceptibility, pathogen virulence and conducive environment	Necessity conditions; logic of quarantine, resistance and forecasting	Bidirectional environment-organism effects; carry-over between seasons	High; each apex is operationally definable	Grulke (2011); Savary *et al*. (2019)
Tetrahedron with management or time as fourth vertex	Human intervention and temporal dynamics act as an independent determinant	Agronomic reality of managed systems	Molecular mechanism; microbial mediation	Moderate; management is measurable but not comparable across systems	Chappelka & Grulke (2016)
Microbiota-integrated framework with	Resident microbiota determine whether pathogen encounter	Dysbiosis evidence; protective and disease-permissive	Quantitative contribution of the microbiota relative	Low; community state is high-dimensional and	Leveau (2024); Paasch *et al*. (2023);

Formulation	Core claim	What it captures well	What it fails to capture	Parameter identifiability	Supporting references
health as the focal outcome	results in disease	community states	to host genotype	context-dependent	Pfeilmeier *et al*. (2021)
Three-apex triangle with composite apices (position taken here)	Microbiota act as modifiers within each apex rather than as a separate apex	Mechanistic routing of microbial effects; retains falsifiability	Emergent community-level properties not reducible to apex effects	Moderate; requires apex-specific attribution experiments	Chen *et al*. (2020); Trivedi *et al*. (2020); Velásquez *et al*. (2018)
Correlative bioclimatic projection	Disease risk follows the match between pathogen thermal requirements and realised climate	Large-scale spatial and temporal risk patterns	Immune suppression; apoplast water manipulation; microbial state	High but mechanistically shallow	Bebber (2015); Chaloner *et al*. (2021)

Note. Parameter identifiability refers to the practical feasibility of estimating the framework's components from available field or experimental data rather than to theoretical identifiability

4. Thermal Extremes and the Host Apex

4.1 The Salicylic Acid Node and the Strength of the Mechanistic Case

The best-supported mechanism by which warming alters plant disease operates through salicylic acid. In *Arabidopsis thaliana*, a shift from approximately 23 to 28 degrees Celsius reduces accumulation of salicylic acid and expression of its biosynthetic and signalling genes, and this reduction is accompanied by increased susceptibility to *Pseudomonas syringae* (Huot *et al*., 2017). The same work established that the temperature effect is not confined to the host, since the bacterial type III secretion system is more strongly deployed at the elevated temperature, so that a single environmental variable moves host and pathogen in opposing directions relative to disease resistance.

The decisive advance was the identification of a specific thermosensitive node. Warm temperature was shown to impair the recruitment of the transcriptional regulators CBP60g and SARD1 to the promoters of salicylic acid biosynthesis genes, and constitutive expression of CBP60g restored salicylic acid accumulation and pathogen resistance at the elevated temperature without the growth penalty typically associated with constitutive defence activation (Kim *et al*., 2022). That the effect was reproduced in a crop species of the genus *Brassica* moves this result beyond the model system and converts a correlative observation into a manipulable mechanism. Genetic complementation of this kind is the appropriate evidential standard for causal claims in this literature, and it has been met for this node but for very few others.

The picture is less uniform when the two branches of the immune system are considered separately. Moderately elevated temperature has been reported to favour pattern-triggered responses while suppressing effector-triggered outputs in *Arabidopsis thaliana*, indicating that the immune system is not simply downregulated by warming but reconfigured (Cheng *et al*., 2013). Effector-triggered immunity itself responds heterogeneously, since elevated temperature abolished the hypersensitive cell-death response associated with some effector-receptor pairs while leaving bacterial growth restriction intact for others, which implies that thermosensitivity resides in particular downstream outputs rather than in recognition as such (Menna *et al*., 2015). Short episodes rather than sustained conditions are also sufficient to compromise defence, since a transient heat treatment suppressed flagellin-induced responses and reduced bacterial resistance, with recovery over a period of days (Janda *et al*., 2019). This last observation is agronomically important because it matches the duration of a realistic heatwave rather than the sustained shift used in most controlled experiments.

The immunity-growth trade-off provides a further layer. The small ubiquitin-like modifier ligase SIZ1 was shown to mediate a temperature-dependent balance between immunity and growth in *Arabidopsis thaliana*, so that the same regulatory machinery that suppresses defence at elevated temperature supports thermomorphogenic growth (Hammoudi *et al*., 2018). Interpreted through the disease triangle, this indicates that the host apex does not merely weaken under heat but is reallocated, and that breeding for thermoresilient immunity will encounter a physiological constraint rather than a simple deficiency. Recent appraisals of the cellular basis of thermal sensitivity and of the prospects for engineering around it reach a compatible conclusion, while noting that the number of validated nodes remains small (Castroverde *et al*., 2025; Thakur & Yang, 2026; Zarattini & Fagard, 2026).

4.2 Temperature Dependence of Crop Resistance Genes

Evidence from crops complicates any general statement that heat weakens resistance. Several characterised resistance genes are effective specifically at elevated temperature. The wheat stem rust resistance gene Sr21 confers resistance to the Ug99 race group of *Puccinia graminis* f. sp. *tritici* at high temperature and is ineffective at lower temperature, a pattern opposite to the conventional expectation (Chen *et al*., 2018). The wheat leaf rust resistance gene Lr13 likewise operates as a high-temperature resistance gene, with the additional complication that the underlying allele is associated with hybrid necrosis, so its deployment carries a breeding cost (Yan *et al*., 2021). In rice, the executor gene Xa7 confers resistance to bacterial blight that is enhanced rather than lost under elevated temperature, and this property has been explicitly characterised as climate resilience (Chen *et al*., 2021; Zhao & Zhang, 2021).

Opposing examples are equally well documented. Resistance conferred by the rice blast gene Pi54 against *Magnaporthe oryzae* was substantially reduced at elevated temperature in two genetic backgrounds, with the magnitude of the loss depending on the background, which indicates that thermosensitivity is a property of the gene within a genome rather than of the gene alone (Onaga *et al*., 2017). In the *Ralstonia solanacearum* pathosystem, elevated temperature changed the genetic architecture of quantitative resistance, with loci effective at standard temperature losing effect and previously undetected loci becoming detectable (Aoun *et al*., 2017). Solanaceous hosts under combined high temperature and high humidity have been shown to shift toward cytokinin-mediated defence rather than to lose defence altogether, again indicating reconfiguration rather than simple suppression (Yang *et al*., 2022). Temperature increase has also been reported to modify susceptibility to *Verticillium* wilt in species of *Medicago* and to favour more aggressive pathogen strains, introducing a pathogen-evolutionary dimension to the same variable (Sbeiti *et al*., 2023).

The unifying interpretation is that thermal sensitivity is a property of individual resistance mechanisms rather than of plant immunity as a category, and that its direction is not predictable from first principles. This has a direct implication for breeding programmes, which have historically characterised resistance under standard glasshouse conditions and have therefore had limited capacity to distinguish genes that will fail under heatwave conditions from genes that will not.

4.3 Cold and the Neglected Tail of the Distribution

Climate extremes are frequently equated with heat, which obscures a well-characterised set of low-temperature effects. Low temperature enhances salicylic-acid-dependent immunity in *Arabidopsis thaliana* through genes that are repressed by ethylene, and calmodulin-binding transcription activators mediate the interaction between cold exposure and salicylic acid pathway genes (Kim *et al*., 2017; Li *et al*., 2020). The cold-signalling regulator INDUCER OF CBF EXPRESSION 1 directly activates salicylic acid signalling, providing a molecular route by which cold acclimation and immunity are coupled (Li *et al*., 2024). These findings indicate that the temperature-immunity relationship is not monotonic and that warming may relieve as well as impose constraints depending on the starting point. In cropping systems where late frosts persist or become more variable, both tails of the temperature distribution are relevant, and treating extremes as synonymous with heat introduces a systematic bias into risk assessment.

