Metapopulation Structure Predicts Population Dynamics in the Cakile maritima–Alternaria brassicicola Host-Pathogen Interaction

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Abstract: In symbiotic interactions, spatiotemporal variation in the distribution or population dynamics of one species represents spatial and temporal heterogeneity of the landscape for the other. Such interdependent demographic dynamics result in situations where the relative importance of biotic and abiotic factors in determining ecological processes is complicated to decipher. Using a detailed survey of three metapopulations of the succulent plant Cakile maritima and the necrotrophic fungus Alternaria brassicicola located along the southeastern Australian coast, we developed a series of statistical analyses—namely, synchrony analysis, patch occupancy dynamics, and a spatially explicit metapopulation model—to understand how habitat quality, weather conditions, dispersal, and spatial structure determine metapopulation dynamics. Climatic conditions are important drivers, likely explaining the high synchrony among populations. Host availability, landscape features facilitating dispersal, and habitat conditions also impact the occurrence and spread of disease. Overall, we show that the collection of extensive data on host and pathogen population dynamics, in combination with spatially explicit epidemiological modeling, makes it possible to accurately predict disease dynamics—even when there is extreme variability in host population dynamics. Finally, we discuss the importance of genetic information for predicting demographic dynamics in this pathosystem.

Keywords: metapopulation, disease dynamics, epidemiology, spatially explicit model, Cakile maritima, Alternaria brassicicola.

Introduction

Ecological and genetic factors driving plant disease dynamics interact across multiple spatiotemporal scales, ranging from single leaves through to populations connected within larger landscapes (Borer et al. 2016). The importance of specific biological and physical phenomena to the epidemiology of the system will vary across these different scales, although all may interact. For example, at fine scales, in order for disease to develop, susceptible hosts, infective pathogens, and a suitable microenvironment must co-occur (McNew 1960; Scholthof 2007; Barrett et al. 2009). Host and pathogen population structure (e.g., host density and genetic diversity) may also strongly influence disease dynamics at the population scale, while factors affecting colonization and extinction (e.g., pathogen dispersal, distance between habitat sites) can affect landscape-scale dynamics (Smith et al. 2011). Integrating across scales and developing the capacity to predict when and where epidemics will develop across landscapes are critical for developing sustainable strategies for the management of plant diseases in wild and domesticated systems (Meyer et al. 2017).

Spatiotemporal variation in any of the elements listed above can result in distinctly different epidemiological dynamics across a landscape. In addition, feedback loops between partners can develop, particularly in natural systems where the spatiotemporal dynamics of any one of the species in an interaction also affect the spatial and temporal heterogeneity of the landscape for the other. This leads to complex demographic dynamics where the relative importance of the roles of biotic factors, including genetic variability, and abiotic factors in determining epidemiological processes can be complicated to decipher (Boulangeat et al. 2012). Indeed, confounding effects among the diversity of processes in an interaction as well
as our inability to directly observe critical processes can hamper statistical inference in natural populations. Recently, experiments conducted at large spatial scales have identified the role of host genetic variation and environmental conditions relative to pathogen dispersal in determining the spatiotemporal dynamics of powdery mildew (*Erysiphe alphitoides*) on the pedunculate oak (*Quercus robur*; Ekholm et al. 2017). However, long-term studies of natural systems do not manipulate the plant-pathogen metapopulation structure. In such cases, a mechanistic-statistical approach (Soubeyrand et al. 2009) can be used to infer the processes governing the interaction. As this approach separately models the actual host-pathogen dynamics and, conditioned on these dynamics, the observations, any knowledge of the mechanisms governing the interaction can easily be incorporated. The mechanistic-statistical approach has been used to infer crucial parameters determining the spatiotemporal dynamics of several plant diseases (Soubeyrand et al. 2009; Fabre et al. 2010; Bousset et al. 2015; Pleydell et al. 2018; Abboud et al. 2019) with other applications in ecology (Wikle 2003; Barnagaud et al. 2014; Soubeyrand and Roques 2014; Williams et al. 2017).

Pathologists have long recognized the important role of both the macro- and microenvironment for pathogen reproduction. However, comprehensive assessment of the influence of a broad suite of demographic and environmental factors for naturally occurring epidemics remains rare (Barrett et al. 2009). Tack and Laine (2014) and Penczykowski and colleagues (2015) investigated how various environmental factors influenced disease development and persistence in a series of metapopulations of *Plantago lanceolata* attacked by *Podosphaera plantaginis* and demonstrated the importance of spatial variation in winter environment in explaining the production of resting spores and subsequent epidemic development. More recently, Ericson and colleagues (2017) demonstrated that spatial variation in host population dynamics and the physical environment, in addition to historical patterns of disease severity, influenced temporal changes in the epidemic behavior of 31 populations of the rust pathogen *Uromyces valerianae*. In particular, monthly assessment of precipitation, temperature, and sea level revealed the relative importance of winter sea-level changes on subsequent disease development.

In spatially structured populations, realized variation in environmental conditions also depends on habitat quality, the dispersal ability of individuals, and the spatial configuration of habitat patches. Together, these elements strongly influence spatial variation in the density, size, and spatial continuity of populations (Parratt et al. 2016). Wild plant populations are no exception; their distributions generally represent a diversity of patterns of different spatial and temporal scales and complexity. Such heterogeneities in the host population have important consequences for disease dynamics. For example, Thrall and colleagues (2003) used experimental metapopulations to investigate how variation in the degree of isolation among plant demes influenced pathogen population dynamics in the *Linum marginale–Melampsora lini* plant-pathogen system. They found that the spatial distribution of host populations affected disease dynamics and genotypic composition of local pathogen populations largely via stochastic extinction and recolonization events. Similarly, while the incidence and severity of rust caused by *U. valeriana* showed considerable temporal and spatial variability among populations of its host *Valeriana salina*, including those in proximity to one another, an underlying association between disease prevalence and distance between populations was detectable (Ericson et al. 2017).

