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Linking winter conditions to regional disease dynamics in a wild plant–pathogen metapopulation

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Summary

- Pathogens are considered to drive ecological and evolutionary dynamics of plant populations, but we lack data measuring the population-level consequences of infection in wild plant–pathogen interactions. Moreover, while it is often assumed that offseason environmental conditions drive seasonal declines in pathogen population size, little is known about how offseason environmental conditions impact the survival of pathogen resting stages, and how critical the offseason is for the next season's epidemic.
- The fungal pathogen *Podosphaera plantaginis* persists as a dynamic metapopulation in the large network of *Plantago lanceolata* host populations. Here, we analyze long-term data to measure the spatial synchrony of epidemics and consequences of infection for over 4000 host populations. Using a theoretical model, we study whether large-scale environmental change could synchronize disease occurrence across the metapopulation.
- During 2001–2013 exposure to freezing decreased, while pathogen extinction–colonization–persistence rates became more synchronized. Simulations of a theoretical model suggest that increasingly favorable winter conditions for pathogen survival could drive such synchronization. Our data also show that infection decreases host population growth.
- These results confirm that mild winter conditions increase pathogen overwintering success and thus increase disease prevalence across the metapopulation. Further, we conclude that the pathogen can drive host population growth in the *Plantago–Podosphaera* system.

Introduction

Pathogens are numerous, diverse and a functionally significant component of wild plant communities. They have been shown to promote plant diversity and to act as important drivers of community composition (Friess & Maillet, 1996; Borer *et al.*, 2007; Bradley *et al.*, 2008; Allan *et al.*, 2010; Bagchi *et al.*, 2014). Diseases are believed to have strong negative effects on the growth, development and reproduction of host plants (Brown & Tellier, 2011), and these negative fitness consequences are at the heart of coevolutionary theory proposed for explaining how diversity is maintained in host–pathogen interactions (Jayakar, 1970; Leonard, 1977; Frank, 1993; Sasaki, 2000; Bergelson *et al.*, 2001). However, long-term data on the frequency and consequences of infection for host populations are lacking for wild plant–pathogen associations (Burdon & Thrall, 2009, 2014). Such data are essential for quantifying the influence that pathogens have on the growth and evolution of plant populations.

Available data from wild pathosystems suggest that diseases are typically persistent on the regional scale (Seabloom *et al.*, 2010), even when ephemeral in individual plant populations (Smith *et al.*, 2003; Antonovics, 2004; Burdon & Thrall, 2014; Jousimo *et al.*, 2014). Across pathosystems, the proportion of populations with disease ranges between 1% and 70%, with high rates of pathogen colonization and local extinction leading to a frequent turnover of pathogen populations and large fluctuations in prevalence from year to year (*Silene latifolia–Microbotryum violaceum*, Thrall & Antonovics, 1995; *Filipendula ulmaria–Triphragmium ulmariae*, Smith *et al.*, 2003; *Plantago lanceolata–Podosphaera plantaginis*, Laine & Hanski, 2006; Soubeyrand *et al.*, 2009; and *Valeriana salina–Uromyces valerianae*, Ericson *et al.*, 1999). If pathogen colonization–extinction events occur asynchronously across populations, this may translate into spatially heterogeneous selection pressure on hosts and their pathogens, yielding hotspots and coldspots of coevolution across the region (Thompson, 1999, 2005; Smith *et al.*, 2011). Conversely, spatially synchronous disease dynamics should yield larger clusters of simultaneously infected populations and thus, more homogeneous selection on the host and pathogen across the region. However, little is known about how synchronous the colonization–extinction dynamics of plant pathogens are across space, or how environmental factors drive changes in synchrony over time.

In general, spatial synchrony in disease dynamics is expected when large-scale events, such as weather, overwhelm local stochasticity and heterogeneity in, for example, pathogen survival and reproduction (Grenfell *et al.*, 2001). Thus, a change in climatic

