*Ecology Letters*

WILEY

ECOLOGY LETTERS

SYNTHESIS

# Impacts of Weather Anomalies and Climate on Plant Disease

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**Received:** 26 March 2024 | **Revised:** 19 November 2024 | **Accepted:** 20 November 2024

**Editor:** Laura Figueroa

**Funding:** This work was supported by National Institutes of Health (R01AI102918, R01AI168097, R35GM133439) and National Science Foundation (DEB-2011147).

## ABSTRACT

Predicting the effects of climate change on plant disease is critical for protecting ecosystems and food production. Here, we show how disease pressure responds to short-term weather, historical climate and weather anomalies by compiling a global database (4339 plant–disease populations) of disease prevalence in both agricultural and wild plant systems. We hypothesised that weather and climate would play a larger role in disease in wild versus agricultural plant populations, which the results supported. In wild systems, disease prevalence peaked when the temperature was 2.7°C warmer than the historical average for the same time of year. We also found evidence of a negative interactive effect between weather anomalies and climate in wild systems, consistent with the idea that climate maladaptation can be an important driver of disease outbreaks. Temperature and precipitation had relatively little explanatory power in agricultural systems, though we observed a significant positive effect of current temperature. These results indicate that disease pressure in wild plants is sensitive to nonlinear effects of weather, weather anomalies and their interaction with historical climate. In contrast, warmer temperatures drove risks for agricultural plant disease outbreaks within the temperature range examined regardless of historical climate, suggesting vulnerability to ongoing climate change.

## 1 | Introduction

Infectious disease outbreaks cause massive losses in crop yields (Savary et al. 2019), threaten food security (Ristaino et al. 2021) and imperil wild plants (Dudney et al. 2021). Plant–disease systems are highly sensitive to environmental effects (Garrett et al. 2006; Kocmánková et al. 2009), and as the world experiences rapid change, understanding how plant–disease systems will respond to novel weather caused by anthropogenic climate forcing is critical (Burdon and Zhan 2020; Miller et al. 2022).

Multiple aspects of the climate (i.e., temperature, rainfall and drought and $CO_2$) can each affect plant–disease systems in various ways, including impacts on plant and pathogen distributions, pathogen transmission, vector biology, host resistance, pathogen virulence and host–pathogen evolutionary processes

(reviewed in Coakley, Scherm, and Chakraborty 1999; Garrett et al. 2006; Singh et al. 2023). Despite increasing research, it still remains difficult to predict how future climate change will impact these complex systems (Burdon and Zhan 2020; Elad and Pertot 2014; Jiranek et al. 2023; Laine 2023; Singh et al. 2023). Additionally, beyond the effects of the current climate that hosts and pathogens experience, it is possible that changes in disease pressure will reflect both plant and pathogen responses to ongoing climate change and the legacy of historical conditions to which both organisms are adapted. To better anticipate and mitigate climate change–plant disease effects and their implications for global food security, plant biodiversity and conservation and ecosystem management, we can examine how current weather and its change relative to historical climate have together shaped plant disease around the world.

Temperature is well known to affect various biological rates of both hosts and disease-causing agents (i.e., pathogens, parasites and pests), often leading to large impacts on the overall levels of disease in a system (Harvell et al. 2002). Organism thermal performance curves (TPCs) are typically unimodal with an intermediate optimal temperature before decreasing sharply approaching a critical thermal maximum (Angilletta 2009; Dell, Pawar, and Savage 2011). Whether warming will lead to increased or decreased disease in a system thus depends on how a change in temperature affects key host rates (e.g., growth rate and defence pathways) relative to its effects on parasite rates (e.g., transmission rate and replication rate) (Molnár et al. 2013). Beyond the effects of temperature per se, temperature anomalies—deviations from historical averages—may distinctly affect plant disease by taking organisms away from the temperatures to which they are adapted. Like other thermal responses, impacts of anomalies may also be nonlinear. However, responses to temperature anomalies are less well understood, a critical research gap as anomalous weather becomes more common with climate change (IPCC 2023).

One theory that aims to explain temperature effects on disease is the thermal mismatch hypothesis, which posits that nonoptimal temperatures reduce parasite performance less than host performance, resulting in a relative benefit to parasites (Cohen et al. 2017, 2020). A proposed mechanism for this 'thermal mismatch' is that, on average, small-bodied organisms such as parasites have functionally wider thermal breadths (i.e., the range of temperatures at which an organism has strong performance) than larger bodied organisms (Rohr et al. 2018), possibly due to faster acclimation or adaptation. One prediction that follows is that plant populations adapted to cooler climates may experience greater disease pressure in warm weather, and that warm-adapted populations may conversely experience greater outbreaks in cooler weather. The thermal mismatch hypothesis therefore predicts a negative interaction between historical (climate) and current (weather) temperature effects on disease due to the smaller parasites performing relatively better at abnormal temperatures compared to their larger bodied hosts. Support for the thermal mismatch hypothesis comes from observations that animal populations adapted to cold climates experience larger disease outbreaks under unusually warm weather, while animals adapted to warm climates experience more disease under cold weather (Cohen et al. 2020).

Plants are inherently sensitive to environmental conditions, including average climate and interannual variations in weather. In addition, climatic effects beyond temperature affect plant diseases, including water availability (Laine 2023; Velásquez, Castroverde, and He 2018). Moisture levels have long been known to regulate plant infections (Colhoun 1973), and both drought and extreme high precipitation levels could increase disease in different contexts. Indeed, drought can increase physiological stress and therefore vulnerability to pathogen attack (Ramegowda and Senthil-Kumar 2015) (but this relationship can be nuanced: see Desprez-Loustau et al. 2006; Garrett et al. 2006), while precipitation, including extreme precipitation, can increase the spread of certain plant parasites (McElrone et al. 2010; Salinari et al. 2006) and wash away contact pesticides (Chakraborty and Newton 2011). Here, we provide one of the first tests of the thermal mismatch hypothesis in plants and extend the theory to also consider whether plant and parasite adaptation to historical precipitation levels mediates effects of short-term rainfall and moisture on disease.

Importantly, weather–disease dynamics in agricultural systems may differ from those in wild systems. First, wild plants should generally be adapted to local climates while agricultural plants have been moved around the planet for centuries (Hufford, Berny Mier y Teran, and Gepts 2019), often experiencing artificial selection for high yields across broad environmental conditions. Second, extreme weather and disease pressure are often mitigated in agricultural systems (e.g., through irrigation, shading, or pesticide application) but not in wild systems. Together, these factors suggest that in contrast to wild plants, the historical climate where agricultural plants are currently grown may not be as predictive of their sensitivity to disease under novel weather, and that, in general, agricultural systems may be less prone to the effects of precipitation and temperature compared to wild systems.