Ultimately, characterizing the epidemiological complexities of a host-pathogen interaction and its underlying drivers is essential to understanding spatial and temporal variation in coevolutionary trajectories. Disease epidemiology is the visible manifestation of the interaction occurring between individual hosts and pathogens; it is driven by environment, genetics, and life-history traits of interacting host and pathogen populations. Although disease may be constantly present in a metapopulation as a whole, fluctuations in population size at the individual deme level may result in changes in population structure at both the local and metapopulation scales through bottlenecks, genetic drift, and extinction events (Burdon and Thrall 1999; Smith et al. 2011; Ericson et al. 2017). Furthermore, spatiotemporal variation in the presence and severity of disease determines the potential selective pressure hosts experience and hence helps predict genetic and evolutionary changes in both host and pathogen (Jousimo et al. 2014).

Although the *Plantago* and *Valeriana* studies noted above spanned many years (12+ and 28+ years, respectively), they both assessed epidemic development at a single time point in each annual epidemic. This approach precludes detailed investigation of the relative importance of different biotic and abiotic factors in driving the development of individual epidemics. Here we take advantage of a highly detailed survey (every 6 weeks for 5 years) of three metapopulations of the plant *Cakile maritima* and its pathogenic fungus *Alternaria brassicicola*. The aim of this work was to quantify the respective roles of habitat, weather conditions, and dispersal in modulating the spatial and temporal dynamics of host-pathogen interactions. In an earlier publication, Thrall and colleagues (2001) provided a preliminary analysis of this system with regard to epidemiological patterns and their relationship to some physical aspects of population sites through the course of a single year. Here we use the multiyear data set to develop a spatially explicit predictive model of disease dynamics to conduct
a more complete analysis of the factors that drive disease epidemiology in the *Cakile-Alternaria* interaction.

**Material and Methods**

**Host-Pathogen Association**

*Cakile maritima* (Brassicaceae) is a succulent annual, naturalized to Australia (Rodman 1986), where it grows on sandy to rocky beaches along the southern coast (Thrall et al. 2000; Cousens et al. 2013). The dimorphic seeds have distinctly different dispersal capabilities (Payne and Maun 1981). On ripening, the distal segment separates from the plant and, because of its thick buoyant wall, can disperse long distances via ocean currents. For example, colonization along shorelines is estimated to progress at rates of 48–98 km per year (Rodman 1986). In contrast, the proximal segment of the fruit remains attached to the plant, germinating in the following year or becoming buried and entering the local seed bank. On the southeastern coast of Australia, seedling germination occurs continuously throughout the winter, thus allowing for some overlap between generations (Thrall et al. 2000).

In our study area, populations of *C. maritima* are frequently infected by *Alternaria brassicicola*, a necrotrophic fungus that causes black lesions on leaves, stems, and developing fruits. Field observations and glasshouse studies indicate that resistance to infection is quantitatively based in this system (Thrall et al. 2001, 2005). Disease prevalence and severity is variable, but often all plants in a population are infected by the end of a growing season. *Alternaria brassicicola* readily survives on dead infected stems up to several months (Humpherson-Jones 1989) but has no known sexual cycle (but see Bock et al. 2002, 2003), mean density estimates were obtained for each population by counting all adult plants within 1 × 50-m transects parallel to the shoreline with 4–6 replicates. Splash dispersal of conidia probably accounts for most local spread, although aerial dispersal can occur (Rotem 1994). The occurrence of seed and fruit infection is high, and vertical transmission leads to infection in up to 60% of seedlings germinating from diseased fruits (Oliver et al. 2001). Among-population spread of the pathogen may occur through water dispersal of infected seeds, as the pathogen can survive immersion in seawater on infected seeds for weeks (Oliver et al. 2001). Note, however, that the fruits themselves can stay buoyant and viable after long exposure to seawater (Rodman 1974). The biology of *A. brassicicola* suggests that extended survival of older diseased adult plants or buildup of debris from dead plants may be critical for interseason pathogen persistence.

**Metapopulation Survey**

The coast of New South Wales, between Durras (35°40’S, 150°17’E) and Central Tilba (36°19’S, 150°7’E), consists of an extensive series of sandy to pebbly beaches separated by rocky, often forested headlands or points. This physical structure simplifies studies of within- and among-population disease dynamics by making individual potential host habitats easily identifiable. Furthermore, movement of seeds among potential habitats is likely limited to transport by sea, while pathogen dispersal among host populations is likely to be largely restricted to dispersal of infected fruits. Severe storms can lead to entire plant populations being washed away, and hence subsequent recolonization of beaches by host and pathogen is likely to play an important role in the long-term maintenance of the system (Thrall et al. 2001).

In the winter of 1998 (July), three separate areas were identified along a 120–km stretch of coastline: the Durras (DU), Moruya-Bodalla (MB), and Central Tilba (CT) subregions, tracking north to south along the coast. These subregions were chosen to (a) allow replicated investigations of epidemiological and evolutionary processes across multiple spatial scales, (b) provide areas where the physical structure of the coast was such that individual populations could be readily delineated, and (c) investigate aspects of the physical environment that might relate to disease prevalence and dynamics. Together, the three subregions included a total of 61 potential populations of *C. maritima* and *A. brassicicola*: 23 contiguous beaches in DU, 15 in MB, and 23 in CT. The distance separating the northernmost point of DU from the southernmost point of CT is approximately 83 km.