conditions during the offseason (i.e. the period between the growing seasons) may increase region-wide synchrony in pathogen survival from one year to the next. A decline in pathogen population size is typically associated with the offseason stage (Burdon & Elmqvist, 1996; Harvell *et al.*, 2002; Altizer *et al.*, 2006; Barrera *et al.*, 2013). However, the effects of offseason climatic conditions on pathogen survival depend on host and pathogen life-histories. Some host-specific pathogens can continue growing during the offseason because their host plants persist year-round (e.g. *Empetrum hermaphroditum–Arwidssonia empetri* in the tundra of northern Sweden; Olofsson *et al.*, 2011). Other pathogens survive the offseason on a 'green bridge' provided by alternative hosts (Agrios, 2005). In systems where parasites cannot grow or persist systemically within their hosts during the offseason, the production of specialized resting structures is a common strategy for survival (Combes, 2001; Agrios, 2005). For these pathogens, variation in the survival of resting structures may be a key driver of disease ephemerality at the population level (Tack & Laine, 2014). In temperate regions, survival of pathogen resting structures over the winter offseason may depend in part on average winter temperatures and snowfall. These are also the climatic variables undergoing the most dramatic changes at higher latitudes (IPCC, 2014). Because climate change is occurring over a large spatial scale, those changes that impact pathogen survival during the offseason might synchronize the persistence of pathogen populations and the prevalence of disease across regions.

Given how dynamic and variable the frequency of infection appears to be at the population and metapopulation levels, it is perhaps not surprising how little is known about the consequences of infection for growth rates of wild plant populations. To date, the best evidence of population-level consequences of fitness loss due to infection comes from agricultural settings, where the negative effects of infection on the growth and yield of host plants are well established (Agrios, 2005). Available studies documenting the fitness consequences of infection in wild plant populations demonstrate that infection can reduce the overwinter survival of infected plants (Jarosz & Burdon, 1992), decrease seedling survival and production of viable seed (Ericson *et al.*, 2002; Springer, 2009) and cause host sterilization, which can reduce the host population growth rate (Alexander & Antonovics, 1988). To date, evidence supporting pathogens as having significantly negative effects on wild host populations remains highly mixed (as reviewed in: Jarosz & Davelos, 1995; Alexander, 2010). While there are several long-term studies of plant–pathogen interactions in the wild (e.g. Ericson *et al.*, 1999; Antonovics, 2004; Smith *et al.*, 2011), investigations of how pathogens impact their hosts are typically carried out under experimental settings, or as snapshots in a single population and/or season, or focus on annual or short-lived perennial hosts. Hence, they yield little insight into how pathogens regulate host population dynamics across regional scales and multiple years. Especially for perennial hosts, the effects of infection on offseason survival or seed production are not evident in one growing season. Thus, to understand how disease impacts host population growth rates, it is imperative to measure the consequences of infection across multiple populations and years.

In this study, we analyze long-term epidemiological data to identify the link between offseason conditions and disease dynamics, and the consequences of infection for wild populations of ribwort plantain, *P. lanceolata*, which hosts the powdery mildew fungus *P. plantaginis*. The host grows in a network of *c.* 4000 meadows, which have been surveyed for mildew presence/absence at the end of the growing season annually from 2001 to 2013. Previous studies have demonstrated that regionally *P. plantaginis* persists as a metapopulation through frequent extinctions and colonizations of local host populations, with infection typically persisting for only 1–2 yr within a given host population (Jousimo *et al.*, 2014). The spatial configuration of the pathosystem is critical for how infection dynamics play out regionally, and some of the variation in colonization dynamics is explained by precipitation during the growing season (Laine & Hanski, 2006; Jousimo *et al.*, 2014). Because the host plant dies back to rootstock during winter, the pathogen can only survive the winter offseason by producing specialized resting structures (Tack & Laine, 2014). A high rate of extinction of local pathogen populations during the offseason keeps disease prevalence low at the metapopulation level, yet factors affecting extinction dynamics remain poorly understood (Jousimo *et al.*, 2014; Tack & Laine, 2014). In particular, climatic conditions during the growing season do not explain pathogen extinction rates (Jousimo *et al.*, 2014). In this study, we ask: do environmental conditions during the offseason explain trends in extinction rate and disease prevalence at the metapopulation level? Has the degree of synchrony in disease extinction and colonization rates across the study area changed through time? If so, using a modeling approach we ask: can changes in region-wide spatial synchrony of disease be driven by environmental change affecting the offseason survival of the pathogen? Previously, Laine (2004) established that the pathogen can decrease host density at small spatial scales, but the impact of infection on host fitness depends on environmental conditions. Thus, we also ask: what are the consequences of disease for annual host population growth, measured over all populations across a time series of 11 yr? This investigation of the frequency and consequences of pathogen infection in wild plant populations features thousands of populations surveyed each autumn for more than a decade (Fig. 1).