To study the potentially nonlinear and interactive effects of weather, weather anomalies and historical climate, including both temperature and precipitation, we assembled an extensive population-level database of plant disease prevalence in wild and agricultural systems from the literature ($n = 4339$ plant populations) and then paired georeferenced climate and weather data with each observed population. We used this novel database to test our predictions that: (1) contemporaneous (i.e., current during the disease survey) weather, weather anomalies and historical climate affect plant disease prevalence; (2) these weather and climate effects are stronger in wild than agricultural systems; and (3) that in warm or wet climates, disease is more likely when weather is abnormally cool or dry, and vice versa (i.e., thermal/precipitation mismatch) in wild but not agricultural systems.

## 2 | Materials and Methods

### 2.1 | Database Construction: Literature Search for Plant Disease Surveys

We conducted a systematic literature review of published plant disease surveys to compile a global, spatiotemporal database of plant disease prevalence (number of infected plants/number of

plants sampled). We searched the Web of Science via Stanford University Library in February 2021 using combinations of the search terms parasit*, survey*, disease*, pest*, pathogen*, damage*, vir*, plant*, crop*, tree*, forest*, prevalence*, incidence*, percent* and proportion*, which returned 1800 studies (see Supporting Information for full search string). Because we found that certain geographic regions—most notably, South America, Central America and the Malay Archipelago—were underrepresented, we conducted additional searches in Web of Science using combinations of the search terms above with the names of each country in these regions and screened an additional 582 non-mutually exclusive studies.

We screened abstracts as including potentially relevant data or not, using the criteria defined below. We then read each study scored as having potentially relevant data and determined if the study included information on: (1) an approximate location or latitude/longitude coordinates, (2) month(s) and year in which the survey took place (up to a maximum of six consecutive months), (3) identity of plant host and disease-causing agent, (4) sample size of the population survey and (5) either disease prevalence (number of infected plants/number of total plants surveyed) or the number of infected samples. Studies also had to (6) use randomly selected samples to calculate prevalence, (7) occur outdoors (i.e., not in a glasshouse) and (8) be conducting a survey of current infections; in other words, not only survey insect or pathogen *damage* to a plant, which could have occurred in the past, but instead survey active infestations or infections. The location and time that the survey took place (to month) were required so that we could match each population observation with appropriate weather and climate data, and we restricted the data to surveys of a maximum length of six consecutive months so that the period was short enough to reflect the contemporaneous weather that each system experienced. Identity of the plant host and disease-causing agent and population sample size and sampled prevalence were necessary to run the subsequent binomial mixed effects analysis. Using these strict screening criteria resulted in a rejection rate of approximately 95% of the 2382 studies screened, as most studies were missing at least one piece of required information. If information was partly present, we attempted to contact the corresponding authors via email to obtain the missing information. Studies included in our analyses are listed in Table S1.

Extracted data for each disease survey included host and parasite taxonomic information, the month(s) in which the survey occurred, plant sample size and disease prevalence and survey location information. If the study did not include taxonomic information, we searched the colloquial name and used the Integrated Taxonomic Information System to retrieve it. Disease-causing agent was categorised as virus, bacteria, eukaryotic parasite or pest, where eukaryotic parasites included fungal and oomycete parasites and pests were larger organisms (insects, nematodes or mites) in which active prevalence was calculated (i.e., not only recording signs of past herbivory). We recorded latitude and longitude if provided by the study, and if this information was not provided, we extracted approximate latitude and longitude from Google Maps for the centroid of the named location(s) of the survey. Additionally, we recorded the approximate spatial scale over which a survey occurred by using the ruler tool in Google Maps along with information provided by each study (e.g., 'survey occurred throughout X county') to measure the average distance across the survey range, with a minimum distance cutoff of 1 km. Median distance across the dataset was 10 km, and the maximum distance was 500 km.

## 2.2 | Database Construction: Climate and Weather Data

We paired each observation of plant disease prevalence with the climate re-analysis data extracted using Google Earth Engine (Gorelick et al. 2017). First, we specified circular buffers around the centre coordinates of each observation with diameter equal to the approximate spatial distance of the survey (minimum: 1 km; median: 10 km; and maximum: 500 km). We then extracted temperature and precipitation (weather) data from the ERA5-land monthly averaged dataset (Sabater 2019) for the location and month(s) in which each prevalence survey occurred and calculated the mean temperature and mean daily precipitation over the months of the survey period (hereby referred to as contemporaneous temperature and contemporaneous precipitation). Next, we extracted the *monthly* historical average temperature and precipitation (30-year averages from 1960 to 1990) from WorldClim Climatology V1 (Hijmans et al. 2005) for the same months in which each survey took place (i.e., if a survey occurred from April to May 2002, we extracted average 30-year temperature and precipitation for April–May in that location). Finally, we extracted historical *annual* average temperature and precipitation data (30-year averages from 1960 to 1990) for the location of each observation from the WorldClim BIO Variables dataset (Hijmans et al. 2005). We italicised *monthly* and *annual* to highlight the distinction in time periods between these two historical averages. These weather and climate data were separately extracted and recorded for each surveyed population in each study (i.e., each observation in our database) and are calculated as the per-pixel average across all pixels within the study area. Observations were excluded from the analysis if any of these climate variables were unavailable for that location.

Our database captured the strong positive correlation between the *monthly* historical temperature and contemporaneous temperature ($r = 0.928$) and the *monthly* historical precipitation and contemporaneous precipitation ($r = 0.687$) (Figure S1). We used these data to calculate new features: Temperature anomalies (contemporaneous temperature—*monthly* historical temperature) and precipitation anomalies (contemporaneous precipitation—*monthly* historical precipitation), which reflect the conditions that the plant–disease systems experienced during the disease survey relative to what would be typical at that time in that location. Temperature anomalies were weakly correlated with contemporaneous temperature ($r = 0.220$), while precipitation anomalies were positively correlated with contemporaneous precipitation ($r = 0.706$) (Figure S1). No strong correlations were detected between any temperature metrics with any precipitation metrics (Figure S1). To reduce the potential for large outlier effects, we excluded the bottom 2.5% and top 2.5% of data points with respect to contemporaneous temperature, historical *annual* temperature, contemporaneous precipitation and historical *annual* precipitation. The distribution of climatic variables, separated by parasite type, is shown for agricultural and wild systems in Figures S2 and S3, respectively.