4.4 From Controlled Environments to the Field

Three features of the experimental base limit translation. The first is the near-exclusive use of constant elevated temperature, whereas field extremes are diurnally fluctuating episodes superimposed on a seasonal cycle, and the transient-heat result indicates that duration and recovery are themselves determinants of outcome (Janda *et al*., 2019). The second is taxonomic concentration, since the mechanistic literature is dominated by *Arabidopsis thaliana* and by a small number of bacterial pathogens, while the pathogens responsible for the largest field losses are fungal and biotrophic or necrotrophic in ways not represented by the model. The third concerns the temperature actually experienced by the infection court. Leaf surface temperature diverges substantially from air temperature under high radiation and low transpiration, and phyllosphere organisms have been shown to operate close to their thermal safety margins during such episodes, with surface temperatures exceeding air temperature by a margin sufficient to alter microbial survival (Pincebourde & Casas, 2019). Studies that report air temperature therefore mis-specify the variable that matters, and the mis-specification is largest precisely during the extremes of interest.

Field and natural-population evidence supplies a necessary corrective. In a naturally occurring fungal pathosystem, high temperatures reduced pathogen growth, infection and transmission, demonstrating that the thermal optimum of the pathogen can be exceeded before that of the host, with a net reduction in disease (Chen *et al*., 2024). Such outcomes are consistent with the non-monotonic thermal response functions used in bioclimatic projection and are one reason those projections indicate decreased rather than increased infection risk in parts of the low latitudes (Chaloner *et al*., 2021). Any synthesis that reports only heat-induced immune suppression selects on one side of this distribution.

Table 3 summarises the principal mechanistic findings on temperature and host immunity together with the design features that determine how much weight each can bear. The pattern across the table is informative: the studies with the strongest causal warrant, those employing genetic complementation, are also those conducted under the least realistic thermal regimes and in the fewest host species, whereas the studies conducted in crops or in the field rarely establish mechanism. This inverse relationship between causal warrant and agronomic realism is the principal methodological weakness of the host-apex literature.

5. Water Extremes and the Contested Microenvironment

5.1 Apoplast Hydration as an Actively Manipulated Variable

Water availability at the infection court has a stronger and more immediate effect on many foliar diseases than temperature does, and the mechanistic account of how it operates has changed substantially. High atmospheric humidity was long treated as a permissive condition for bacterial and fungal infection. It is now established that hemibiotrophic bacteria actively create the aqueous conditions they require. *Pseudomonas syringae* employs type III effectors to establish a water-soaked apoplast, and mutants unable to do so are compromised in virulence even under otherwise favourable conditions, which demonstrates that apoplast hydration is a pathogen-determined variable rather than an ambient one (Xin *et al*., 2016). The broader significance of leaf water status for plant-microbe interactions was subsequently articulated as a general principle rather than as a feature of one pathosystem (Aung *et al*., 2018).

Two independent studies then identified the host pathway through which this manipulation occurs. Bacterial effectors were shown to converge on abscisic acid signalling, inducing stomatal closure and thereby raising apoplast water potential to create the aqueous

environment required for bacterial multiplication, and the strategy proved to be evolutionarily conserved across distinct bacterial genera (Hu *et al*., 2022; Roussin-Léveillée *et al*., 2022). The convergence of two groups on the same mechanism through different entry points is a stronger evidential configuration than either study alone provides. A subsequent analysis established the reciprocal dynamic, showing that humidity-driven depletion of abscisic acid determines the outcome of competition between plant and pathogen for leaf water (Yasuda *et al*., 2026). Taken together, these results reposition humidity within the disease triangle. It is not an environmental parameter acting on two passive biological apices but the object of an active contest between them, and its value at the infection court is partly an outcome of the interaction rather than an input to it.

Table 3. Representative evidence on temperature effects on host immunity and resistance, with design features affecting interpretation

Finding	Host and pathogen system	Thermal treatment	Basis of causal inference	Principal interpretive limitation	Supporting references
Warm temperature suppresses salicylic acid accumulation and defence gene expression while enhancing bacterial type III secretion	*Arabidopsis thaliana* and *Pseudomonas syringae*	Constant shift from about 23 to 28 degrees Celsius	Parallel host and pathogen transcriptomic and phenotypic analysis	Constant temperature; single model pathosystem	Huot *et al*. (2017)
CBP60g promoter recruitment is the thermosensitive step; constitutive expression restores resistance at warm temperature	*Arabidopsis thaliana* and a *Brassica* crop species	Constant elevated temperature	Genetic complementation with restoration of phenotype in a second species	Growth-stage and field validation not established	Kim *et al*. (2022)
Effector-triggered outputs are differentially thermosensitive; cell death is lost while growth restriction is retained for some effector-receptor pairs	*Arabidopsis thaliana* and *Pseudomonas syringae*	Constant elevated temperature	Comparison across multiple effector-receptor pairs	Does not identify the thermosensitive molecular step	Menna *et al*. (2015)
A transient heat episode suppresses pattern-triggered immunity and bacterial resistance with recovery over days	*Arabidopsis thaliana* and *Pseudomonas syringae*	Short-duration heat treatment	Time-resolved immune marker and growth assays	Recovery dynamics not tested in crops	Janda *et al*. (2019)
Resistance gene function is temperature-dependent in both directions across crop pathosystems	Wheat, rice and tomato with rust, blast and bacterial pathogens	Contrasting constant temperature regimes	Genetic mapping and near-isogenic comparison	Thermal regimes rarely mimic field heatwaves; genotype background effects large	Aoun *et al*. (2017); Chen *et al*. (2018, 2021); Onaga *et al*. (2017); Yan *et al*. (2021)
Low temperature enhances salicylic-acid-dependent immunity through cold-signalling regulators	*Arabidopsis thaliana* and bacterial pathogens	Low-temperature exposure	Mutant analysis and direct promoter binding	Cold tail of the distribution rarely considered in climate risk framing	Kim *et al*. (2017); Li *et al*. (2020, 2024)
High temperature can reduce pathogen growth, infection and transmission in a natural pathosystem	Wild plant host with a fungal pathogen	Field-relevant temperature gradient	Replicated infection and transmission assays	Mechanism not identified; wild rather than crop system	Chen *et al*. (2024)

Note. Causal inference is graded by whether the study manipulated a candidate mechanism genetically or chemically, rather than by statistical significance. CBP60g = CALMODULIN-BINDING PROTEIN 60-LIKE g

This has a consequence for how humidity extremes should be assessed. Predictions based on ambient relative humidity implicitly assume that the pathogen accepts the ambient value. Where the pathogen can manipulate stomatal behaviour, a period of moderate ambient humidity may be sufficient for infection, while under very low humidity the same manipulation may fail. The relationship between ambient humidity extremes and disease is therefore expected to be threshold-like rather than linear, and the threshold should differ among pathogens according to whether they possess the relevant effectors. Evidence adequate to test this expectation across pathogen groups has not been assembled.