Beginning in July 1998, all beach populations were censused for disease prevalence approximately every 6 weeks over a period of 5 years, leading to 42 censuses in total (figs. 1, 2). At each census, a minimum of 100 adults and juvenile host plants were haphazardly chosen in each population and scored for the presence/absence of infection (Thrall et al. 2001). All individuals were assessed if the population size was less than 100. We use these data to explore the extent to which physical factors such as wind exposure and beach width influence disease persistence and epidemic development. Because the data set is much more extensive than that presented in our earlier study (Thrall et al. 2001), comprising five seasons of epidemiological information, the current study is not only able to assess the generality of our previous analysis of a single year’s epidemic but also to determine the extent of seasonal variation, the degree to which pathogen populations within subregions are in synchrony, and whether this depends on population connectedness.

At peak growing season in each year (February 1999–2003), mean density estimates were obtained for each *Cakile* population by counting all adult plants within 1 × 50-m transects parallel to the shoreline with 4–6 replicates. Then, using total beach length and population width (the...
average width of the zone within which plants were observed), adult population size was estimated. The total beach length and population width were used to compute the surface occupied by the plant population (SURF). At the low points of annual epidemics, populations were classified as extinct, healthy, or diseased. However, local extinctions and recolonizations were only confirmed if host plants or the pathogen were absent for a minimum of 3 months, that is, three consecutive surveys.

At the beginning of the study, each beach was physically characterized with regard to distance (m) from mean high-tide mark to foredune, distance (m) from foredune to back of beach or the start of permanent vegetation (= shelf width), substrate suitability for Cakile (sand, gravel, shingle; rank scores ranged from 0 [sand] to 5 [rock]), degree of wind exposure (rank scores ranged from 0 [very sheltered] to 5 [very exposed]), aspect (compass direction), access to the sea (beaches ranged from completely open [rank score = 0] to those fronted by exposed rock shelves with no open access [rank score = 4]), beach backing (e.g., steep cliffs, mature forest), and presence/absence of a creek that may affect water movement and possibly allow plants to grow farther from the shore in more sheltered areas. To account for correlations between these variables and assess how metapopulation dynamics were affected by beach physical characteristics, we carried out a Hill and Smith (1976) analysis, equivalent to a principal component analysis mixing continuous and categorical data. The first axis (PC1) of this multivariate analysis ranged from larger and open-to-sea beaches with sandy substrate (negative values) to small and rocky beaches (positive values; figs. S1, S2; figs. S1–S14 are available online) and explained 44% of the variance. The second component (PC2) ranged from beaches exposed to wind (negative values) to sheltered beaches (positive values; figs. S1, S2) and explained 17% of the variance.

In addition, distances between beaches, as well as overall beach lengths, were calculated using 1:6,250 scale aerial photographs (Surveyor-General’s Department, New South Wales) and a curvimeter (Tokyo Sakurai). Interbeach distances were determined by following the coastline, as this is the most relevant measure for waterborne seed

Figure 1: Total plant counts (top) and observed levels of disease prevalence (bottom) over a 5-year period for each of three subregions along the coast of New South Wales: Durras (DU), Moruya-Bodalla (MB), and Central Tilba (CT).
movement. Finally, daily rainfall totals and maximum/minimum temperature readings were obtained from the Australian Bureau of Meteorology for the weather stations closest to each of the three subregions (DU: Batemans Bay [station 69134]; MB: Moruya Heads [station 69018]; CT: Narooma [station 69022]). From these data, monthly cumulative rainfall (variable RAIN; fig. S3) and the average of the maximal temperature (variable Tmax; fig. S3) were obtained.

*Figure 2:* *Cakile maritima* total population sizes (gray) and numbers of diseased plants (black) at the point in each growing season when *C. maritima* population sizes are at their maximum. Data are shown for each of three subregions across a 5-year period; circle size reflects population size.

**Model Overview.** We developed a spatially explicit metapopulation dynamics model for two interacting species. The pathogen depended on plant presence to colonize and develop on a given beach. Its local growth was assumed to be a function of weather conditions, healthy plant density, and potential exchanges among local populations inhabiting the same subregion. In the same way,
the abundance of healthy and infectious plants on a given focal beach were assumed to be influenced by both local and among-population processes. This resulted in estimation of parameters describing the spatial scale at which the metapopulation dynamics were structured but also provided information on the role of environmental conditions experienced by focal and source populations in determining local population dynamics. The model structure is hierarchical, meaning that observed dynamics are assumed to be imperfect observations of the true unobserved dynamics (the process model) as described by the observation protocol (the data model). Recall that the time lag between two observations is approximately 6 weeks.

**Process Model for the Dynamics of Healthy Plants.** We modeled the abundance of healthy plants, \(NH_{i,t}\), on beach \(i\) at time \(t\) as a Poisson distribution with mean \(AH_{i,t}\), depending on the dynamics of the other beaches \(j \in R_i\), where \(R_i\) was the subregion to which beach \(i\) belongs:

\[
\begin{align}
NH_{i,t} | AH_{i,t} & \sim \text{Poisson}(AH_{i,t}), \quad (1a) \\
AH_{i,t} & = \sum_{j \in R_i} m_{ij}^H \times C_{ij} \times [NH_{j,t-1} + (1 - q)NI_{j,t-1}]. \quad (1b)
\end{align}
\]

Here \(NI\) denotes the abundance of infectious plants (for more details, see eq. [4]), and \(q\) describes the probability of vertical transmission (see eq. [4b]).