## 2.3 | Analyses

With the goal of comparing how contemporaneous weather, *annual* historical climate, anomalous weather and their interactions affected wild versus agricultural plant disease systems, we fit a series of models separately to each type of system. Because temperature and precipitation variables were uncorrelated (Figure S1), we chose to fit the temperature and precipitation models separately, which allowed us to reduce model complexity and to avoid comparing too large a number of alternative models.

For both temperature and precipitation, we fit nine alternative models for both wild and agricultural systems (Tables S2 and S3). All models were fitted as binomial mixed-effects models using the glmmTMB function from the glmmTMB package v. 1.1.8 (Brooks et al. 2017) in R (R Core Team 2023). For each model, the response variable was disease prevalence (number of infected plants/number of plants sampled), and the unit of observation was the plant population ($n = 3776$ observations in agricultural systems, $n = 623$ observations in wild systems). The binomial model accounts for the number of trials (i.e., the sample size or the number of plants that were surveyed in each population for disease prevalence) and is thus a form of weighted regression. Because multiple observations can arise from a single study, every model included a study ID random effect to avoid pseudoreplication and a random effect for host plant order to control for non-independence across host phylogeny. We used Akaike information criterion (AIC) to compare the model performance. For the best performing models and for models presented in any of the figures, we analysed the multicollinearity of variables using the performance package v. 0.11.0 (Lüdecke et al. 2021) and report these results in Tables S12–S17. Plots and tables were created using the R packages ggplot2 v. 3.4.4 (Wickham 2016), ggbreak v. 0.1.2 (Xu et al. 2021), grid (R Core Team 2023), patchwork v. 1.2.0 (Pedersen 2024), writexl v. 1.5.0 (Ooms 2024), kableExtra v. 1.4.0 (Zhu 2024), viridis v. 0.6.5 (Garnier et al. 2024) and corrplot v. 0.92 (Wei and Simko 2021), and the packages dplyr v. 1.1.4 (Wickham et al. 2023) and broom.mixed v. 0.2.9.5 (Bolker and Robinson 2024) were used for data manipulation and model assessment, respectively.

Here, we describe the models used for investigating temperature effects. Model 1 (the null model) included study ID and plant order random effects as described above, a fixed effect for parasite type and no fixed effects for temperature. These random effects and the fixed effect of parasite type are included in all models below. Model 2 included fixed effects for contemporaneous temperature (average temperature during the survey), temperature anomaly (contemporaneous temperature—*monthly* historical temperature) and *annual* historical temperature. Model 3 was intended to test for the presence of a thermal mismatch and therefore included an interaction between *monthly* historical temperature and temperature anomaly, linear effects of both of these terms and a linear effect of *annual* historical temperature. Models 4–6 included the same fixed effects as model 2, plus an additional quadratic effect of temperature anomaly, contemporaneous temperature or *annual* historical temperature, respectively. Models 7–9 included a linear and quadratic effect of temperature anomaly, plus fixed effects for either contemporaneous temperature (7), *annual* historical temperature (8) or neither of those terms (9). These same nine temperature models were fitted separately to the data from wild and agricultural systems. The nine precipitation models that were fitted in wild and agricultural systems are analogous to the temperature models described above, with contemporaneous daily precipitation, daily precipitation anomaly and *annual* historical daily precipitation substituted in for the temperature terms. We note that daily precipitation in millimetres is the unit at which precipitation was measured, but that these data are reported by the climate data providers as an average value across the month(s) of interest.

Although temperature and precipitation variables were largely uncorrelated (Figure S1), we wanted to explore whether the model results from our best performing temperature and precipitation models changed when accounting for the other variables. We therefore analysed two combined models: One for each wild and agricultural systems which combined the variables from the best-fit temperature model with variables from the best-fit precipitation model. For all models, spatial correlation structures were not explicitly accounted for, as they cannot be included as random effects in a binomial mixed-effects model because this type of model does not include a parameter that solely defines the variance (Cohen et al. 2019b). Two studies (Abbate and Antonovics 2014; Prendeville et al. 2012) constituted a large portion ($n = 259$ combined) of the 623 observations in wild systems. We therefore repeated our model fitting and model comparison for wild systems while removing the observations from these two studies to test if our results were robust to their absence.

## 3 | Results

Our complete database comprised 4339 population-level observations of infectious disease prevalence (number of plants infected/number of plants surveyed) in plants. The data span four broad types of disease-causing agents (Figure 1a), 28 host plant orders (Figure 1b) and six continents in the time period 1984–2019 (3776 agricultural plant populations from 90 studies and 623 wild plant populations from 16 studies; Figure 2; Table S1). Various transmission modes were reported in the studies, including vector-borne, seed-borne, water-borne, wind-borne and soil-borne transmission.

In wild systems, contemporaneous temperature, historical *annual* temperature and temperature anomalies were each important in explaining variation in disease prevalence (Table S2). Specifically, the best model for wild systems (based on AIC) included each of these as predictors alongside an additional quadratic effect of temperature anomalies, with the fixed effects explaining 33% of the variance and the entire model explaining 93% (Table S2; Model 4). The quadratic temperature anomaly effect was concave-down, peaking at 2.7°C (Figure 3a), contemporaneous temperature had a negative effect (Figure 3b) and *annual* average temperature had a strong negative effect (Figure 3c). The model that included an interaction between *monthly* historical temperature and temperature anomalies (model 3), used to explore possible thermal mismatches, was not the most parsimonious but revealed

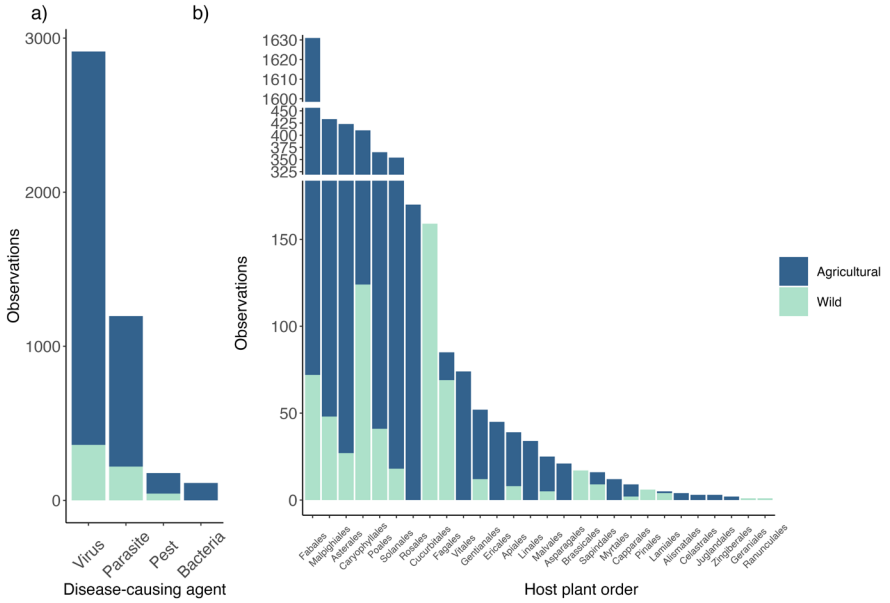


**FIGURE 1** | Pathogen, parasite and host lineages. (a) Number of survey observations by type of disease-causing agent across agricultural (blue) and wild (green) systems. 'Parasite' represents eukaryotic parasites. (b) Number of survey observations by host plant order across agricultural and wild systems.