5.2 Drought as a Context-dependent Modifier

Drought presents the clearest case in which a climate extreme does not act in a single direction. A recent appraisal characterised drought as both a damper and an aggravator of plant disease, with the direction determined by pathogen lifestyle, drought timing

relative to host phenology, and whether the limiting factor for the pathogen is host susceptibility or free water (Choudhary & Senthil-Kumar, 2024). For foliar pathogens dependent on leaf wetness, water deficit is frequently suppressive. For vascular and root pathogens, and for opportunistic necrotrophs that exploit compromised host tissue, water deficit is frequently promotive. Stress combination research reached a compatible conclusion earlier and from a different direction, establishing that the response of a plant to simultaneous abiotic and biotic stress is not predictable from the responses to each stress applied separately, and that unique transcriptional and metabolic states arise under combination (Suzuki *et al*., 2014).

The signalling basis of this context dependence is partly understood. Abscisic acid, which mediates drought responses, antagonises salicylic-acid-dependent defence, so that a drought-conditioned plant is expected to be more susceptible to biotrophs and potentially less susceptible to necrotrophs whose success depends on jasmonate-associated pathways (Saijo & Loo, 2020). The antagonism is well established in controlled systems but its quantitative importance in the field, where drought intensity varies continuously and interacts with nutrient status, remains poorly constrained. Field-scale evidence indicates that the interaction between temperature and humidity, rather than either variable alone, determines crop vulnerability. A recent analysis of cotton found that the joint effect of temperature and humidity determined yield vulnerability to *Verticillium* wilt under climate change, with neither variable adequate in isolation (Zhang *et al*., 2026). This supports the general claim that single-variable climate risk assessments for disease are likely to be misleading, without yet establishing how large the error is for other pathosystems.

5.3 Soil Water, Warming and the Soilborne Compartment

The soilborne compartment responds to climate extremes on a different timescale and with a different mechanistic logic. A global-scale analysis found that the relative abundance of soilborne plant pathogens in soil communities increases with warming, and the pattern held across biomes rather than being driven by a single region (Delgado-Baquerizo *et al*., 2020). This is an ecological association rather than a demonstration of increased disease, and the distinction matters because relative abundance in bulk soil is several inferential steps removed from infection of a crop root. Experimental work has narrowed that gap. Warming was shown to intensify soil pathogen negative feedback on a temperate tree species, with the effect attributable to soil biota rather than to direct thermal effects on the host, providing a causal link between warming and pathogen-mediated host performance (Liu & He, 2021). The same authors had earlier shown that the disease triangle framework could be applied directly to test soilborne pathogen effects on community-level outcomes, which indicates that the framework retains operational utility in this compartment (Liu & He, 2019).

Soil water status modifies microbial function in ways that are not simply proportional to the intensity of the perturbation. Nitrifier communities were found to be unresponsive to elevated temperature and elevated carbon dioxide but strongly affected by drought, demonstrating that different climate drivers act on distinct functional guilds rather than on soil communities as a whole (Séneca *et al*., 2020). Extrapolating from any single driver to a general soil-microbial response to climate change is therefore unsafe, and the same caution applies to soilborne pathogen populations, which have rarely been quantified with absolute rather than relative methods under manipulated water regimes.

5.4 Extremes and the Physical Transport of Inoculum

A component of the environmental apex that receives little attention in climate-impact assessment is the physical process by which inoculum reaches susceptible tissue. Spore liberation from plant surfaces depends on the interaction between surface water and air movement, and jumping-droplet condensation combined with wind has been shown to disperse plant pathogen spores synergistically, with dispersal efficiency far exceeding that of either mechanism alone (Mukherjee *et al*., 2021). Because condensation events depend on dew-point dynamics, and because extreme heat followed by rapid nocturnal cooling alters those dynamics, climate extremes may influence epidemic development through dispersal even where they do not alter host susceptibility or pathogen growth rate. The importance of the foliar pathogen compartment for realised yield loss has been demonstrated at national scale, where phyllosphere fungal pathogens rather than soil or root communities were identified as the dominant contributor to sorghum yield loss across a wide environmental gradient (Ren *et al*., 2024). The transport pathway therefore deserves explicit representation in risk models, and its current omission is a plausible source of the discrepancy between mechanistic expectation and observed epidemic behaviour.

6. The Pathogen Apex: Thermal Performance, Range Dynamics and Toxin Risk

6.1 Non-linear Thermal Performance and the Direction of Change

Pathogen responses to temperature are unimodal, with growth and infection rising to an optimum and falling beyond it. This elementary property generates outcomes that are frequently misrepresented in general statements about climate and plant disease. Where current temperatures lie below a pathogen's optimum, warming increases risk; where they lie above it, warming reduces risk. A global assessment that coupled pathogen-specific thermal response functions to crop distributions produced exactly this spatial pattern, with increased infection risk projected at higher latitudes and decreased risk in some low-latitude regions, and with projected risk tracking projected yields (Chaloner *et al*., 2021). The correspondence with yield projections is notable because it was not imposed by the model structure, and it provides limited independent support for the thermal-matching approach.

Empirical confirmation of the descending limb exists. High temperatures reduced growth, infection and transmission of a naturally occurring fungal pathogen, with the reduction expressed in transmission as well as in infection, which compounds the epidemiological effect across generations (Chen *et al*., 2024). A long-term study of white pine blister rust found that climate change produced non-linear shifts in disease, with the relationship between climate and infection changing sign across the observed range rather than intensifying monotonically (Dudney *et al*., 2021). Such findings undermine any framing in which climate extremes

uniformly increase plant disease, and they indicate that the practically important question is the position of each pathosystem relative to the pathogen's thermal optimum, which is known for remarkably few economically significant pathogens.

Two limitations qualify the thermal-matching approach itself. Thermal response functions are typically derived from in vitro growth assays that omit the host, and the temperature at which a pathogen grows fastest in culture need not be the temperature at which it infects most efficiently, because infection depends on host immune status that is itself temperature-dependent. The dual-direction effect described for the bacterial pathosystem is the clearest illustration of this problem (Huot *et al*., 2017). The functions also use air temperature, whereas the thermal environment of the leaf surface may deviate substantially and narrows the safety margin of phyllosphere organisms during extremes (Pincebourde & Casas, 2019). Both limitations bias the approach in unknown directions, and neither has been quantified.

6.2 Range Dynamics and Emergence

The observation that crop pests and pathogens have moved poleward is among the most widely cited results in this field. Analysis of first-record data for several hundred pest and pathogen taxa indicated a mean poleward movement of approximately 2.7 kilometres per year since 1960, consistent in sign with warming (Bebber *et al*., 2013). The result is robust in direction but rests on records whose generation depends on surveillance effort, which has itself increased and is unevenly distributed geographically. Subsequent work by the same group acknowledged that economic and physical determinants shape observed distributions alongside climate, and a broader appraisal set out the conditions under which range expansion can be attributed to warming rather than to trade, land-use change or detection intensity (Bebber, 2015). The appropriate interpretation is that warming contributes to observed range shifts within a multi-causal system, not that observed shifts measure the climate effect.