Spatial dependencies in dynamics among beaches that belonged to the same subregion were accounted for by the parameter \(m_{ij}^H\), an exponentially decreasing function of the coastal distance \(d_{ij}\) between beaches \(i\) and \(j\):

\[
m_{ij}^H = \delta_{ij} \times m_{0ij}^H + (1 - \delta_{ij})(1 - m_{0ij}^H) \\
\times \frac{1}{2 \times D_{ij}^H} \exp\left(-\frac{d_{ij}}{D_{ij}^H}\right), \quad (2)
\]

where \(\delta_{ij}\) is equal to 1 if \(i = j\) and 0 otherwise. The parameter \(m_{0ij}^H\) represents the proportion of nondispersing propagules, whereas \(D_{ij}^H\) is a scale parameter indicating the spatial scale at which metapopulation processes were structured. The parameters \(m_{0ij}^H\) and \(D_{ij}^H\) were allowed to differ among subregions because they were directly affected by metapopulation structure.

Colonization by healthy plants, \(C\), was a log-linear function of habitat characteristics of the source (\(j\)) and focal (\(i\)) beaches and weather conditions occurring in the subregion:

\[
\log(C_{ij,t}) = \alpha_{0ij,year} + \alpha_i \times PC_{i,j} + \alpha_j \times PC_{j,i} + \alpha_{ij} \\
\times PC_{i,j} + \alpha_{ij} \times PC_{j,i} + \alpha_{ij} \times \text{RAIN}_{i,j} \\
+ \alpha_{ij} \times T \max_{i,j}, \quad (3)
\]

where \(\alpha_{0ij,year}\) was a year-specific intercept.

**Process Model for the Dynamics of Infectious Plants.** As for healthy plants, we modeled the abundance of infectious plants on beach \(i\) at time \(t\), \(NI_{i,t}\), as a Poisson distribution with mean \(AI_{i,t}\). However, in this case, \(AI_{i,t}\) was dependent on both the plant and pathogen metapopulation dynamics:

\[
\begin{align}
\frac{NI_{i,t}}{AI_{i,t}} & \sim \text{Poisson}(AI_{i,t}), \quad (4a) \\
AI_{i,t} & = \sum_{j \in R_i} m_{ij}^P \times C_{ij} \times [NI_{j,t-1} + (1 - q)NI_{j,t-1}]. \quad (4b)
\end{align}
\]

In equation (4b), the first term corresponds to disease spread by direct maternal descent (for an evaluation of vertical transmission in the *Cakile-Alternaria* system, see Oliver et al. 2001). These infections were assumed to occur with probability \(q\), the probability of vertical transmission events. The term \(m_{ij}^P\) accounted for spatial dependencies between beach \(i\) and beach \(j\) and was assumed to be equal to \(m_{ij}^H\). The growth rate of a local population of diseased plants, \(C\), was also assumed to be equal between healthy and diseased plants (eq. [3]).

The second term of equation (4b) corresponds to newly infected plants resulting from pathogen spores coming into contact with a healthy plant, that is, horizontal transmission. As for the plant, spatial dependencies among local pathogen populations within the same subregion were accounted for by the parameter \(m_{ij}^P\), an exponentially decreasing function of the coastal distance between populations:

\[
m_{ij}^P = \delta_{ij} \times m_{0ij}^P + (1 - \delta_{ij})(1 - m_{0ij}^P) \\
\times \frac{1}{2 \times D_{ij}^P} \exp\left(-\frac{d_{ij}}{D_{ij}^P}\right), \quad (5)
\]

where \(\delta_{ij}\) is equal to 1 if \(i = j\) and 0 otherwise. The parameter \(m_{0ij}^P\) represents the proportion of nondispersing propagules, whereas \(D_{ij}^P\) is a scale parameter indicating the spatial scale at which metapopulation processes were structured. The parameters \(m_{0ij}^P\) and \(D_{ij}^P\) were allowed to differ among subregions because they were directly affected by metapopulation structure.

Pathogen colonization, \(C^p\), was a log-linear function of weather conditions and the physical area covered by the plant population:

\[
\log(C^p_{ij,t}) = \beta_{0ij,year} + \beta_i \times \text{RAIN}_{i,j} + \beta_j \times T \max_{i,j} + \beta_{ij} \times \text{SURF}_{i,j}, \quad (6)
\]

where \(\beta_{0ij,year}\) is a year-specific intercept. Equation (4b) already takes plant population size into account through the variable \(NH_{i,t}\) and thus the physical area covered by the plant population was used instead of plant density in equation (6).
Data Model. On the beach $i$ at time $t$, only a fraction $(N_{\text{tot}}^{\text{obs}}_i = N_{\text{Hobs}}^{i} + N_{\text{Iobs}}^{i})$ of the total plant population $(N_{\text{tot}}^{i} = N_{\text{H}}^{i} + N_{\text{I}}^{i})$ was observed and classified as healthy $(N_{\text{Hobs}}^{i})$ or diseased $(N_{\text{Iobs}}^{i})$. In addition, at peak growing season in each year, the total plant population size $(N_{\text{tot}})$ was estimated (see “Metapopulation Survey”). As shown in figure S7, a linear relationship was found between $\log(N_{\text{tot}})$ and the proportion of sampled plants at the nearest date when plant abundance was estimated, leading to the following data model for $N_{\text{tot}}^{\text{obs}}$:

\[
\begin{align}
N_{\text{tot}}^{\text{obs}}_i | N_{\text{tot}}^{i}, p_i & \sim \text{binomial}(N_{\text{tot}}^{i}, p_i), \\
\log(p_i) &= a_0 + a_1 \log(N_{\text{tot}}^{i}).
\end{align}
\] (7a) (7b)

Then, the classification of plants according to their infectious status can typically be described by a hypergeometric distribution. Indeed, the hypergeometric distribution gives the probability to observe $N_{\text{Hobs}}^{i}$ healthy plants in a subsample of size $N_{\text{tot}}^{i}$ of a population composed of $N_{\text{H}}^{i}$ healthy and $N_{\text{I}}^{i}$ diseased plants:

\[
N_{\text{Hobs}}^{i} \sim \text{hypergeometric}(N_{\text{tot}}^{i}, N_{\text{H}}^{i}, N_{\text{I}}^{i}),
\] (8)

where $N_{\text{H}}$ and $N_{\text{I}}$ are given by the process model defined by equations (1)–(6).

