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A multi-year case study highlighting the influence of hydrological conditions on epidemic dynamics in a natural plant pathosystem

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The scale of influence of hydrological and thermal conditions on disease remains uncertain for most wild plant pathosystems, thus restricting our ability to predict the impacts of climate change. Analysis of the spatiotemporal spread of a fungal rust pathogen throughout four naturally occurring flax populations over the course of five growing seasons reveals relationships between epidemic magnitude and snow cover, relative humidity and temperature, as well as an unexpectedly significant effect of severe drought on disease progression. These results indicate that climate change will likely disrupt wild plant epidemics, and points to a need for further epidemiological studies characterizing the effects of environmental conditions on population-level disease dynamics in natural pathosystems.

Keywords: Climate change, drought, epidemics, flax, flax rust, fungal disease, hydrology, plant pathogens



Introduction

Plant pathogens constitute a major barrier to global food security (Savary et al. 2019), and drive eco-evolutionary dynamics in natural plant populations (Gilbert 2002, Termorshuizen 2016). Climate change is expected to modulate epidemiological dynamics (McCann 2020, Chaloner et al. 2021, Raza and Bebbler 2022), including those in natural pathosystems, which have received comparatively little attention thus far (Abbate and Antonovics 2014, Singh et al. 2023). Anticipating such dynamics will require substantial knowledge of the underlying interactions between pathogens, their hosts, and the environment (Scholtz 2007).

In natural ecosystems, transmission of plant pathogens commonly results in seasonal epidemics (Burdon 1993, Alexander 2010, Miller et al. 2022). During these epidemics, disease prevalence (cumulative number of infected plants/total number of plants) and disease severity (intensity of infections) both accumulate over time, often conforming to a predictable disease progression curve (Nutter et al. 1997, Arneson 2001,



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Rodrigues et al. 2019). While independent features of host populations (e.g. density, size, resistance genetics) and independent features of invading pathogens (e.g. virulence, host range) are all thought to play roles in modulating the course of plant pathogen epidemics (Parleviet 1979, Jarosz and Davelos 1995, Oliva et al. 2013, Duggal et al. 2023), environmental conditions are thought to be key to shaping within-season disease dynamics (Colhoun 1973, Velásquez et al. 2018, Singh et al. 2023). Our understanding of the environmental determinants of disease has improved recently in agricultural settings (Grabow et al. 2016, Gao et al. 2018, Beruski et al. 2019), but significant uncertainty remains in how the same conditions drive epidemic outcomes in natural pathosystems (Miller et al. 2022, Singh et al. 2023).

Here, we tracked the spatiotemporal spread of *Melampsora lini*, a fungal rust pathogen, throughout four wild populations of Lewis flax *Linum lewisii* over the course of five growing seasons. Concurrently, we collected local weather data from each population site. Using these data, we fit logistic equations to the prevalence time series (i.e. population-level disease progression) of observed epidemics, and then fit generalized additive models to correlate variation in the fitted logistic parameters with variation in local environmental conditions. We identify the major correlates of epidemic magnitude and evaluate candidate explanations for why disease dynamics diverged in 2023 epidemics, highlighting an empirical, significant disruptive effect of drought (Choudhary and Senthil-Kumar 2024).

Material and methods

Data collection

Detailed information about the long-term flax rust epidemiological dataset (e.g. precise study site locations, pathosystem biology, technical equipment specifications, study design rationale, specific protocols, etc.) is provided in Miller et al.

2022. In summary: four 200-m² plots (BT, CC, GM, HM) spanning a large elevational gradient in the Rocky Mountains (Fig. 1A) were monitored via measurements of epidemiological and ecological dynamics of *L. lewisii* (henceforth: flax) and *M. lini* (henceforth: flax rust) over the course of five growing seasons (~ mid-June to early-August each year). In all five years, the number of diseased plants was recorded towards the beginning of the season. Subsequently, every week, previously non-diseased plants were checked for new infections. In addition to this population-level epidemiological data, various within-host disease progression data were tracked for several focal plants (~ 10% of each plant population).