Emergence events provide complementary evidence with different limitations. Wheat blast, caused by the *Triticum* pathotype of *Magnaporthe oryzae*, was restricted to South America for several decades before appearing in Bangladesh, where genomic analysis identified a South American lineage rather than a local host jump (Islam *et al*., 2016). Subsequent genomic surveillance established that a single pandemic clonal lineage underlies the intercontinental spread and identified variation relevant to fungicide sensitivity, and the disease has since been reported from southern Africa (Latorre *et al*., 2023). The pathosystem requires warm and humid conditions during heading, which links its establishment to climatic suitability, but the transfer event itself was almost certainly mediated by seed movement rather than by climate (Ceresini *et al*., 2018). Emergence therefore illustrates the interaction between climatic suitability and human transport rather than a climate effect in isolation, a conclusion consistent with broader assessments of emerging fungal and oomycete threats and of pandemic risk to food security (Fones *et al*., 2020; Ristaino *et al*., 2021). Institutional review has reached the same position, emphasising that climate alters the suitability envelope while trade and movement determine arrival (IPPC Secretariat, 2021).

Regionally specific modelling shows the same interaction at finer resolution. Coffee leaf rust risk in Colombia was reconstructed from climate reanalysis data, and the analysis indicated that the epidemic of the early 2010s coincided with conditions of intermediate rather than extreme suitability, which complicates simple attribution to a warming trend (Bebber *et al*., 2016). Attribution in this literature is genuinely difficult, and confident causal statements are rarely warranted at the level of individual epidemics.

6.3 Toxigenic Fungi and the Food-safety Dimension

A distinct category of risk arises where climate extremes alter not disease severity but toxin production. Modelling of aflatoxin B1 in maize across Europe indicated that a moderate warming scenario would convert regions currently at low risk into regions of elevated risk, with the change driven primarily by temperature and water stress during grain filling (Battilani *et al*., 2016). The ecological and genomic basis of this response has been reviewed, with oxidative stress in the fungus identified as a plausible link between host water deficit and toxin biosynthesis (Perrone *et al*., 2020). A comprehensive appraisal concluded that climate change is shifting both the geography and the spectrum of mycotoxin contamination, while noting that projections rest on a small number of modelled scenarios and on laboratory dose-response relationships (Casu *et al*., 2024).

Interaction with atmospheric composition adds further complexity. Elevated carbon dioxide altered maize chemical defence against *Fusarium verticillioides* in a manner that depended on concurrent water availability, so that the effect of one component of global change was conditional on another (Vaughan *et al*., 2014). This is the food-safety instance of the general pattern that compound conditions cannot be inferred from single-factor experiments. The practical significance is high, because toxin thresholds are regulatory rather than agronomic, and a change in contamination that has negligible effect on yield may nonetheless render a harvest unmarketable.

6.4 Pathogen Evolution under Altered Selection

Climate extremes alter the selective environment of pathogen populations as well as their immediate performance. Temperature increase has been associated with the emergence of more aggressive *Verticillium* strains in species of *Medicago*, indicating that thermal conditions can select on virulence rather than merely modulate its expression (Sbeiti *et al*., 2023). The evidence base for this proposition is thin relative to its importance. Most pathogen thermal biology is conducted on a small number of isolates, and standing variation in thermal tolerance within field populations is rarely quantified, even though this variation determines the rate at which a population can track a changing thermal environment. Vector-borne pathogens introduce an additional layer, since warming alters vector development, abundance and feeding behaviour, and virus epidemiology consequently responds to climate through host, vector and virus simultaneously (Trębicki, 2020).

Table 4 sets out the principal instances in which the literature reports contradictory climate-disease outcomes and evaluates the candidate explanations for each. The contradictions are not, for the most part, evidence of error. They arise from differences in the position of the system relative to thermal optima, from the confounding of temperature with humidity, from differences between relative and absolute measures of pathogen abundance, and from the distinction between controlled-environment and field observation. Recognising this is a prerequisite for interpreting the microbiome evidence examined next, where the same sources of apparent contradiction operate with greater force because community-level measurements are compositional and because experimental control is harder to achieve.

Table 4. Apparently conflicting findings on climate extremes and plant disease, with candidate explanations

Apparent conflict	Evidence on one side	Evidence on the other side	Most plausible explanation	Residual uncertainty	Supporting references
Warming increases versus decreases infection risk	Projected increases in infection risk at higher latitudes tracking crop yields	Reduced pathogen growth, infection and transmission at high temperature; sign reversal in a long-term rust study	Position of the system relative to the pathogen thermal optimum; unimodal response functions	Thermal optima are unknown for most economically important pathogens	Chaloner *et al*. (2021); Chen *et al*. (2024); Dudney *et al*. (2021)
Heat suppresses versus does not suppress resistance	Warm temperature suppresses salicylic acid immunity; transient heat suppresses pattern-triggered responses	Sr21, Lr13 and Xa7 confer resistance specifically at elevated temperature	Thermosensitivity is a property of individual resistance mechanisms rather than of immunity as a category	Whether thermoresilient genes remain effective under fluctuating field heatwaves	Chen *et al*. (2018, 2021); Huot *et al*. (2017); Janda *et al*. (2019); Yan *et al*. (2021)
Drought increases versus decreases disease	Drought predisposes hosts to vascular and opportunistic pathogens	Drought suppresses foliar pathogens dependent on leaf wetness	Pathogen lifestyle and the identity of the limiting factor; timing relative to host phenology	Quantitative importance of hormone antagonism under field drought gradients	Choudhary & Senthil-Kumar (2024); Saijo & Loo (2020); Suzuki *et al*. (2014)
Soil warming increases pathogen load versus has no demonstrated disease effect	Relative abundance of soilborne pathogens increases with warming globally; warming intensifies pathogen negative feedback	Nitrifier and other functional guilds show driver-specific rather than general responses	Relative abundance in bulk soil is several inferential steps from root infection; guild-specific responses	Absolute quantification of pathogen inoculum under manipulated warming is scarce	Delgado-Baquerizo *et al*. (2020); Liu & He (2021); Séneca *et al*. (2020)
Range shifts attributed to warming versus to human activity	Mean poleward movement of pest and pathogen first records since 1960	Economic and physical determinants and surveillance effort shape observed distributions; wheat blast transfer was consistent with seed movement	Climate alters suitability while trade and movement determine arrival; both contribute	Relative contribution of each process has not been partitioned quantitatively	Bebber (2015); Bebber *et al*. (2013); Ceresini *et al*. (2018); Islam *et al*. (2016)

Note. Residual uncertainty identifies what would need to be measured to resolve the conflict rather than restating the disagreement

7. Microbiome Disruption under Climate Extremes

7.1 Conserved Directional Change in Root Communities under Drought

Drought produces changes in root-associated bacterial communities that are consistent enough across hosts and soils to be described as a signature. Comparative work across grass species established that drought shifts community composition in a direction shared among hosts while host identity continues to exert selection, indicating that the drought effect is superimposed on rather than replacing host filtering (Naylor *et al*., 2017). Detailed analysis in *Sorghum bicolor* showed that drought delays the developmental trajectory of the root microbiome and enriches monoderm lineages, principally actinobacterial taxa with thick cell walls, relative to diderm lineages (Xu *et al*., 2018). The consistency of this enrichment across systems was subsequently synthesised as a conserved bacterial response to water limitation (Xu & Coleman-Derr, 2019), and a recent cross-study analysis reported a conserved bacterial signature characterising plant microbiome responses to drought across independent datasets (Cosma & Abeel, 2026).

Two observations give this pattern mechanistic content rather than leaving it as description. Genome-resolved metagenomics identified iron metabolism as a discriminating functional axis in drought-affected rhizosphere communities, linking community change to a specific and physiologically plausible resource constraint (Xu *et al*., 2021). Metabolite-level work has since proposed

that a limited set of hub metabolites at the root-microbiome interface mediates drought-responsive recruitment, which would provide a tractable point of intervention if confirmed across systems (Pantigoso *et al*., 2025). Persistence is the second notable feature. Prolonged drought in rice imparted compositional changes that remained after rewatering, establishing that the community carries a legacy of the extreme rather than returning to its prior state (Santos-Medellín *et al*., 2021).