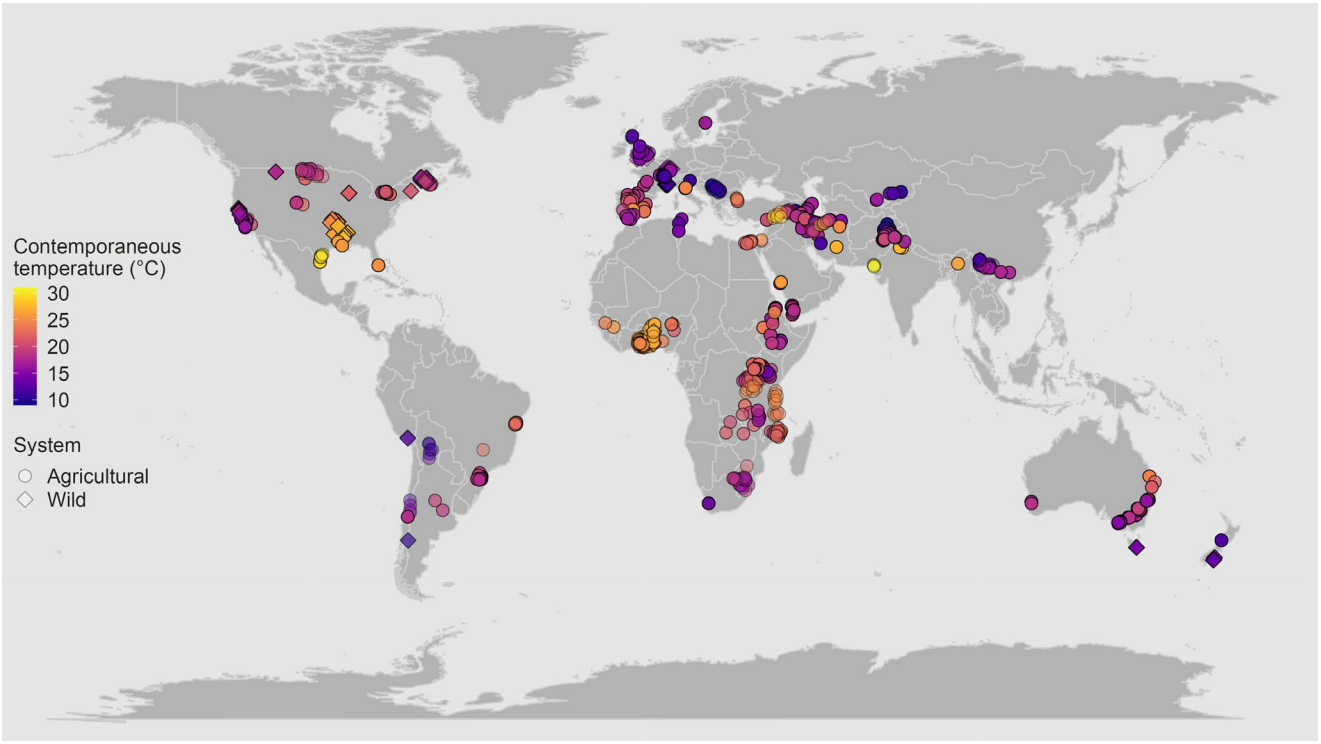


**FIGURE 2** | Global database of plant disease spanning geography, weather and climate. Records of plant disease prevalence in agricultural (circle) and wild (diamond) populations (4339 total observations). Point colour represents the average contemporaneous temperature during the disease survey for each location (°C).

a significant, negative interaction between these terms, supportive of a thermal mismatch.

In contrast, in agricultural systems, the maximum amount of variance that any of the temperature models explained was just 5%, little more than the 4% explained by the null model that only included a fixed effect of parasite type (Table S2). The best model by AIC included all temperature predictors with an interaction between *monthly* historical temperature and temperature anomaly; however, this was not supportive of a thermal mismatch effect, as the estimated interaction between these predictors was positive (i.e., larger anomalies at higher temperatures led to higher disease prevalence). Because none of these temperature models explained significant variance in the data, we