High-resolution weather data was collected for each site. Specifically, temperature, relative humidity, dew point, leaf moisture, rainfall quantity, soil water content, solar radiation intensity, wind speed and wind direction were all sampled in the corner of each site at 5 min intervals for the duration of each growing season. Local snow cover duration data was pulled from the RMBL spatial data platform (Breckheimer et al. 2021).

Data processing

The first and last fifteen minutes of weather data, in addition to aberrant data points resulting from infrequent equipment/software malfunctions, were removed from each site and year. Disease incidence data was collated from weekly epidemiological surveys. Weekly disease depletion rates were calculated using repeated temporal observations of focal plants in each plot. Specifically, the proportion of focal plants whose number of diseased stems (2019–2022) or whose disease class (2023) went from 1+ to 0 in consecutive weeks was calculated. From these epidemiological data, weekly disease prevalence was interpolated for each epidemic following

$$\text{Prevalence} = (I_{t-1} + J_t - \gamma_t \times I_{t-1}) / N \quad (1)$$

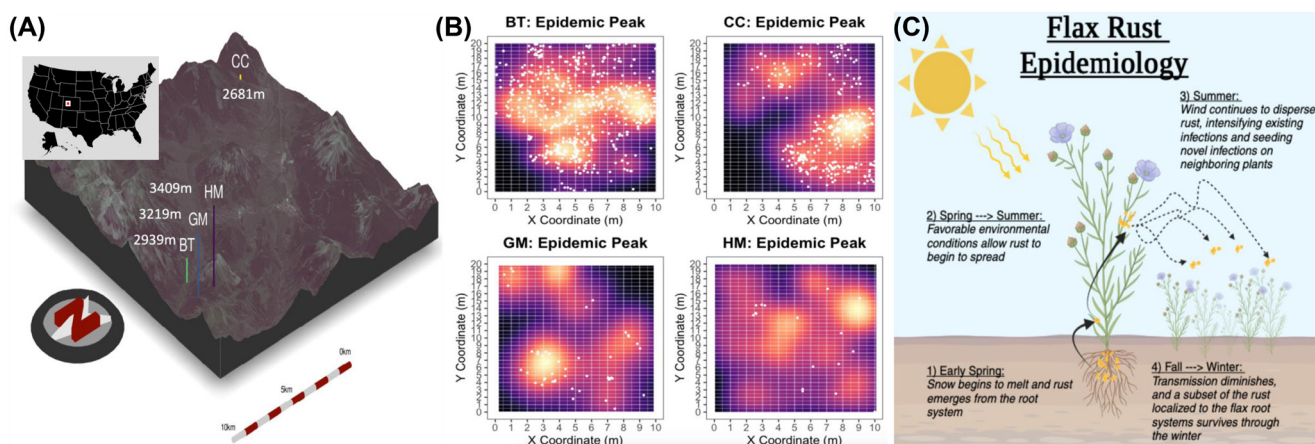


Figure 1. Flax rust study system. (A) The four 200-m² flax population sites that were monitored over the course of this study are depicted along with the topography of the surrounding Rocky Mountain area (adapted from Miller et al. 2022). (B) The peak magnitudes of the 2022 epidemics are depicted in each of the four sites. White dots signify infected plants and lighter background colors represent higher local densities of flax. (C) A schematic highlights the relevant biological and environmental processes that are hypothesized to drive flax rust epidemiology in this study system (created in Biorender).

where I represents the cumulative number of infected plants, J represents weekly new infections, γ represents the weekly disease depletion rate, and N is the total population size. Unique population surveys were performed each year. However, due to uncertainty in the BT 2019 demographic survey, the size (N) of that year's population was taken from the 2020 survey, with the assumption that demography did not shift drastically between the two years (Bierzychudek 2014).