Interpretation requires caution on three counts. Amplicon sequencing reports relative abundance, so enrichment of desiccation-tolerant lineages may reflect differential survival of other taxa rather than active recruitment, and few studies have applied absolute quantification to distinguish these possibilities. Monoderm enrichment is consistent with passive selection by desiccation, and the alternative interpretation that the host actively recruits these taxa requires evidence of host benefit that has not been supplied in most studies. The magnitude of the drought effect is also context-dependent: in wheat, soil properties, agricultural management and cultivar interacted to determine the structural and functional response of the rhizosphere to drought, which means that any single-site result has limited generality (Breitkreuz *et al*., 2021). Broad comparative work on root microbiome assembly across angiosperm species similarly showed that host phylogeny structures communities in ways that constrain how far a drought signature can be treated as universal (Fitzpatrick *et al*., 2018).

7.2 Warming, thermal Margins and the Aerial Compartment

The phyllosphere responds to thermal extremes on a shorter timescale and with less buffering than the rhizosphere. Leaf surface temperatures exceed air temperature under high radiation, and phyllosphere organisms have been shown to operate within narrow thermal safety margins during such episodes, so that a moderate rise in air temperature can translate into lethal conditions at the surface (Pincebourde & Casas, 2019). The implication is that heatwaves may remove protective resident taxa before they impair host immunity, opening a window of susceptibility that neither host-focused nor pathogen-focused analysis would detect. The aerial compartment has been argued to face distinctive pressures under anthropogenic change for precisely this reason (Perreault & Laforest-Lapointe, 2022). Direct experimental tests of whether heatwave-induced phyllosphere mortality increases subsequent disease are, to date, absent from the literature, and this is among the clearest empirical gaps identified by the present synthesis.

In soil, long-term warming produced declines in microbial diversity accompanied by respiration increases exceeding predictions, with communities adapting in ways that altered function rather than merely composition (Nottingham *et al*., 2022). The pathogen-specific counterpart is the global increase in the relative abundance of soilborne plant pathogens with warming (Delgado-Baquerizo *et al*., 2020). These results are consistent with a reduction in the biotic buffering capacity of soil communities under sustained warming, but neither establishes that crop disease increases as a consequence, and the inferential distance between a shift in community composition and an epidemic in a managed field remains substantial.

7.3 Host Control of Community Composition and the Dysbiosis Concept

The strongest evidence that microbial community state matters causally comes from host genetics. Disruption of a plant genetic network involving vesicle trafficking and immune components produced leaf communities that were abnormal in composition and abundance and were accompanied by tissue damage, and reconstitution experiments indicated that the abnormal community rather than the host mutation was the proximate cause of the damage (Chen *et al*., 2020). The respiratory burst oxidase homologue RBOHD was independently shown to be required for microbiota homeostasis in leaves, which identifies reactive oxygen production as a control point for community composition rather than only for pathogen suppression (Pfeilmeier *et al*., 2021). Gnotobiotic work then demonstrated the reciprocal relationship, showing that a normal microbiota is required for proper immunocompetence, so that community state and immune state are mutually determining rather than sequentially related (Paasch *et al*., 2023). Protection by the resident community against a bacterial pathogen has also been demonstrated directly in a synthetic assemblage (Vogel *et al*., 2021), and community assembly in the phyllosphere has been shown to depend on priority effects and on a small number of strains with disproportionate influence (Carlström *et al*., 2019).

These results justify the use of the term dysbiosis in plant systems, but they also expose its imprecision. Dysbiosis describes a departure from a reference state, and no consensus reference state exists for any crop microbiome. An influential treatment argued that dysbiotic communities should be expected to diverge in many directions rather than converging on a single abnormal configuration, following the principle that healthy states resemble one another while unhealthy states differ individually (Arnault *et al*., 2023). If this holds, then the search for a diagnostic dysbiotic signature is misdirected, and variance rather than mean composition is the informative statistic. Most climate-microbiome studies report central tendency and treat dispersion as noise, so the evidence needed to evaluate this proposition has largely not been collected even though it exists in the underlying datasets.

7.4 Whether Community Disruption Causes Altered Disease Outcomes

The causal chain from climate extreme to community disruption to altered disease has not been demonstrated end to end in any crop system, and assembling it from partial evidence requires care. The link from community composition to disease outcome is the best established of the two segments. Disease-suppressive soils were shown to owe their suppressiveness to specific bacterial groups and their biosynthetic capacity (Mendes *et al*., 2011), and pathogen challenge was subsequently shown to activate disease-suppressive functions within the endophytic root microbiome, establishing that suppression is an inducible community property rather than a fixed soil attribute (Carrión *et al*., 2019). Infection recruits protective consortia whose benefit can be transferred (Berendsen *et al*., 2018), rhizosphere community structure differs between wilt-resistant and wilt-susceptible outcomes in tomato (Kwak *et al*., 2018), and experimental disruption of particular bacterial groups in the tomato rhizosphere increased the incidence of bacterial wilt, which is among the few experiments that manipulate the community and measure disease (Lee *et al*., 2021). Community properties rather than individual taxa also matter, since trophic network architecture and functional diversity have been shown to determine pathogen

invasion success (Hu *et al*., 2020; Wei *et al*., 2015). Infection in turn restructures the community and leaves a soil-borne legacy mediated by altered root exudation (Gao *et al*., 2021; Yuan *et al*., 2018).

The link from climate extreme to community disruption is equally well established, as set out above. The weak segment is the junction between them. Very few studies impose a climate extreme, characterise the resulting community and then challenge with a pathogen under the same conditions, which is the design required to attribute a disease outcome to climate-induced community change rather than to direct climatic effects on host or pathogen. Two recent studies approach this standard. A host genotype-specific rhizosphere fungus was shown to enhance drought resistance in wheat, with the benefit dependent on host genotype, which establishes that stress-associated microbial recruitment can be functionally consequential rather than incidental (Yue *et al*., 2024). More directly, nucleotides enriched under heat stress were shown to recruit beneficial rhizosphere microorganisms that protected plants from both heat and root rot, which links a thermal extreme, a defined chemical signal, a community change and a disease outcome within a single experimental system (Liu *et al*., 2025). Related work demonstrated that a cry-for-help response can be triggered to assemble a disease-suppressing rhizosphere community, establishing the general mechanism if not its operation under field extremes (Liu *et al*., 2024).

The confidence that can reasonably be placed in the microbiome segment of the framework is therefore asymmetric. That climate extremes restructure plant-associated communities is well supported by convergent evidence across hosts, compartments and methods. That community state influences disease outcome is well supported by manipulative experiments, although chiefly in soilborne bacterial pathosystems under controlled conditions. That climate-induced community disruption is a quantitatively important cause of altered disease in the field remains a plausible hypothesis supported by two or three studies rather than an established result, and it is currently invoked with greater confidence than the evidence sustains. This distinction has practical consequences, since microbiome-based crop protection products are being developed on the assumption that restoring a disrupted community will restore disease resistance under stress, an assumption examined in the following sections.