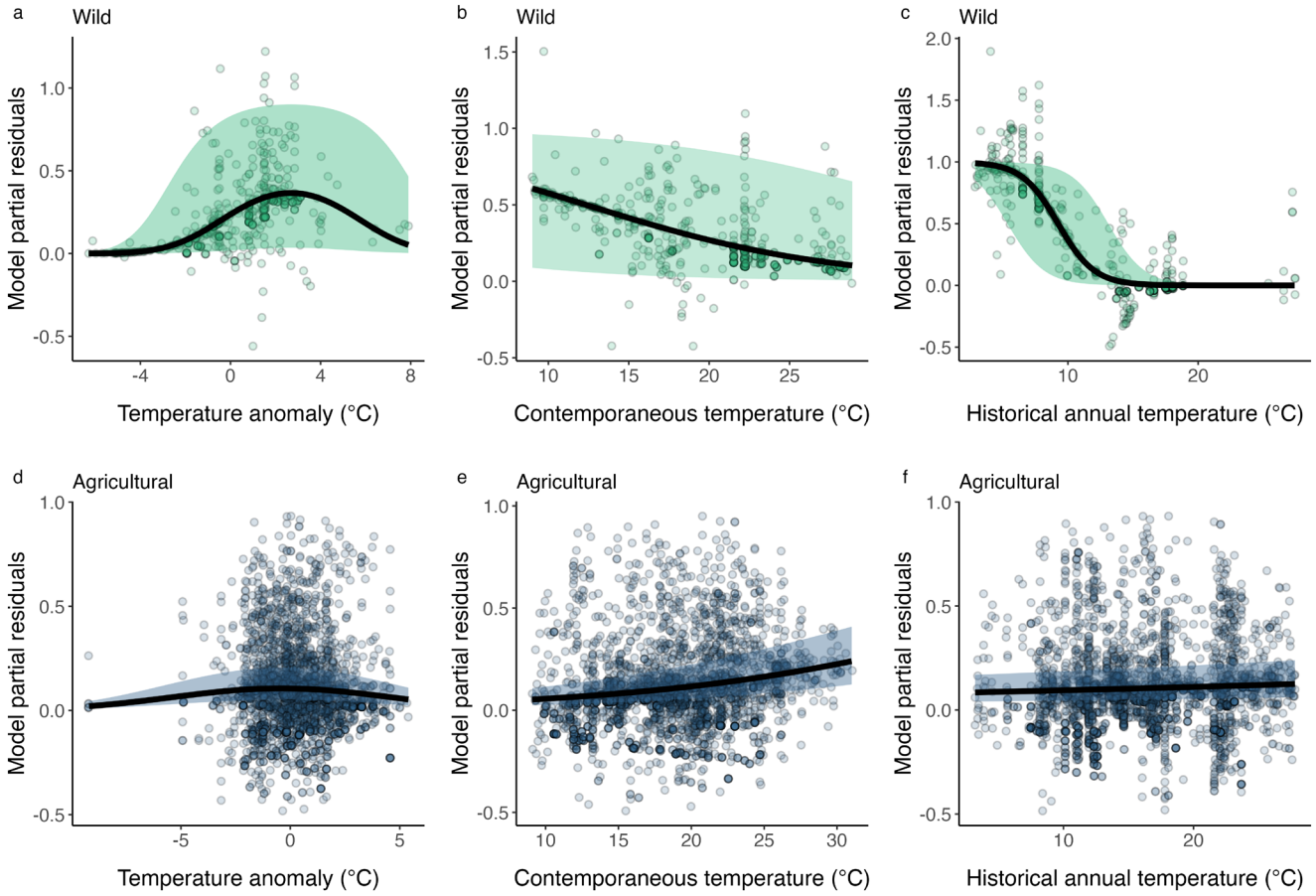


**FIGURE 3** | Estimated effects of temperature anomalies (a, d), contemporaneous temperature (b, e) and *annual* average temperature (c, f) on disease prevalence in wild (a–c; green symbols) and agricultural (d–f; blue symbols) systems. Effects for both types of systems are estimated from model 4 (Table S2), which includes linear effects of contemporaneous temperature and *annual* average temperature, as well as linear and quadratic effects of temperature anomalies. Shaded regions represent 95% confidence intervals; circles represent model partial residuals. Partial residuals are calculated as model errors plus the model-estimated relationship between the variable and prevalence. Temperature anomalies are calculated as contemporaneous temperature—*monthly* historical temperature, and contemporaneous temperature represents the mean temperature over the months of the survey period.

show (Figure 3d–f) the model fits for model 4—the best model for wild systems that includes a quadratic effect of temperature anomalies—for agricultural systems to allow a visual comparison of the same model in both system types. Here, we see a small concave-down anomaly effect peaking at −0.37°C (Figure 3d), a positive effect of contemporaneous temperature on prevalence (Figure 3e) and a little meaningful effect of *annual* average temperature (Figure 3f).

As found for temperature, precipitation explained additional variation in wild systems but had little explanatory power in agricultural systems (Table S3). The best precipitation model for wild systems included the *monthly* historical precipitation by precipitation anomaly interaction that was negative, evidence for a precipitation mismatch in wild systems (Figure 4). This model, which also included linear effects of *monthly* historical, *annual* historical and precipitation anomalies, explained an additional 14% of the variance compared to the null model, with the overall model including random effects explaining 83% of the variance in disease in wild plant systems. The various precipitation models did not meaningfully explain any additional variance in agricultural systems (Table S3), and a visual investigation of model fits shows little to no effect of precipitation anomalies (Figure 4a), contemporaneous precipitation (Figure 4b) or *annual* historical precipitation in agricultural systems (Figure 4c). In both wild and agricultural systems, respective versions of model 3 revealed significant negative interactions between *monthly* historical precipitation and precipitation anomalies, suggestive of a precipitation mismatch. However, this model explained little variation in the data compared to the null model in agricultural systems (Table S3). Model outputs (coefficients, *p*-values, etc.) are reported for each of the four best performing models for temperature in wild systems (Table S4), temperature in agricultural systems (Table S5), precipitation in wild systems (Table S6) and precipitation in agricultural systems (Table S7).

Repeating the wild system model comparisons while removing the two studies with a large number of observations led to the same best model being selected for temperature (Table S8). However, one key result changed: We estimated a significant, positive effect of contemporaneous temperature in wild systems, contrasting with the significant negative effect estimated with the full complement of data (Figure 3b; Table S4). For precipitation, the top two models switched order in terms of AIC, though