Data analysis

To explore the effects of environmental conditions on epidemic dynamics, we first fit logistic equations (Eq. 2), using the Gauss–Newton algorithm in the *nls()* function in R ver. 4.2.0 (www.r-project.org), to observed epidemics that displayed expected sigmoidal growth (i.e. excluding epidemics from 2023, and those with insufficient temporal coverage such as HM 2021 and CC 2021).

$$y = \frac{K}{1 + e^{-r \times (t - k_{0.5})}} \quad (2)$$

The logistic function (Eq. 2) has three parameters describing different aspects of flax rust epidemics: K , the maximum prevalence (i.e. upper asymptote in the disease progression curves), r , the rate of epidemic growth, and $k_{0.5}$, the date at which half-maximum prevalence is achieved.

To explore how climate drivers might affect these epidemiological features, we fit generalized additive models

(GAMs), using the *gam()* function from the 'mgcv' package (family='gaussian', method='REML'), to explore empirical relationships between each parameter (Eq. 2) and average measures of temperature and humidity (i.e. mean daily values averaged from 1 July to 25 July – a subset of the growing season that captures the majority of epidemic growth) – and annual snow cover duration in eleven epidemics from the BT, CC, GM sites. These focal climate variables were selected based on 1) previous research highlighting their dominant effects on plant pathogens (Garrett et al. 2006, Laine 2008, Donnelly et al. 2011, Velásquez et al. 2018), including flax rust (Lawrence et al. 2007), and 2) the fact that their data was collected in each year and site (unlike other conditions like rainfall and leaf moisture which were incompletely represented in the dataset). Each model also included a random site effect (as in Miller et al. 2022) to account for the repeated observations of disease and to control for non-environmental confounders that we did not measure (e.g. differential pathogen virulence and/or host resistance, etc.), or which couldn't be accommodated in the GAMs due to the small epidemic dataset size (e.g. population density). We used the *gam.check()* and *concurvity()* functions to ensure that the conditions of regression were satisfied for each model.

Results

Between 2019 and 2022, disease progression displayed prototypical sigmoidal growth (Fig. 2) (Xu 2006). In most years,

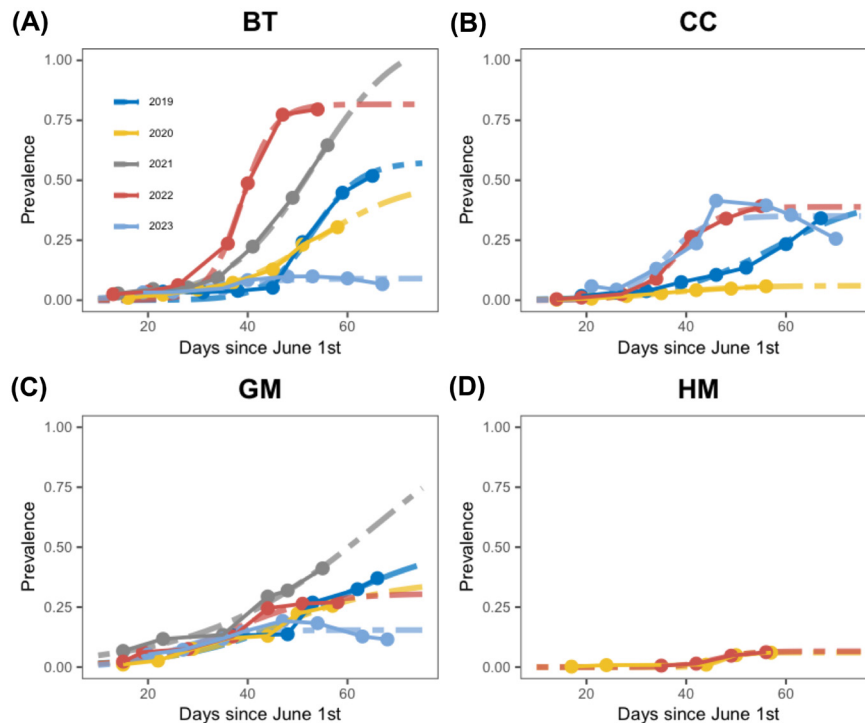


Figure 2. Fitted logistic models of flax rust epidemics. Interpolated weekly rust prevalence (points and solid lines) and NLS-fitted logistic curves (dashed lines) are shown for (A) BT, (B) CC, (C) GM and (D) HM sites. Epidemics with insufficient prevalence data for curve-fitting (e.g. HM: 2021, CC 2021) are excluded.