8. Compound Extremes and the Failure of Additive Reasoning

Climate extremes rarely arrive singly. Drought and heat co-occur more often than independence would predict, their joint frequency is increasing faster than that of either component, and their combined occurrence is projected to respond steeply to additional warming (Zhang *et al*., 2022; Zscheischler *et al*., 2018, 2020). Plant pathology has, with few exceptions, continued to study one factor at a time. This is a substantive problem rather than a matter of experimental convenience, because the physiological response of a plant to combined stresses is not the sum of its responses to each stress applied separately, and unique transcriptional and metabolic states arise under combination that are not predictable from single-factor data (Suzuki *et al*., 2014).

The interaction between water status and temperature is the most consequential instance for plant disease. Both variables act on stomatal behaviour, both act on apoplast water potential, and both are manipulated by bacterial effectors operating through abscisic acid signalling (Hu *et al*., 2022; Roussin-Léveillée *et al*., 2022; Yasuda *et al*., 2026). A pathogen strategy that raises apoplast water content should be favoured under warm and humid conditions and should fail under warm and dry conditions, so the same temperature increase would be expected to have opposite effects depending on the accompanying humidity. Solanaceous hosts under combined high temperature and high humidity switch to cytokinin-mediated defence rather than losing defence, which is consistent with a qualitatively distinct response to the combination (Yang *et al*., 2022). Field-scale confirmation that the joint effect of temperature and humidity, and not either variable alone, determines vulnerability has been obtained for *Verticillium* wilt in cotton (Zhang *et al*., 2026). The general lesson is that single-variable climate-disease relationships derived from either controlled experiments or correlative analysis should be treated as conditional on the unmeasured second variable.

Rising atmospheric carbon dioxide interacts with both. Wheat plants and pathogens acclimatised to elevated carbon dioxide showed increased rather than decreased disease severity, which indicates that the fertilisation effect does not translate into improved defence (Váry *et al*., 2015). The underlying mechanism involves hormone signalling, since disease susceptibility of tomato under elevated carbon dioxide varied with antagonism between salicylate and jasmonate pathways, producing pathogen-specific rather than uniform outcomes (Zhang *et al*., 2015). A recent appraisal concluded that the immunological risks of elevated carbon dioxide may offset its productivity benefits for some pathosystems, while emphasising that the evidence base remains small and dominated by short-term enclosure studies (Smith & Luna, 2023). The interaction with water availability has been demonstrated directly, since the effect of elevated carbon dioxide on maize defence against *Fusarium verticillioides* depended on concurrent drought (Vaughan *et al*., 2014). Vector-borne viruses add a further route, as warming alters vector development, abundance and feeding behaviour alongside its effects on host and virus (Trębicki, 2020).

The methodological implication is specific and actionable. Factorial designs crossing temperature with water status, applied as realistic episodes rather than as sustained shifts, and extended to include a pathogen challenge, are the minimum required to characterise disease risk under compound extremes. Such designs exist in the abiotic stress literature and in a small number of pathology studies, but they are not standard, and their absence is the single largest obstacle to converting mechanistic understanding into quantitative risk prediction.

9. Implications for Crop Protection

9.1 Thermoresilient Resistance and Its Constraints

The identification of a thermosensitive step in salicylic acid biosynthesis and the restoration of warm-temperature resistance by constitutive expression of CBP60g provide the clearest proof of concept that immune thermosensitivity is engineerable, and the

reproduction of the effect in a crop species strengthens the case considerably (Kim *et al*., 2022). Prospects and constraints for translating this and related findings have been set out in detail, with the recurring conclusion that the number of validated targets remains small relative to the diversity of pathosystems requiring protection (Castroverde *et al*., 2025).

Natural variation offers a complementary and more immediately deployable route. Resistance genes that function specifically at elevated temperature exist in the major cereals and have been characterised at the molecular level, including Sr21 against stem rust, Lr13 against leaf rust and the executor gene Xa7 against bacterial blight (Chen *et al*., 2018, 2021; Yan *et al*., 2021; Zhao & Zhang, 2021). Deploying them is not straightforward. Lr13 is associated with hybrid necrosis, which constrains its use in crossing programmes (Yan *et al*., 2021). Thermosensitivity is background-dependent, as shown for Pi54 in rice, so a gene characterised as thermoresilient in one genetic background may not behave identically in another (Onaga *et al*., 2017). The underlying immunity-growth trade-off means that constitutive enhancement of defence risks yield penalties, and the SIZ1 result indicates that the same regulatory machinery serves both processes (Hammoudi *et al*., 2018). The practical recommendation supported by this evidence is modest but clear: resistance phenotyping should be conducted under at least two thermal regimes, one of which should simulate a realistic heatwave, so that thermally conditional resistance is detected before rather than after commercial deployment.

9.2 Microbiome-based Interventions and the Field-efficacy Gap

The proposition that plant-associated microorganisms can be managed to improve stress and disease resilience is supported by strong mechanistic evidence and weak field evidence, and the gap between the two is the central problem in this area. Mechanistically, microbiome-mediated stress resistance is well documented, synthetic communities have rescued hosts from root disease by activating induced systemic resistance, and a desert-derived synthetic community conferred salt stress resilience in the presence of a resident soil microbiome rather than only under gnotobiotic conditions (Li *et al*., 2021; Liu *et al*., 2020; Schmitz *et al*., 2022). The conceptual case for harnessing rhizosphere communities for drought-resilient production has been argued in detail, and strategies for precision management across agroecosystems have been mapped (de Vries *et al*., 2020; French *et al*., 2021).

The obstacles are structural rather than incidental. Introduced organisms must establish in the presence of an existing community that resists invasion, and invasion resistance is itself a function of community network architecture and functional diversity, which vary among fields (Hu *et al*., 2020; Wei *et al*., 2015). The conflicts inherent in developing soil microbial inoculants, including the tension between broad-spectrum products and site-specific efficacy, were set out explicitly and have not been resolved (Kaminsky *et al*., 2019). Colonisation timing matters, since priority effects govern phyllosphere assembly and early seedling colonisation shapes subsequent community trajectory, which suggests that seed and seedling application windows are more promising than later intervention (Carlström *et al*., 2019; Joubert *et al*., 2025). Host genetic control of the associated community offers an alternative route that avoids the establishment problem entirely, by selecting plant genotypes that recruit beneficial communities rather than applying organisms directly, and this approach has been reviewed as a breeding target (Escudero-Martinez & Bulgarelli, 2023). Its feasibility is supported by the demonstration that drought-protective fungal recruitment in wheat is host genotype-specific (Yue *et al*., 2024).

A specific and under-recognised deficiency is that biological products are seldom evaluated under the stress conditions that motivate their use. Efficacy trials are typically conducted under conditions favourable to the introduced organism, whereas the intended application is during or after a heatwave or drought, when the introduced organism is subject to the same thermal and water constraints as the resident community. The phyllosphere thermal safety margin result indicates that these constraints may be severe (Pincebourde & Casas, 2019). Evidence that beneficial recruitment can be driven by defined host-derived signals under heat stress, and can protect against a soilborne disease under those conditions, is therefore particularly valuable, and suggests that signal-based approaches may prove more robust than organism-based approaches under extremes (Liu *et al*., 2025; Pantigoso *et al*., 2025). Reviews of soil and phytomicrobiome management for disease suppression under climate change reach compatible conclusions while noting the same evidential shortfall (Chen *et al*., 2023).