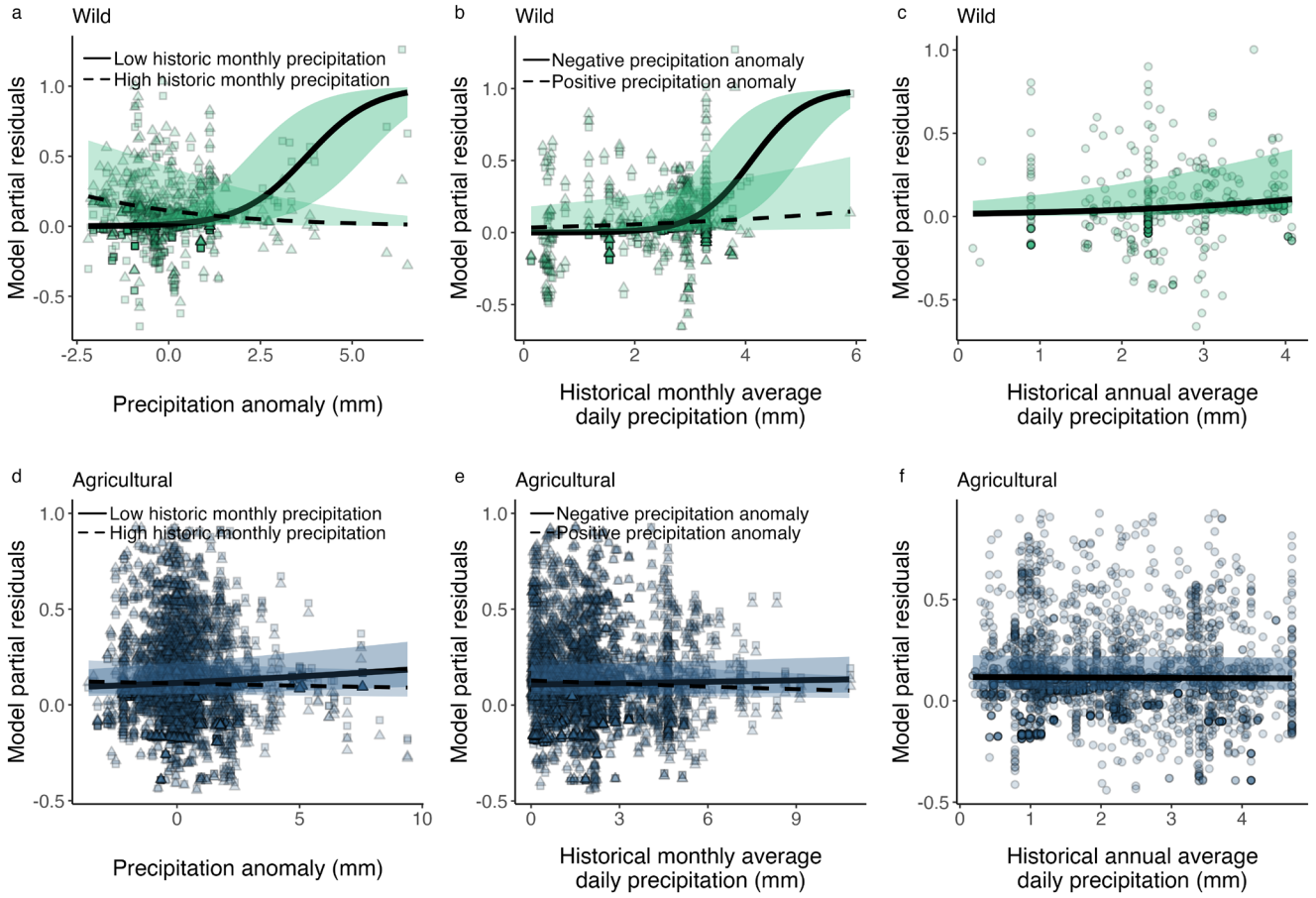


**FIGURE 4** | Estimated effects of precipitation anomalies (a, d), *monthly* historical precipitation (b, e) and *annual* average precipitation (c, f) on disease prevalence in wild (a–c; green symbols) and agricultural (d–f; blue symbols) systems. Effects for both types of systems are estimated from model 3 (Table S3), which includes linear effects of precipitation anomalies, *monthly* historical precipitation and *annual* average precipitation, as well as an interaction between precipitation anomalies and *monthly* historical precipitation. Shaded regions represent 95% confidence intervals; points represent model partial residuals. The effects of this interaction are shown with the different estimated effects in panels (a, b) (wild) and (d, e) (agricultural). The solid lines show the effects of the variable when the interacting variable is set to the 90% quantile in the data, while the dashed lines show the effect of the variable when the interacting variable is set to the 10% quantile in the data. We note that the estimated effects (solid and dashed lines) extend across all data in the x-axis, and therefore beyond the 10% and 90% quantiles. Model partial residuals associated with each of these two scenarios are represented by either squares (low or negative interacting variable) or triangles (high or positive interacting variable). Model partial residuals are represented by circles in panels c and f where there are no interacting variables. Partial residuals are calculated as model errors plus the model-estimated relationship between the variable and prevalence. Precipitation anomalies are calculated as contemporaneous precipitation—*monthly* historical precipitation.

the precipitation mismatch model still explained the most variation (Table S9).

Finally, we analysed models for both the wild and agricultural systems which combined the variables from the best-fit temperature model with variables from the best-fit precipitation model. For wild systems, we found no qualitative (and little quantitative) change in our estimates for the effects of historical temperature, temperature anomalies, monthly historical precipitation, precipitation anomalies or the precipitation mismatch (Table S10). The only variable that was qualitatively different in the combined model was contemporaneous temperature, which was estimated as having no significant effect. For agricultural systems, we found no qualitative change in our estimates for the effects of monthly historic temperature, temperature anomalies, thermal mismatches, contemporaneous precipitation or precipitation anomalies (Table S11). The small but significant positive and negative respective effects of *annual* average temperature and *annual* historical precipitation found in the separate best-fit models were both replaced by non-significant estimates in the combined model.

## 4 | Discussion

We revealed strong effects of weather and climate in wild plant disease systems, compared to relatively weak effects in agricultural systems, aligning with our hypothesis. Specifically, alternative temperature and precipitation models that included various combinations of linear and nonlinear effects of contemporaneous weather, historical climate and anomalous weather explained substantial variation in disease prevalence in wild systems, while none of these models explained noteworthy variation in agricultural systems (Tables S2 and S3).

We found a clear signal for the nonlinear effect of temperature anomalies in wild systems, which showed that disease prevalence increased with temperature anomalies up to 2.7°C warmer than the historical average for the study period. Disease prevalence decreased with contemporaneous temperature in wild systems, but this effect was noisy with wide confidence intervals, and reversed in direction when two large studies were excluded from the analyses. Moreover, contemporaneous temperature was estimated as having no significant effect in either direction when accounting for both precipitation and temperature variables in a combined model, together suggesting no strong evidence of an effect of contemporaneous temperature alone. There was also a large, negative effect of historical *annual* average temperature (Figure 3c); however, we caution the interpretation of this result, as this metric is an average measure of historical year-long climate and thus captures biogeographic patterns and differences in parasite type in the dataset rather than necessarily any mechanistic effects of year-round temperature. Although the parasite type was controlled for in the model, the wild contemporaneous temperature and annual historical temperature results may be partly mediated by uneven distributions of parasite types across these two variables (Figure S3). Specifically, in our wild dataset, systems with viral parasites tended to occur at warmer *annual* and *monthly* historical and contemporaneous temperatures compared to systems with eukaryotic parasites (Figure S3). This pattern does not necessarily reflect the true distribution of these parasite types and is more likely mainly caused by two influential studies in our wild dataset that had relatively large sample sizes: Prendeville et al. (2012) include 155 viral observations in relatively warm southern USA, while Abbate and Antonovics (2014) include 104 eukaryotic parasite observations in the relatively cold French Alps. The distribution of parasite types was not biased in any direction across the temperature anomaly variable (Figure S3) or any of the agricultural climatic variables (Figure S2).