disease prevalence was highest at the BT site, followed by CC, GM, and finally by HM (Fig. 2). Considerable intrasite variation in maximum epidemic prevalence across years (e.g. CC 2020) suggests that epidemic ceilings are influenced heavily by seasonally and locally varying variables like environmental conditions.

We used GAMs to explore associations of maximum disease prevalence (Eq. 2: K), epidemic timing (Eq. 2: $k_{0.5}$), and epidemic growth rates (Eq. 2: r) with local environmental conditions (Supporting information). Models including temperature, humidity, snow-cover duration, and a random site/population effect explain 49.9% of deviance in maximum epidemic prevalence, 47.7% of deviance in epidemic timing, and 87.1% of deviance in epidemic growth rates ($n=11$). In line with previous work, we find that higher average relative humidity and longer snow cover duration correlate with greater epidemic peaks ($p < 0.08$, $p < 0.15$). Higher average temperatures show no significant effect on maximum disease prevalence ($p < 0.61$). Epidemic timing is not significantly correlated with any environmental driver ($p > 0.05$), although longer snow cover durations do correspond with later epidemics ($p < 0.15$). By contrast, high relative humidity and low average temperature are significantly associated with more rapid epidemic progression ($p < 0.05$).

Epidemic trajectories in 2023 deviated considerably from those of prior years, and weekly incidence data indicated that this was a result of elevated rates of disease depletion (Supporting information). Strikingly low levels of rainfall (percent difference between 2023 and previous 3-year average: BT=98.4, CC=100, GM=99.1, HM=99.5%) and leaf moisture (percent difference: BT=72.6, CC=69.9, GM=98.2, HM=93.3%) in the 2023 season relative to previous years (Fig. 3B) in a context of more comparable temperature (Fig. 3D), suggest that drought was a primary driver of the observed epidemic abnormalities. While few studies have directly assessed the effects of drought on the

epidemiological dynamics of wild plant pathogens, this result aligns with previous work implicating 1) the importance of rain and leaf moisture for fungal pathogens (Huber and Gillespie 1992, Ji et al. 2023), and 2) the detrimental effects of drought on plant disease (Zhan et al. 2022).

Discussion

Overview

Leveraging a unique five-year dataset on the spread of a plant pathogen in populations across an altitudinal gradient, our results indicate an important role of environmental variation, with relative humidity in particular amplifying the speed and eventual magnitude of outbreaks. Further, striking deviation of disease progression observed in 2023 rust epidemics suggest that drought and resulting disease depletion were the key drivers.

Thermal and hydrological conditions explain substantial variation in normal epidemics

Water availability (i.e. atmospheric humidity, soil moisture, rainfall, etc.) favors the spread of wind-dispersed fungal plant pathogens (Velásquez et al. 2018), and for flax rust specifically, high humidity or the presence of free water is required for spore germination (Lawrence et al. 2007). Additionally, optimal thermal ranges for pathogen infection and spread amongst hosts are ubiquitous (Velásquez et al. 2018). For flax rust infecting agricultural flax, this optimal range is relatively cool: mycelial growth requires moderately low temperatures (16–17°C), and temperatures above 23–27°C inhibit germination (Lawrence et al. 2007). However, while our results indicate a strong effect of humidity on both the speed and scale of outbreaks, temperature showed a significant effect only on disease progression rates. This suggests either that