9.3 Forecasting, Surveillance and Integrated Management

Disease forecasting systems were developed for stationary climates and typically relate infection risk to weather variables through relationships calibrated on historical data. Extremes push these relationships outside their calibration range, and the non-linear and sign-changing responses documented above indicate that extrapolation is unsafe. Progress in modelling climate effects on arable crop disease has been reviewed, with the persistent difficulty identified as the coupling of pathogen process models to crop and management models at compatible resolution (Newbery *et al*., 2016). The application of machine learning to large heterogeneous datasets has been proposed as a route to anticipating pathogen emergence, with the important caveat that such methods inherit the geographical and taxonomic biases of the surveillance data on which they are trained (Garrett *et al*., 2022). Priorities for the discipline have been articulated with surveillance capacity and data integration prominent among them (Wang *et al*., 2024).

Integrated crop management provides the framework within which these components are deployed, and its adaptation to climate-smart production for both low-input and high-input systems has been set out in detail (Richard *et al*., 2022). Two elements deserve particular emphasis under extremes. Sowing date and cultivar maturity determine whether the susceptible growth stage coincides with the period of highest climatic risk, and adjusting this coincidence is among the few interventions available at low cost and short notice. Monitoring of toxigenic fungi requires separate treatment from disease monitoring, because the regulatory threshold for mycotoxin contamination may be exceeded in crops showing no visible disease, and projected geographical shifts in aflatoxin risk imply that surveillance networks will need to extend into regions with no history of contamination (Battilani *et al*., 2016; Casu *et al*., 2024; Perrone *et al*., 2020). Dispersal processes sensitive to condensation and wind dynamics represent a further component that

current forecasting largely omits (Mukherjee *et al*., 2021). A recent overview of dynamics, risks and mitigation reaches similar conclusions about the need to integrate rather than merely accumulate these elements (Kumar & Mukhopadhyay, 2025).

10. Future Directions

The priorities set out here follow from the specific evidential deficits identified in the preceding sections rather than from a general call for more research. They are ordered by the ratio of expected information gain to experimental cost, and the first three are achievable within the current funding and infrastructure landscape.

The most immediate priority is the replacement of constant-temperature treatments with realistic episodic regimes in mechanistic pathology. Almost every molecular result on temperature and immunity derives from a sustained shift to a single elevated temperature, whereas the agronomically relevant exposure is a heatwave of three to seven days superimposed on a diurnal cycle and followed by recovery. The one study that applied a transient treatment found suppression of pattern-triggered immunity with recovery over days, a result that cannot be obtained from constant-temperature designs and that changes the interpretation of the entire literature (Janda *et al*., 2019). The required design is a controlled-environment experiment applying diurnally fluctuating heatwave profiles of defined amplitude, duration and timing relative to host growth stage, with pathogen challenge imposed during, immediately after and one week after the episode. Outcome measures should include defence hormone concentration, immune marker transcript abundance and pathogen population size, so that the temporal offset between immune recovery and pathogen establishment can be quantified. This work would establish whether the window of susceptibility created by a heatwave is short enough to be managed by timing of protective interventions, which is directly actionable.

The second priority is closure of the causal chain linking climate extremes to microbial community change to disease outcome. The two segments of this chain are separately well supported, and the junction is not (Delgado-Baquerizo *et al*., 2020; Lee *et al*., 2021; Santos-Medellín *et al*., 2021). The design required is a factorial experiment in which a climate extreme is imposed, the resulting community is characterised with absolute rather than only relative quantification, and pathogen challenge is applied both to the perturbed community and to a community experimentally restored toward the unperturbed state. Only the restoration arm permits attribution of the disease outcome to community change rather than to direct climatic effects on host or pathogen. The experiment should be conducted in at least two soils and two host genotypes, because soil and cultivar interact with drought in shaping rhizosphere responses (Breitkreuz *et al*., 2021). Absolute quantification is essential because compositional data cannot distinguish enrichment from differential loss, an ambiguity that currently affects the interpretation of the conserved drought signature (Cosma & Abeel, 2026; Xu & Coleman-Derr, 2019).

The third priority is the routine evaluation of resistance and of biological products under stress rather than under optimal conditions. Resistance genes with opposite thermal behaviour are known in the same crop species, and thermal conditionality is background-dependent (Chen *et al*., 2018; Onaga *et al*., 2017; Yan *et al*., 2021). Biological control and microbiome products are similarly evaluated under conditions favourable to the introduced organism, although their intended use is during stress episodes when the organism is itself constrained (Kaminsky *et al*., 2019; Pincebourde & Casas, 2019). The change required is procedural rather than conceptual: phenotyping and efficacy protocols should include a heatwave or drought arm as standard, and reporting should state the thermal and water regime under which efficacy was established. The cost is modest relative to the cost of deploying a product or cultivar that fails under the conditions it was intended to address.

The fourth priority concerns spatial and taxonomic representativeness. Mechanistic understanding is concentrated in *Arabidopsis thaliana* and a small number of bacterial pathosystems, in temperate settings, whereas the largest losses and the highest exposure to extremes occur in tropical and subtropical smallholder systems (Savary *et al*., 2019). Surveillance data inherit the same bias, which propagates into machine-learning approaches trained on them (Garrett *et al*., 2022). Targeted investment in mechanistic and epidemiological work on tropical pathosystems, including those of sorghum, cassava, cowpea and banana, would address a deficit that no amount of additional temperate-zone research can remedy. This is a longer-term priority because it requires capacity as well as funding.

The fifth priority is methodological integration between mechanistic and projective approaches. Bioclimatic projection and molecular pathology currently operate without exchange, and neither can validate the other (Chaloner *et al*., 2021; Newbery *et al*., 2016). A tractable intermediate step is the derivation of host-inclusive thermal response functions, in which the temperature dependence of infection is measured in planta across a thermal gradient rather than inferred from in vitro pathogen growth. Such functions would be directly usable in projection models and would incorporate the host immune thermosensitivity that current functions omit. For a small number of high-value pathosystems this is achievable within a single funding cycle and would provide the first empirical test of how much error the current approximation introduces.

A sixth priority, longer in horizon, concerns pathogen evolution. Standing variation in thermal tolerance within field pathogen populations is almost unquantified, yet it determines the rate at which populations can track a shifting thermal environment and therefore the durability of any thermal-mismatch benefit projected for low-latitude regions (Sbeiti *et al*., 2023). Population-scale thermal phenotyping of field isolates, combined with genomic surveillance of the kind applied to wheat blast, would convert a currently unexamined assumption into a measurable parameter (Latorre *et al*., 2023).

Table 5 sets out these priorities with the specific deficits they address and the outcomes that would signal progress. The sequencing matters as much as the content: priorities one to three are procedural changes to existing research practice and could be adopted immediately, whereas priorities four to six require investment in new capacity and will not yield results within a single project cycle.