Parameter Inference. We computed a Bayesian joint posterior distribution for inference of the parameters via a Markov chain Monte Carlo (MCMC) method using the JAGS software (Plummer 2017). Parameter $q$ was not identifiable and was fixed at 0.3 following previous work on the Cakile-Alternaria system (Oliver et al. 2001). Sensitivity of the results to that value was assessed, and except for a very low value of parameter $q$, the results were found robust (see “Supporting Information T4”; “Supporting Information” secs. T1–T4 are in the supplemental PDF, available online). We specified noninformative priors for all the other parameters (for more details, see “Supporting Information T3”) and computed three MCMC-chains of 41,000 iterations. We discarded an adaptation period of 1,000 iterations and a burn-in of 20,000 iterations, and used the remaining 20,000 iterations, thinned by 20, for inference. As assessed by the Gelman and Rubin statistic (Gelman et al. 2004; Flegal et al. 2020), these settings resulted in acceptable convergence for all parameters ($R < 1.1$ for all parameters, multivariate $R = 1.05$, multivariate effective sample size = 2,120).

Evaluation of Model Performances. Goodness of fit was assessed through posterior predictive distributions of replicated data from the posterior distribution of the parameters (Stern and Cressie 2000; Kéry 2010). If the model fits the data well, then the replicated data should be similar to the observed data. This was assessed by computing the proportion of observed data that fall inside the 95% credibility interval (CI) of replicated data, the relative bias, and the relative interquartile distance of replicated data. We also analyzed how these quantities were related to the mean and variance of population size. The robustness of model predictions was assessed by removing data according to different scenarios, reestimating parameters, and predicting the missing data. Seven scenarios were investigated. In scenarios 1, 2, and 3, respectively, 5%, 10%, and 30% of the data were picked at random over time and space. In scenarios 4 and 5, all the data for three beaches in each subregion (representing around 15% of the entire data set) were removed. The three beaches were chosen as a spatial cluster in scenario 4 or at random in scenario 5. In scenarios 6 and 7, all the data for five time steps (representing around 12% of the entire data set) were removed. The five time steps were chosen as a temporal cluster in scenario 6 or at random in scenario 7. For each scenario, 50 data replicates were built. The model was evaluated according to its capacity to predict synchrony in pathogen prevalence at the region-wide scale as well as temporal variation in synchrony. In addition, a pairwise synchrony statistic was used to study synchrony as a function of geographic distance between beaches (see “Supporting Information T1”). In the following, results obtained using the full data set are referred to as scenario 0.

Results

Descriptive Analysis

Plant population dynamics were generally seasonal in all three subregions, with maximal abundances observed at the end of spring (November–December) and minimal abundances observed at the end of winter (fig. 1), which is consistent with the biology of Cakile maritima. However, plant populations were present year-round on some beaches, suggesting extended survival of some individuals. Disease prevalence also demonstrated seasonal dynamics, with disease developing in the plant populations from late spring and high levels of prevalence being maintained until late winter (fig. 1). Metapopulation dynamics appeared to be marked by clear differences in plant abundances and disease levels within subregions and across years, translating into high variability when comparing dynamics among local populations (figs. 1, 2). Synchrony among populations within a subregion was relatively high, likely because plant and pathogen population dynamics were mainly driven by climatic conditions at this scale. However, the synchrony statistic was far from its maximal value of 1, showing that local processes could drive metapopulation dynamics away from perfect synchrony (see “Supporting Information T1”). Region-wide
synchrony varied across years (see “Supporting Information T1”) with a negative correlation from one year to the next. One potential explanation for this pattern is that high levels of synchrony in population dynamics increase the risk of simultaneous local extinctions, leading to amplified differences in population dynamics in the following year.

Local extinctions of plant populations occurred mainly during winter (see “Supporting Information T2”). This pattern was similar among the three subregions. For the pathogen, the probability of extinction started to increase at the beginning of winter and remained elevated until the end of spring. Recolonization of extinct local populations occurred mostly during spring, first by healthy plants then by the pathogen. For CT and MB, there was marked seasonality in recolonization events, whereas for DU the probability of recolonization was more stable through time (see “Supporting Information T2”).