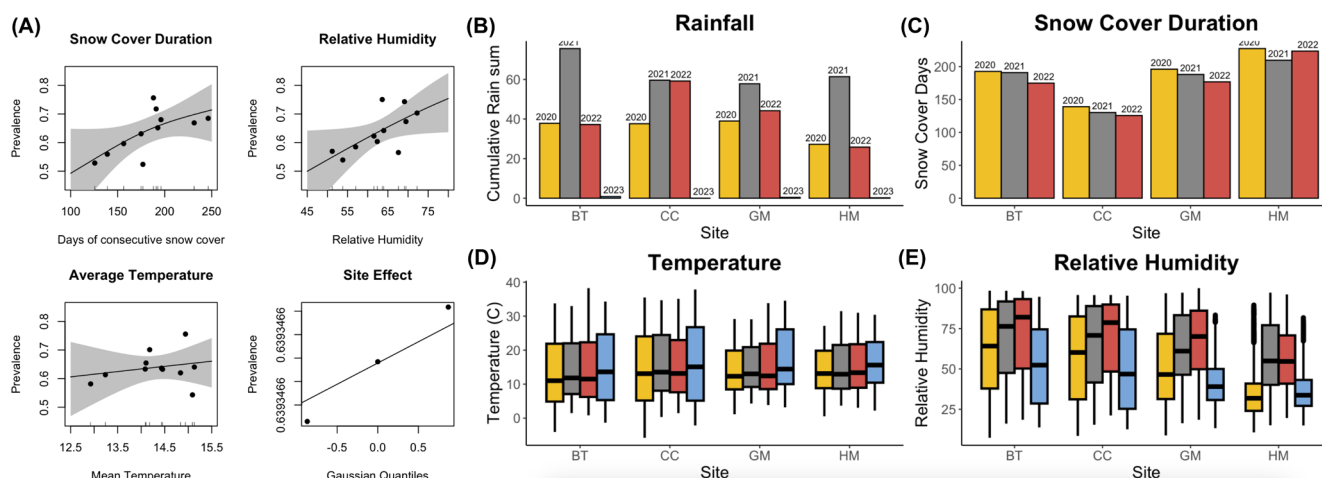


Figure 3. Exploring empirical associations between weather conditions and epidemic dynamics. (A) Fitted GAM model explaining maximum epidemic prevalence. (B–E) Cumulative sums (bar plots) and averages (box plots) for weather conditions tracked between 1 July and 25 July, and snow cover duration for each site. (B) Site × Year: cumulative rainfall. (C) Site × Year: snow cover duration. (D) Distribution of observed temperatures. (E) Distribution of observed relative humidity values.

hydrology has a greater constraining effect on natural plant epidemics than temperature, or that the range of temperature encountered in this study was not large enough to considerably perturb epidemic magnitude, while that of humidity was, potentially affecting both within and between host spread (Miller et al. 2022, Jiranek and Duggal unpubl.).

Highlighting the disruptive effects of drought on 2023 epidemics

By mid-July, the trajectory of epidemics in 2023 had deviated considerably from the corresponding trajectories of previous years (Fig. 3A). Removing disease depletion (defined as the event of a plant previously identified as diseased showing no evidence of infection in a subsequent survey) from Eq. 1 returns sigmoidal trajectories in line with those observed in previous years, including after adjusting these for disease depletion (Supporting information), suggesting an underlying cause for these deviations. Given that within-season plant death, primarily occurring in seedlings which were rarely diseased, likely did not contribute significantly to population-scale disease depletion (Jiranek and Duggal unpubl.), abiotic-stress-induced plant tissue loss was likely the primary mechanism of disease depletion.