We found some evidence for the thermal mismatch hypothesis in wild systems, in which the effects of temperature anomalies were larger in locations with historically colder temperatures in those same months and vice versa. This interactive effect was consistent with the idea that climate maladaptation is an important driver of disease outbreaks and parallels findings in animal systems (Cohen et al. 2017, 2019a, 2020). At the same time, however, a model that included nonlinear effects of temperature anomalies alongside contemporaneous and *annual* average temperatures outperformed this interactive model. Additionally, the aforementioned bias in distribution of eukaryotic versus viral parasites across climate zones could have led to similar patterns if certain parasite types respond to temperature differently. We did not seek to fit more complex models by combining quadratic terms with interactions between climatic predictors and parasite types because that would have resulted in relatively small sample sizes in each of these categories across climate and weather. While we did not find conclusive support for an effect of thermal mismatches in wild plant systems, our results suggest that only considering the temperature (or precipitation) that a wild plant–disease system is currently experiencing may be insufficient for understanding disease pressure. Instead, researchers of wild plant systems may need to also consider both nonlinear effects of temperature anomalies and possibly interactions between these anomalies and their current weather or historical climates.

In agricultural systems, models with temperature predictors explained little additional variation compared to the null model without temperature predictors (Table S2). However, the temperature models were still more parsimonious than the null, and the top two models showed significant positive effects of monthly historical temperature and contemporaneous temperature, respectively (Figure 3). There was also a significant effect of temperature anomalies, though this was mediated by an interaction with monthly historical temperature in the best model. This result, combined with the weak effects of *annual* historical temperature, suggests that warmer contemporaneous temperature leads to higher disease in agricultural systems in general. This effect may be explained by agricultural plant populations typically having comparatively short evolutionary histories in their present-day locations relative to wild systems. Additionally, they are frequently artificially selected for traits that enhance crop production and other human-preferred characteristics (Yamasaki, Wright, and McMullen 2007), including for disease resistance in light of climate change (Chapman et al. 2012). We note, however, that crop and forestry species and subspecies varieties are in part chosen based on their ability to grow and produce under a certain climate (Aitken et al. 2008), and gene flow from wild relatives can increase local crop genetic diversity (Hufford, Berny Mier y Teran, and Gepts 2019).

We hypothesised that the agricultural systems would generally be less adapted to and sensitive to local climatic conditions than wild populations because selection for human-preferred characteristics may not be directly aligned with adaptation to local climate (Mourtzinis, Specht, and Conley 2019). The absence of evidence for thermal mismatches in agricultural systems supports this hypothesis. Instead, warm temperatures increased disease prevalence on average across all agricultural systems, and these increases were slightly larger for agricultural plants grown in warm climates compared to those grown in cooler climates (indicated by the positive interaction between anomalies and *monthly* historical temperature in best-fit model 3; Tables S2 and S5). It is possible that differences in agricultural mechanisation (e.g., access to tractors; Daum and Birner 2020) underpin some biogeographic differences in the response of agricultural disease to weather variation. While we did not have fine-grained data to explore the potential effects of these factors here, it could be beneficial to incorporate socioeconomic data into future work that seeks to make more local-scale predictions of climate effects on agricultural disease.

Considering potential nonlinearities of host and parasite thermal performance from a more mechanistic standpoint may be beneficial in both wild and agricultural systems. Mechanistically linking host thermal performance curves (TPCs) to parasite TPCs in mathematical disease models can successfully predict how temperature will impact disease systems (Kirk et al. 2020; Mordecai et al. 2017; Shocket et al. 2018). Indeed, this approach has shown that warming in a marine crab–barnacle host–parasite system led to local parasite extinction due to the host thermal optimum occurring at a warmer temperature than that of the parasite (Gehman, Hall, and Byers 2018). It is plausible that a number of plant–disease systems would follow the typical unimodal

pattern in which warming leads to increased disease up to a certain temperature but then depresses disease as the temperature warms beyond the thermal limits of the parasite. To test the generality of this pattern, however, researchers will need to systematically collect performance data at a range of temperatures for different plant hosts, plant parasites and plant–disease combinations. These data could both parameterize nonlinear mechanistic models to predict disease across temperature, as well as test the underlying assumptions of the thermal mismatch hypothesis that most parasites have wider thermal breadths than their hosts. Some of these temperature-dependent data are already being collected: Chaloner, Gurr, and Bebber (2021) used data from 80 fungal and oomycete crop pathogens to show that potential crop yield gains in the future may be offset by increased disease risk under climate change. Eventually, even more complex experiments may allow researchers to parameterize mechanistic models of how temperature *anomalies* affect a disease system: For example, a factorial design in which key rates (e.g., parasite growth rate and host mortality rate) are measured at different exposure temperatures for plants and parasites that have been adapted to different temperatures. Until these data are comprehensively collected across more types of disease systems, studies such as ours can provide general insights into how we may expect temperature to affect different broad groups of plant–disease systems.

The effects of precipitation unsurprisingly differed from those of temperature; however, one general finding was shared: precipitation effects were far more influential in wild systems compared to agricultural systems. Overall, precipitation effects were weaker than temperature effects, a result that has previously been shown experimentally for foliar fungal diseases in a wild alpine meadow system (Liu et al. 2019). In wild systems, precipitation effects did not explain as much variance in the data compared to temperature, with the precipitation fixed effects in the top model explaining 14% more variance than the null model compared to 22% for the temperature fixed effects (Tables S2 and S3). The best performing model showed evidence of a precipitation mismatch in wild systems, with negative effects of precipitation anomalies on disease in plants that typically experience high precipitation, but positive effects of anomalies for plants that typically experience low precipitation. This suggests that wild plants in historically wet areas will be more susceptible to disease under drought conditions, while wild plants from dry areas are more susceptible during periods of anomalously high rainfall. There are various plausible mechanisms for this, from direct impacts of water availability on pathogens (Desprez-Loustau et al. 2006) to effects of drought on plant defence (Bostock, Pye, and Roubtsova 2014). Mechanistic models connecting precipitation and plant–pathogen systems have successfully predicted the distribution of these pathogens (Thompson, Levin, and Rodriguez-Iturbe 2013), and collecting water availability–performance curves across more host–parasite systems to then link them via mechanisms including parasite dispersal, within-host growth, host defence, host physiology and phenology would help to parse how precipitation and precipitation anomalies impact more disease systems from different climatic zones.