Table 5. Prioritised research needs, required designs and indicators of progress

Priority	Unresolved problem	Why current evidence is inadequate	Required design and scale	Principal outcome measures	Indicator of progress	Supporting references
1. Episodic rather than constant thermal treatments	Whether heatwave-induced immune suppression creates a manageable window of susceptibility	Nearly all mechanistic work uses sustained temperature shifts unlike field exposure	Controlled environment with diurnally fluctuating heatwave profiles; challenge during, after and one week after the episode	Defence hormone concentration; immune marker transcripts; pathogen population size	Quantified duration of the susceptibility window in at least two crop pathosystems	Janda *et al*. (2019); Kim *et al*. (2022)
2. Closing the climate to microbiome to disease causal chain	Whether climate-induced community change causes altered disease or merely accompanies it	The two chain segments are supported separately; the junction is supported by very few studies	Factorial climate stress by community restoration by pathogen challenge; at least two soils and two genotypes; absolute quantification	Absolute taxon abundance; disease incidence and severity; host immune markers	Demonstration that community restoration recovers resistance under an imposed extreme	Breitkreuz *et al*. (2021); Lee *et al*. (2021); Santos-Medellín *et al*. (2021)
3. Evaluation of resistance and biologicals under stress	Whether resistance genes and biological products retain efficacy under heatwave or drought	Phenotyping and efficacy testing are conducted under conditions favourable to host or to introduced organism	Addition of a standard stress arm to existing phenotyping and efficacy protocols; multi-site	Disease severity under stress and non-stress arms; survival of introduced organisms	Routine reporting of the thermal and water regime under which efficacy was established	Kaminsky *et al*. (2019); Onaga *et al*. (2017); Yan *et al*. (2021)
4. Tropical and subtropical pathosystem coverage	Whether temperate mechanistic findings transfer to the systems bearing the greatest losses	Mechanistic and surveillance data are concentrated in temperate regions and in few host species	Long-term mechanistic and epidemiological programmes in sorghum, cassava, cowpea and banana systems	Pathosystem-specific thermal and water response; yield loss attribution	Mechanistic datasets of comparable depth for at least three tropical pathosystems	Garrett *et al*. (2022); Savary *et al*. (2019)
5. Host-inclusive thermal response functions	How much error is introduced by deriving pathogen thermal functions without the host	Projection models use in vitro pathogen growth optima; host immune thermosensitivity is omitted	In planta infection assays across a thermal gradient for selected high-value pathosystems	Infection efficiency and lesion development as functions of plant temperature	Direct comparison of projections using in vitro and in planta functions	Chaloner *et al*. (2021); Huot *et al*. (2017); Newbery *et al*. (2016)
6. Thermal tolerance variation in field pathogen populations	How rapidly pathogen populations can track a shifting thermal environment	Thermal biology rests on few isolates; standing variation is unquantified	Population-scale thermal phenotyping of field isolates combined with genomic surveillance	Distribution of thermal optima within populations; allele frequency change over seasons	Population-level thermal parameters available for at least one pandemic lineage	Latorre *et al*. (2023); Sbeiti *et al*. (2023)

Note. Priorities 1 to 3 are procedural changes to existing research practice. Priorities 4 to 6 require new capacity and operate on longer timescales

11. Conclusions

Climate extremes have altered the plant disease triangle in ways that are mechanistically specific rather than generally intensifying, and the evidence supports a revision of how the framework is used rather than a replacement of its structure. Moderately elevated temperature suppresses salicylic-acid-dependent immunity through an identified transcriptional node, and the restoration of resistance by genetic manipulation of that node, reproduced in a crop species, establishes causation rather than association. Water status at the infection court is not an ambient condition but an actively contested resource, since bacterial effectors manipulate host

abscisic acid signalling to hydrate the leaf apoplast and the host depletes that hormone in response. These two findings jointly undermine the assumption that the environmental apex acts on passive biological apices, and they explain why single-variable climate-disease relationships have proved unreliable.

The direction of change is not uniform. Resistance genes exist in wheat and rice whose effectiveness increases rather than decreases at elevated temperature, low temperature enhances salicylic-acid-dependent immunity through cold-signalling regulators, and high temperature reduces infection and transmission in pathosystems already near their thermal optimum. Statements that climate extremes will increase plant disease are therefore not supported as a general proposition, and the practically decisive variable is the position of each pathosystem relative to the thermal optima of its components, which is unknown for most economically important pathogens.

Climate extremes restructure plant-associated microbial communities in partially conserved directions, and community state influences disease outcome. Both propositions are well supported. The proposition that links them, namely that climate-induced community disruption is a quantitatively important cause of altered disease under field conditions, is supported by a small number of studies and is presently asserted with more confidence than the evidence sustains. The appropriate treatment of the microbiota within the framework follows from this assessment: resident communities are best represented as modifiers operating within the host, pathogen and environmental apices rather than as a separable fourth apex, because they are simultaneously part of the host phenotype, competitors of the pathogen and a component of the pathogen's environment, and because adding a vertex for every relevant factor reduces the framework's capacity to be falsified.

The central weakness of the field is structural and consistent across its subdisciplines. The studies with the strongest causal warrant employ the least realistic environmental regimes and the narrowest range of host species, while the studies conducted in crops and in the field rarely establish mechanism. Bioclimatic projection and molecular pathology have developed without exchange, and neither can currently validate the other. Resolving this does not require a new theoretical framework. It requires episodic rather than sustained environmental treatments, factorial rather than single-factor designs, absolute rather than relative quantification of microbial communities, evaluation of resistance and biological products under the stress conditions they are intended to address, and investment in the tropical pathosystems where losses are greatest and evidence is thinnest. The disease triangle remains a serviceable organising abstraction; what has changed is that each of its apices must now be treated as internally composite, environmentally contingent and capable of changing on the timescale of a single crop.

12. Limitations

The principal limitation is intrinsic to the review design. A critical narrative synthesis involves judgement in the selection, weighting and interpretation of sources, and although the search concepts, information sources, eligibility criteria and appraisal approach have been stated explicitly, selection and interpretive bias cannot be excluded. Different reviewers applying the same criteria to the same literature could reasonably reach different conclusions about the relative weight of mechanistic and ecological evidence, and the position taken here on the status of the microbiota within the framework is an interpretation rather than a finding.

Access to the literature was incomplete. Subscription bibliographic databases were not available, so retrieval relied on open scholarly indexes, citation searching and institutional sources. Coverage of these indexes is extensive but not identical to that of subscription databases, and material indexed only in the latter may have been missed. The restriction to English-language publications introduces a further bias whose direction is predictable and unfavourable for this particular topic, because locally published work on tropical and subtropical pathosystems, precisely the systems identified here as under-represented, is disproportionately likely to appear in other languages or in regionally indexed outlets.

The evidence base itself constrains what could be concluded. Study designs are heterogeneous to a degree that precludes quantitative synthesis, since molecular, community-ecological and epidemiological studies report incommensurable outcomes on incompatible timescales, and no common effect measure exists. No pooled estimate of the magnitude of any climate-disease relationship is therefore offered, and comparisons among studies are qualitative. Geographical representation is markedly uneven, with mechanistic work concentrated in temperate research systems, and this unevenness limits the generality of every mechanistic conclusion drawn.

Several further constraints apply. Much of the microbiome evidence is observational and compositional, and the distinction between active recruitment and differential survival frequently cannot be resolved from the published data. Long-term evidence is scarce across the field, so the persistence of climate-induced changes beyond one or two seasons is largely unknown, and the few available legacy studies concern single systems. Publication bias is likely to operate in the direction of reporting significant climate effects on disease, and negative or null results, particularly those showing no effect of an imposed extreme, are probably under-represented. The field is also developing rapidly, and conclusions concerning the microbiome segment in particular should be expected to require revision as manipulative studies accumulate.

Declaration of AI Use

This manuscript was prepared through the intellectual contributions of the author(s) to the study design, data analysis, interpretation of results, and scholarly content. Grammarly and ChatGPT were used solely to assist with grammatical refinement and reference formatting during manuscript revision. The author(s) accept full responsibility for the accuracy, integrity, and final content of the manuscript.

Competing Interests

Authors have declared that no competing interests exist.

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