While we cannot exclude effects of higher recorded temperature on plant and/or pathogen physiology and thereby disease depletion rates (Hasanuzzaman et al. 2020, Zhang et al. 2021), multiple lines of evidence collectively indicate drought as the primary cause. First, as discussed above, moisture is a requirement for fungal pathogens (Kolb et al. 2016), including flax rust (Lawrence et al. 2007). Second, epidemics in distinct populations showed the same unique abnormality in 2023 (Supporting information), which suggests a shared environmental cause as opposed to improbable independent evolution of pathogen virulence or plant resistance (Roy and Kirchner 2000). Third 2023 rainfall and leaf moisture levels in July are significantly depressed relative to other years, whereas differences in other environmental conditions are less pronounced (Fig. 3). And fourth, we qualitatively observed that diseased plants in the field late in the season had increasingly desiccated leaves and stems. Drought-induced tissue death (Ravensdale et al. 2011, Zhan et al. 2022), drought-induced plant resistance (Gao et al. 2018), and/or drought-induced pathogen inhibition (i.e. constraints on germination, sporulation, and dispersal) (Wakelin et al. 2018) could all have contributed to the observed declines in infection prevalence. That the magnitude of the CC epidemic was greater in 2023 than in other years is a notable contradiction, suggesting that conditions unrelated to hydrological availability (e.g. host resistance genetics, pathogen virulence, metapopulation spread, etc.) might influence epidemiological dynamics more than anticipated, and need to be investigated further.

Speculations

Much of the analysis of climate change effects on plant pathogen spread in natural ecosystems has focused on

the amplifying effects of temperature (Bebber 2015). Importantly, however, drought causes tissue damage and leaf loss in plants (Vesk and Westoby 2003, Seleiman et al. 2021). Leaf-borne pathogens might therefore be particularly susceptible to reductions in rainfall, and thus pathogen densities could be severely depleted by the predicted increasing occurrence of drought conditions (Cook et al. 2018). The cross-scale effects on population-level disease dynamics (Thrall and Jarosz 1994) could be considerable, potentially even driving local stochastic pathogen extinction (Hartfield and Alizon 2013) where one might otherwise predict pathogen spread.

Tissue dieback and leaf borne pathogens represent one extreme of a continuum along which drought may both facilitate and hinder plant disease depending on contextual features like drought intensity and duration (Choudhary and Senthil-Kumar 2024). In future climates, increased risk of drought is likely to coincide with increases in temperature (Price et al. 2022), thus either amplifying or diminishing effects on pathogen spread. Understanding the ecological and (co)evolutionary consequences of such changing selection pressures (González et al. 2021) in a changing world requires understanding the cross-scale dynamics of pathogens, from individual infections (e.g. mechanistic effects of drought/temperature on resistance and virulence, Pandey et al. 2015, Pandey and Senthil-Kumar 2019, Szczepaniec and Finke 2019) to transmission between individuals and across populations. Further analogous epidemiological data is needed to characterize these largely uncharted latter processes (Miller et al. 2022).

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Author contributions

Keenan Duggal: Conceptualization (lead), Data curation (lead), Formal analysis (lead), Funding acquisition (supporting), Investigation (equal), Methodology (lead), Project administration (equal), Visualization (lead), Writing - original draft (lead), Writing - review and editing (lead). **Juliana Jiranek:** Conceptualization (supporting), Data curation (supporting), Funding acquisition (equal), Investigation (equal), Methodology (equal), Project administration (equal), Supervision (equal), Writing - original draft (supporting), Writing - review and editing (supporting). **Madison Machado:** Data curation (supporting), Investigation (supporting), Writing - original draft (supporting). **Peyton Smith:** Data curation (supporting), Investigation (supporting), Writing - original draft (supporting). **Ian Miller:** Conceptualization (supporting), Data curation (equal), Funding acquisition (equal), Investigation (equal), Methodology (equal), Project administration (equal), Writing - original draft (supporting). **Jessica Metcalf:** Conceptualization (equal), Formal analysis (equal), Funding acquisition (lead), Methodology (equal), Project administration (equal), Supervision (lead),

Visualization (equal), Writing - original draft (equal), Writing - review and editing (equal)

Data availability statement

Data are available from the Dryad Digital Repository: <https://doi.org/10.5061/dryad.98sf7m0tc> (Duggal et al. 2024).

Supporting information

The Supporting information associated with this article is available with the online version.

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