In agricultural systems, precipitation variables explained little variation despite nominally performing better than the null model (Table S3) and revealed no strong relationships with disease pressure (Figure 4). This does not mean that precipitation does not affect disease in any agricultural systems but that at least in this cross-system dataset, the signal of precipitation effects was too weak or heterogeneous to detect. One plausible explanation for this is that farmer intervention can dramatically impact the amount of water available to a plant–parasite system; for instance, some of the agricultural systems are likely irrigated, dampening variation in water availability due to rainfall, while other systems would not have benefitted from any irrigation or could even be drained by ditch systems to reduce saturated conditions. Because interventions like irrigation are not accounted for in our dataset, our measures of precipitation in agricultural systems may not accurately reflect the amount of water the systems experienced, leading to no signal being detected in the data.

Projected effects of climate change on precipitation are more variable than those of temperature, with some areas likely to see large increases in rainfall while other areas experience significant decreases (Collins et al. 2013). This variability suggests that plant disease prevalence will increase in some areas while decreasing in others, similar to projections for how changing rainfall patterns will alter distribution of human diseases such as malaria in West Africa (Yamana and Eltahir 2013) and cholera across Africa (Moore et al. 2017). Projections for how climate change will affect plant diseases will thus need to account for potential differences in how temperature and local precipitation patterns will change relative to each other. For instance, the downy mildew–grapevine system exhibits positive relationships between both temperature and disease and rainfall and disease (Salinari et al. 2006). Despite projected decreases in rainfall for the mildew–grapevine system's region of Italy, disease epidemics are expected to increase because increases in temperature-driven disease will more than offset any reduction due to decreased rainfall (Salinari et al. 2006). Future models that incorporate the direct and possibly interactive effects of temperature and precipitation alongside additional environmental axes (e.g., $CO_2$, which can interact with temperature to regulate plant pest populations (Newman 2004)) are likely to exhibit increased predictive accuracy.

Our analysis gains strength from examining trends across multiple plant–disease systems; however, by doing so, we may mask important system-specific variation. Indeed, some plant–disease systems included from our systematic literature review are likely less appropriate for our analyses than others. For example, we did not have data for whether surveys of wild plant systems occurred in the native range for the plants, and therefore some of the wild plant systems may have had significantly longer periods to adapt to their current climate than others. Another example is that if a survey included a population of plants that can host long-term, chronic infections, it is possible these plants were infected a considerable time before the survey began. In this case, the weather conditions during the survey would not be as informative about the conditions when the plant became infected compared to plant systems that only exhibit infections over a short time. While we excluded surveyed populations that did not have current infections (e.g., those studies which were screened and included surveys showing insect or pathogen *damage* to a plant but not necessarily active infestations or infections), we

did not have data on prior infection status for most of the studies included in our analysis. Importantly, however, because we do not expect these studies to be biased towards warmer or colder weather, the inclusion of these studies may add noise to our results but should not bias them.

Though it is difficult to parse which exact biological mechanisms underpin our results with many different types of host–parasite systems included in our study, there are certain types of mechanisms that are likely at play. Desaint et al. (2021) have shown that elevated temperature impacts plant disease resistance across the majority of different plant–pathogen systems investigated, with a variety of ways in which this can occur. For instance, temperature effects may alter plant development and metabolism (Yang et al. 2018) or plant cell physiology (Bita and Gerats 2013), while simultaneously increasing parasite reproduction rates or virulence (Desaint et al. 2021). Our temperature anomaly results showed that wild plants experiencing warmer temperatures relative to historic averages increase disease risk up until a maximum of 2.7°C warmer than typical, after which point higher temperatures will decrease disease. This suggests that on balance, anomalously warm temperatures up until 2.7°C lead to key parasite rates such as reproduction or growth benefiting more than host rates (or, alternatively, suffering less costs than the hosts), while elevated temperatures past this level hinder the parasite more than it does the host. This type of overall nonlinear disease response is often seen in response to mean temperature in other types of host–parasite systems (e.g., mosquito-borne disease (Mordecai et al. 2019); water-borne disease in *Daphnia* (Shocket et al. 2018)), through which host and parasite trait responses are most important, will differ across plant–parasite systems. Some generalities are likely to exist: For example, transmission in plant–disease systems that require an insect vector will be strongly influenced by the thermal sensitivities of that insect, while transmission in soil-borne systems may be somewhat buffered to changes in environmental temperature. Moreover, features of broad taxonomic groups can also provide insights into the mechanisms underpinning climate effects on individual groups, such as evidence that fungi in soil are better adapted to cool temperatures than bacteria (Pietikäinen, Pettersson, and Bääth 2005).

Understanding how anomalous weather conditions in different climates mediate plant disease outbreaks is critical for anticipating and mitigating climate change impacts for agricultural and wild plant systems, which affect food security (Ristaino et al. 2021) and ecosystem integrity (Jiranek et al. 2023). Our results highlighting the impact of temperature and precipitation anomalies suggest that the evolutionary history of wild plant–disease systems in their historical climate affects vulnerability to disease. Agricultural systems, which have typically had a shorter evolutionary history in their present-day locations, are vulnerable to pathogen outbreaks under warm conditions regardless of historical climate or anomalous conditions, but all weather effects were relatively weaker in these systems. These data-driven conclusions identify general patterns that may apply to understudied systems under anomalous weather and climates. More broadly, our results spotlight the need to study potential disease outbreak effects driven by climate change on biodiversity in wild plant systems, effects which may need to be considered alongside effects of other global change drivers such as habitat loss simultaneously (Laine 2023).

### Author Contributions

All authors conceived of the study. D.K. and V.N. conducted the literature review. D.K., J.M.C. and M.L.C. conducted analyses. D.K. wrote the first draft of the manuscript, and all authors significantly contributed to editing and revising the manuscript.


### Acknowledgements

We thank members of the Mordecai and O'Connor labs and Phil Harmon for valuable feedback on the manuscript. EAM was supported by the National Science Foundation (DEB-2011147, with Fogarty International Center), the National Institutes of Health (R35GM133439, R01AI168097, R01AI102918), the Stanford Woods Institute for the Environment, the Stanford Center for Innovation in Global Health and the Stanford King Center on Global Development. SLF was supported by USDA-EEID (1031035).


### Conflicts of Interest

The authors declare no conflicts of interest.

### Data Availability Statement

Data and supporting R code are archived on Dryad at https://doi.org/10.5061/dryad.p8cz8wb0h.

### Peer Review

The peer review history for this article is available at https://www.webofscience.com/api/gateway/wos/peer-review/10.1111/ele.70062.

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## Supporting Information

Additional supporting information can be found online in the Supporting Information section.