Metapopulation Model

Data Fitting and Robustness of Model Predictions. The Bayesian predictive check revealed a good fit. As exemplified in figure 3, the metapopulation model proved efficient in predicting healthy and diseased plant population dynamics purely from epidemiological observations, environmental variables describing physical characteristics of local habitats, and climatic conditions. When looking at the replicated abundances, 54% (DU), 51% (MB), and 65% (CT) of the observed data for healthy plants and 71% (DU), 76% (MB), and 80% (CT) of the observed data for diseased plants fall inside the 95% CI of predictions. These values reflected the fact that the model was not able to capture the full variability in plant abundances, even if the dynamics are well predicted. In fact, there was a clear relationship between the accuracy of predictions and both the size of the population and its variance: large and highly variable populations were more difficult to predict (fig. S8a, S8b). This was due to the model’s difficulty in capturing the true abundance for elevated peaks (see also fig. 3), leading to a higher bias for such populations (fig. S8d). However, the relative interquartile distances for predictions for large populations were smaller than those for small populations (fig. S8c). Figure 4 (see also figs. S9–S12) compares predictions of synchrony at different scales according to each data removal scenario to synchrony computed on the actual data set. Major differences are obtained in scenario 3, when 30% of the data are removed randomly in space and time. For scenarios 4–6, greater variability among replicates was also observed. In all other cases, the synchrony statistics at all scales are well predicted. This shows that the model was able to reconstruct plant and pathogen dynamics even if a given beach was not included during model inference (fig. 5). Note that synchrony for the subregion DU proved more difficult to predict (figs. 4a, S9).

Spatial Scales of Interactions. Coupling between local plant and pathogen dynamics was quantified by computing the ratio of nondispersing propagules over those arriving via immigration ($m_{i,}/\sum m_{j}$). Coupling in *C. maritima* dynamics between two’ census points (~6 weeks) was low. Across all beaches and subregions, 64%–90% (median, 86%) of observed changes in plant abundance were due to within-beach processes. This result was consistent with the fact that a substantial proportion of the seed produced by plants stays on the home beach (see “Host-Pathogen Association”). In addition, differences were observed across the three subregions, likely reflecting the role of metapopulation spatial structure and physical features in driving exchange among beaches. The importance of local relative to interbeach processes for the exchange of propagules was higher for the Durras and Moruya-Bodalla subregions (the median across beaches was 87% and 86%, respectively) and somewhat lower for the CT subregion (the median across beaches was 67%). Moreover, the comparison of estimated dispersal rates between beaches to a homogeneous scenario where all populations exchange propagules irrespective of distance revealed strong spatial structuring of the plant metapopulation (fig. 6a). This was particularly the case for the CT region, indicating that in this region, local population dynamics are driven by interactions within population clusters. Conversely, for the MB and DU regions, local processes predominated, and the role of other populations within the metapopulation was almost independent of their distance. The Durras subregion was characterized by sheltered and rocky beaches (fig. S2) with high interbeach distances (fig. 3). The Moruya-Bodalla subregion was composed of long, sandy beaches (fig. S2), with interbeach distances comparable to the Central Tilba subregion but with fewer beaches at a distance less than 2 km (fig. 3). These characteristics were consistent with a greater spatial isolation of local populations and a predominance of local processes in the Durras and Moruya-Bodalla subregions.

For the pathogen, across all beaches and subregions, 24%–47% (median, 35%) of the metapopulation dynamics between two census points (~6 weeks) could be attributed to local processes. The contribution of other populations to local dynamics occurred largely independently of distance (fig. 6b), and no clear differences were observed between the three regions. These results clearly indicate that the pathogen dynamics were driven by larger-scale processes than host dynamics.

Impact of Habitat and Environment on Plant Dynamics. All variables had a significant effect on plant population
dynamics (the posterior probability to be either greater than or less than 0 was always higher than 95%; fig. 7). Colonization was more likely, and populations were more persistent on large, sandy beaches ("habitat quality of focal beach"), but seed exchange among beaches was higher for small rocky beaches, probably due to more seeds falling into the water ("habitat quality of source beach"). Beach exposure also played an important role, with sheltered beaches hosting more persistent plant populations ("exposure of focal beach"), while exposed and open-to-sea

Figure 3: Examples of observed (lines) and predicted (shading 95% credibility interval) healthy (gray) and diseased (black) plant dynamics in three randomly chosen beaches in the Durras subregion (b), the Moruya-Bodalla subregion (c), and the Central Tilba subregion (d). The locations of these subregions are displayed in panel a along with the distribution of interbeach distances.
Figure 4: See next page for legend.
beaches favored exchange of plant seeds ("exposure of source beach"). Monthly cumulative rainfall and increases in temperature had negative and positive effects, respectively, on plant population size.

Impact of Habitat and Environment on Pathogen Dynamics. The effect of monthly cumulative rainfall was not significant (95% CI: [-0.014, 0.011]; fig. 7; "Supporting Information T4"). Splash dispersal is important for transmission in this system (Rotem 1994; Chen et al. 2003); however, monthly rainfall was not a good predictor of epidemic increase, probably due to confounding with other negative processes (e.g., increased loss of spores through elevated rain intensity, extreme rainfall washing out plant populations). Conversely, increases in temperature increased disease incidence. The physical area covered by the plant population reduced Alternaria brassicicola colonization ("plant population surface"; fig. 7). This implies that for the same plant abundance, more extended plant populations—that is, decreased density—had lower rates of disease spread.

Discussion
Pathogen metapopulation dynamics are highly variable. Comparison of a number of plant host-pathogen associations involving biotrophic rusts (Linum marginale–Melampsora lini [Burdon et al. 1999; Thrall et al. 2012], Filipendula ulmaria–Triphragmium ulmarnae [Burdon et al. 1995; Smith et al. 2011], Valeriana salina–Uromyces valeriae [Ericson et al. 1999]), mildews (Plantago lanceolata–Podosphaera plantaginis [Laine and Hanski 2006], Lactuca serriola–Bremia lactucae [Lebeda et al. 2008]), and smuts (Silene spp.–Microbotyrum violaceum [Carlsson-Granér 1997; Antonovics 2004]), for which extensive temporal and spatial data are available, provides a clear picture of the temporal and spatial dynamism characteristic of pathogen metapopulations. These studies demonstrate the importance of spatial patchiness in host distribution driving variability in pathogen survival and disease intensity at the individual deme level. A number of generalizations regarding the epidemiology of such systems and consequent effects can be identified, namely, (i) long-term predictability of pathogen presence in the metapopulation as a whole contrasts with marked stochasticity in pathogen presence at the individual deme level; (ii) ephemerality of disease incidence, prevalence, and severity within demes is reflected in temporal and spatial movement in selective pressures across the landscape; and (iii) although host population size influences local pathogen survival, physical differences among sites may generate long-term differences in disease occurrence and severity (Burdon and Thrall 2014).