Materials and Methods

Study system

The host plant *Plantago lanceolata* L. (ribwort plantain) is a monoecious, iteroparous, perennial herb that grows as a rosette (Sagar & Harper, 1964). Plants can live up to 7 yr, with older individuals suffering higher mortality during times of environmental stress (Roach *et al.*, 2009). *Plantago lanceolata* is an obligate outcrosser with wind-dispersed pollen (Ross, 1973). In addition to reproducing sexually, the plant can propagate asexually via clonally produced side-rosettes. *Plantago lanceolata* has a cosmopolitan distribution but is at its range margins in the study area in the Åland archipelago southwest of Finland. There its distribution is fragmented into local small patches that typically

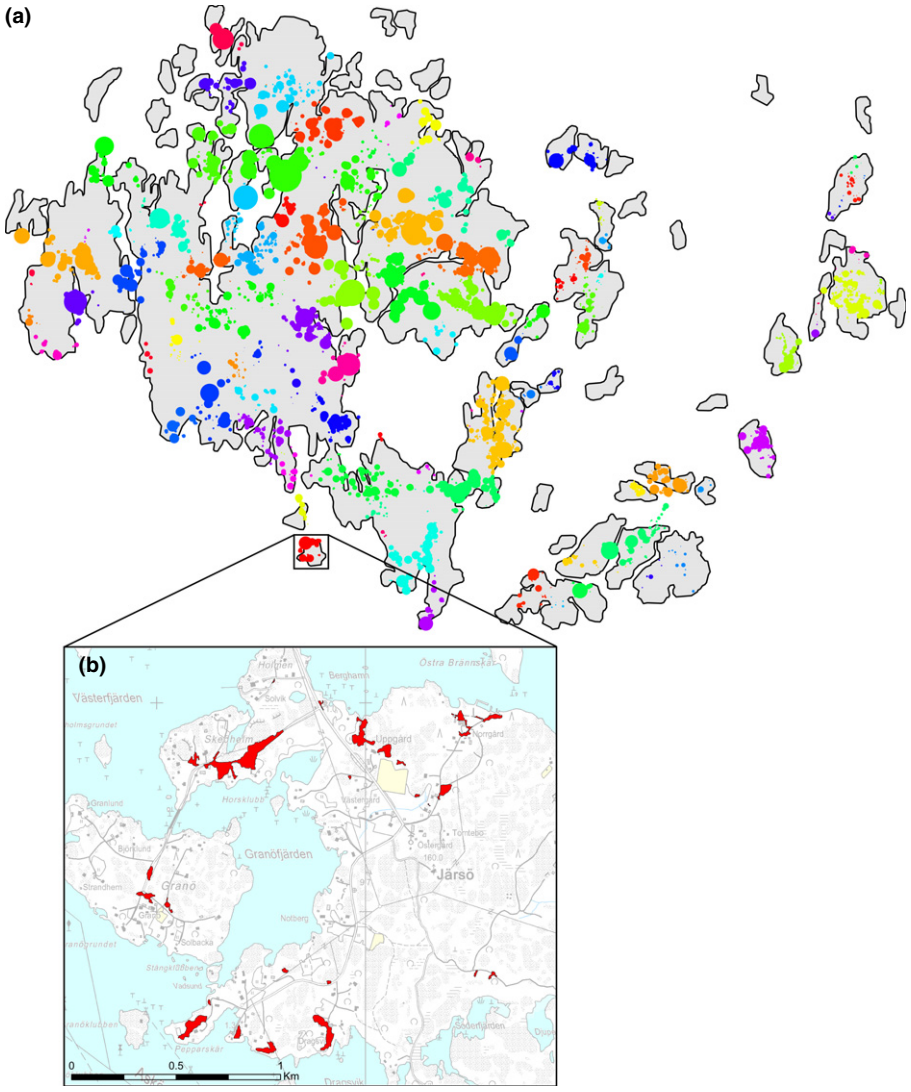

Fig. 1 Maps of the spatial scales of our study. (a) Åland archipelago, with *Plantago lanceolata* host populations (circles) colored according to their semi-independent network (SIN) grouping (117 SINs pictured here). The size of each marker circle is scaled relative to the area of the host population. (b) An example SIN, where *P. lanceolata* populations within that SIN are colored red.