Figure 4: Estimated synchrony in disease prevalence levels at different spatial and temporal scales. a, Global synchrony for the three subregions surveyed: turquoise = Durras (DU); yellow = Moruya-Bodalla (MB); purple = Central Tilba (CT). b, Temporal variation in synchrony for the subregion CT. c, Pairwise synchrony statistics as a function of interbeach distances for the subregion CT. a, b, Horizontal dashed lines and shading represent, respectively, the synchrony statistic computed on the actual data set along with its bootstrap confidence interval (CI). Closed circles and vertical bars for scenario 0 represent the synchrony and its 95% CI computed on a replicated data set, with the model inferred on the full data set. Boxplots for scenarios 1–7 represent the distribution among the 50 replicates of the posterior median of synchrony. c, Boxplots show the distributions of the pairwise synchrony statistic for the real data (D) and the posterior median of the pairwise synchrony statistic computed on predictions for each scenario. Results for subregions DU and MB are displayed in figs. S9–S12, available online.
Figure 6: Estimations of the plant (a) and pathogen (b) dispersal rates expressed relatively to a homogeneous scenario where all populations exchange propagules irrespective of their distance from one another. An estimated value greater (lower) than 1 indicated more (less) exchange than the homogeneous case. Turquoise = Durras (DU); yellow = Moruya-Bodalla (MB); purple = Central Tilba (CT).

Figure 7: Impact of habitat and weather conditions on host and pathogen colonization ($C$ and $C^*$; eqq. [4], [8]) as indicated by estimated values (posterior 95% credibility interval) of parameters $\alpha$ for the host and $\beta$ for the pathogen. Parameters for the plant are displayed in gray and those for the pathogen in black.
In the current study, we used the interaction between Cakile maritima and Alternaria brassicicola to investigate the ecological and environmental drivers of variation in host and pathogen population dynamics across space and time. In contrast to the studies of biotrophic pathogens referenced above, this system represents a discrete mode of parasitism, necrotrophy, characterized by a substantially different set of life-history features. In this context, perhaps one of the most important of these is the ability of the pathogen to survive on dead infected stems and other host tissue—a feature that tends to buffer the amplitude of population crashes at the end of a growing season and reduce the likelihood of local pathogen extinction. This could also enhance pathogen dispersal via ocean currents. Indeed, the full range of analysis presented here found greater synchrony among pathogen populations than for the host. Hence, the relative importance of local processes in driving population dynamics showed a marked difference between the host and the pathogen. Given the large seed production and dispersal characteristics of C. maritima seed, where approximately 50% of the seed produced remains with the parental plant, it was not surprising to find that greater than 60% of changes in host population size on individual beaches were due to local dynamics. In complete contrast, the dynamics of pathogen populations showed a very high influence of nonlocal processes. The degree of synchrony among populations, both within and between subregions, is an important reflection of the extent to which the populations as a whole are acting as true metapopulations—high levels of synchrony among local sites and across seasons implies both host and pathogen populations are responding in a similar way to environmental parameters. Under these conditions, host populations with above-average disease incidence and severity will always be more affected, leading to stable differences among populations in pathogen-induced selection pressures rather than shifting mosaics of hot and cold spots of pathogen-driven selection (Thompson 1999; Smith et al. 2011).

Environmental drivers—that is, spatial structure of the metapopulation and local habitat quality—were also found to be important in determining plant-pathogen dynamics. First, climatic variables were found to be highly correlated with the plant and pathogen population dynamics, showing that part of the synchrony observed in this system is certainly due to this larger-scale environmental forcing. In addition, the physical structure and spatial location of sites and the resultant level of exposure of either individual host plants (Laine 2006) or host populations as a whole (Burdon et al. 1995) may greatly affect the incidence, severity, and predictability of disease occurrence through time. Within individual demes, host population structure was found to influence disease development. Indeed, for a given plant abundance, more fragmented plant populations displayed lower rates of disease increase during seasons. Such relationships between host population size and density have been detected in many host-pathogen systems, although their occurrence may be affected by pathogen dispersal modes or particular life-history strategies (Burdon and Chilvers 1982). At the metapopulation scale, in the initial study of the first 12 months of the data set, Thrall et al. (2001) found that disease persistence was dependent on the survival of infected plants. Survival was reduced on beaches with greater wind exposure and sea access, where the probability of host population extinction was greater. The more sophisticated analysis of the full 5-year data set presented here reinforces the importance of site quality, with less dense, more dispersed host populations sustaining lower disease levels. More specifically, we found that some large and stable populations generally located in open-to-sea beaches act as stable sources for small, rocky, and exposed populations for which marked extinction-colonization dynamics occur for the plant and, thus, the pathogen. Moreover, while high-quality sources are little impacted by metapopulation dynamics, more ephemeral populations are also crucial sources of plant seeds due to their greater exposure to the sea through storms and less stable substrates, potentially acting as stepping-stones connecting more distant populations. The importance of host availability, landscape features facilitating dispersal, and favorable habitat conditions for the occurrence and spread of disease is typical of host-pathogen systems hosting metapopulations (Laine and Hanski 2006; Ericson et al. 2017; Penczykowski et al. 2018). Interestingly, at a larger among-population scale, studies of the Silene dioica and Microbotryum violaceum (anther smut) host-pathogen system have shown a different picture (Carlsson-Granér and Thrall 2002, 2015). In regions where populations are closer together and more connected, disease is present in all populations, but at lower levels than in areas where there are fewer and more isolated populations. In this last case, however, while many populations are free of disease, the ones that are diseased are diseased at high levels. This particular pattern was, in fact, related to patterns of resistance across the metapopulation.

Genetic variation in natural plant-pathogen interactions is likely to be the rule rather than the exception (Laine et al. 2011; Tack et al. 2012). Pathogen genetic diversity can drive disease emergence and epidemiology (Milgroom and Peever 2003), while variation in host resistance can slow pathogen spread (Thrall and Burdon 2000; Laine 2004). Thus, it is widely accepted that understanding the eco-evolutionary processes that generate and maintain genetic variation in host-pathogen associations is critical for predicting patterns of disease occurrence (Carlsson-Granér and Thrall 2002, 2015; Laine...
One expectation is that building predictive frameworks for disease dynamics requires concrete knowledge of the relative roles of demography, environment, and genetic variation in driving disease outcomes (Barrett et al. 2009), and there have been numerous calls for studies of disease that explicitly integrate these factors in both plant (Burdon and Thrall 2014; Tack and Laine 2014; Penczynowski et al. 2015; Fraile et al. 2017) and animal (Savage et al. 2015; Dellicour et al. 2016) host-pathogen interactions.

Particularly in natural systems, characterization of the genetic interaction is generally thought to be crucial in understanding and predicting disease dynamics. For example, this was the case in the *L. marginale*–*M. lini* host-pathogen interaction, where studies have found extensive variation among local populations with regard to the diversity of resistance and infectivity phenotypes and genotypes (Burdon and Jarosz 1992; Burdon et al. 1999; Thrall et al. 2002, 2012) and clear impacts on epidemiological (Thrall and Burdon 2000) and coevolutionary (Thrall et al. 2012) outcomes. Theoretical studies of the interaction between *Silene dioica* and *Microbotryum violaceum* (anther smut) predicted that regional-scale patterns of disease could be explained only by the inclusion of variable host resistance in models (Carlsson-Granér and Thrall 2002); this prediction was later confirmed experimentally (Carlsson-Granér and Thrall 2015).

However, as shown clearly by our analysis of the *Cakile-Alternaria* system, it is possible to reconstruct disease dynamics without any knowledge of genetic variation in traits underlying the host-pathogen interaction. In this case, this could be for several reasons. First, *A. brassicicola* demonstrates important dispersal abilities both in space and time due to survival on dead plant material and through vertical transmission, and *C. maritima* is also a good disperser via marine currents. In such situations, theoretical studies on the coevolutionary dynamics of plant-pathogen interactions predict low genetic differentiation among local plant populations and consequent selection for more generalist pathogen genotypes, even if the pathogen has an advantage in terms of dispersal ability (Gandon et al. 1996; Gandon 2002; Papaïx et al. 2014). Moreover, the three subregions we studied showed marked metapopulation dynamics with frequent local extinctions and recolonization by adjacent populations, likely exacerbated by the fact that *C. maritima* is an annual, disrupting local coevolutionary patterns. In contrast, the perennial *L. marginale* has only limited dispersal ability, and local host extinctions are infrequent, which may contribute to the observed strong patterns of local adaptation (Thrall et al. 2002). Perhaps another crucial difference between these systems is that resistance appears to be quantitative in the *Cakile-Alternaria* interaction but largely determined by major genes in the *Linum-Melampsora* interaction. Because different pathogen genotypes can always infect hosts, at least to some extent, in the former system, this would likely blur epidemiological differences among local demes, even if there was some genetic structure. Overall, the contrast between these systems could potentially explain why little additional predictive information might be gained from the inclusion of genetic data for the *Cakile-Alternaria* association. This is further confirmed by different studies on the *Cakile-Alternaria* association showing moderate levels of variation in genetic diversity with no spatial patterns or clear evidence for genetic structure (Thrall et al. 2000; Bock et al. 2005; Linde et al. 2010). In addition, Ohadi et al. (2016) found evidence for genetic differentiation among *Cakile* populations but over larger scales than the current study. Hence, genetic data are in some way equivocal but not inconsistent with the hypothesis developed here.

Finally, our results show that by collecting extensive data on host and pathogen population dynamics, followed by the development of spatially explicit epidemiological models, it is possible to accurately predict disease dynamics, even when there is extreme variability in host population dynamics. It also suggests that there could be value in articulating a more general framework for distinguishing situations which really do require genetic information for predicting demographic dynamics.

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**Statement of Authorship**

J.P., L.G.B., P.H.T., and J.P. conceptualized this work. J.P. developed the model. J.J.B., L.G.B., and P.H.T. collected the data. J.P. and E.W. analyzed the model and coded the simulations. All authors analyzed the data and interpreted the results. J.P. wrote the original draft. All authors reviewed and edited the manuscript.

**Data and Code Availability**

Data have been deposited in CSIRO (http://hdl.handle.net/102.100.100/366494?index=1; Barrett 2020).

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Left, typical landscape of the coast of New South Wales, Australia, between Durras and Central Tilba, consisting of extensive series of sandy to pebbly beaches separated by rocky, often forested, headlands or points. Clumps of Cakile maritima are visible, some of them being diseased. Right, black spots on C. maritima stems due to infection by Alternaria brassicicola. Photos: Peter H. Thrall.