occur on dry meadows or pastures (Ojanen *et al.*, 2013). In Åland *P. lanceolata* is naturally infected by *Podosphaera plantaginis* (Castagne; U. Braun & S. Takamatsu), a powdery mildew fungus in the order Erysiphales within the Ascomycota (Yarwood, 1978). It is an obligate specialist pathogen dependent on living host tissue. During the growing season, *P. plantaginis* grows on the plant surface, and only its haustoria penetrate the epidermis (Supporting Information Fig. S1). Chains of wind-dispersed spores (conidia) grow vertically from the leaf surface, and disease transmission can occur if these infectious propagules encounter a susceptible host, under suitable environmental conditions. The pathogen survives over the winter offseason through specialized resting structures (chasmothecia) produced on the leaf surface as the host dies back to rootstock (Tack & Laine, 2014). From late autumn to spring, typically eight ascospores develop and mature inside resting structures (Tollenaere & Laine, 2013). The resting structures burst open in spring, and released ascospores can infect living plant tissue (Tack & Laine, 2014). In contrast to seed banks of the host plant, powdery mildew resting structures are likely not to be viable for more than one season of dormancy (Braun *et al.*, 2002). In the interaction between *P. lanceolata* and *P. plantaginis*, disease resistance is strain-specific with the same host genotype blocking infection by some strains of the pathogen while being susceptible to others (Laine, 2007), and there is considerable variation among both host and pathogen genotypes and populations in their resistance and infectivity, respectively (Laine, 2004, 2008).

Surveys of disease in wild populations

The interaction between *P. lanceolata* and *P. plantaginis* is highly amenable to large-scale ecological studies as infection is conspicuous in the field (Fig. S1) and the disease cycle lacks extended latency periods. Hence, field surveys at the end of epidemics yield a direct measure of the host population size and disease prevalence. The metapopulation of *c.* 4000 *P. lanceolata* populations in the Åland Islands has been surveyed at the end of each growing season (September) from 2001 to 2013 (Fig. 1a). We used annual data on host plant population size (*P. lanceolata* coverage, m^2; not measured in 2009–2010) and mildew presence or absence to investigate variation in the frequency of pathogen infection and

consequences of disease for host population growth over the entire 50 × 70 km metapopulation and 13 yr study period. At the beginning of the growing season (July) in 2012 and 2013, we resurveyed all populations in which mildew was present in the preceding autumn. As July coincides with the onset of the epidemic, pairing these data with the September data allowed us to measure the effects of mildew presence on the within-season host population growth. Full details of the methodology and variables measured in the annual large-scale survey can be found in Laine & Hanski (2006) and Ojanen *et al.* (2013), and we refer to Tack & Laine (2014) for additional details of the July surveys.

We obtained records of ambient temperature and snow depth measured daily from 2000 to 2013 at the Jomala weather station in the Åland Islands (19.98633°E, 60.17836°N). Using these data, we calculated a yearly metric of pathogen 'exposure to freezing' as the number of days between 1 October and 30 April in which the minimum air temperature was < 0°C and the snow depth was < 5 cm. We assumed that this would be a biologically meaningful index of the harshness of environmental conditions for the pathogen during the offseason, since freezing temperatures may be detrimental to mildew survival (Spencer, 1978; Tiwari *et al.*, 1999), but a thick layer of snow could serve as protective insulation, as has been suggested for other fungal pathogens (e.g. dwarf bunt, Peterson *et al.* (2009), or stripe rust, Kumar *et al.* (2013), of winter wheat).

Analysis of infection frequency

Throughout this study, we define disease prevalence as the proportion of populations with mildew infection (i.e. prevalence at the metapopulation level). The disease prevalence data were normally distributed and homoscedastic and did not require transformation. We used Pearson correlations to test for temporal trends in metapopulation-level disease prevalence and exposure to freezing over the 2001–2013 time series. We then correlated the metric of exposure to freezing with disease prevalence and pathogen extinction rate (the fraction of infected populations in September of year_t which were uninfected in September of year_{t+1}).

Analysis of consequences of disease for host population growth

We tested for the effects of mildew infection on the annual relative host population growth, using a linear mixed effects model with first order autoregressive error structure (package 'nlme'; R Core Team, 2013). This analysis allowed us to account for temporal autocorrelation between sequential measurements of the host population growth. We quantified the annual relative growth rate of a host population as the change in $\log_e$-transformed *P. lanceolata* coverage, N (in m^2), from September of year_t to September of year_{t+1} as:

$$\log N_{t+1} - \log N_t, \qquad \text{Eqn 1}$$

where host coverage data were available for years 2001–2008 and 2011–2013. The model included fixed effects of pathogen presence in year_t, year_t (a continuous variable), a metric of host density calculated as the log-transformed ratio of *P. lanceolata* coverage to total area of the population (i.e. the proportion covered by *P. lanceolata*) and their interactions. The host population was modeled as a random effect. Using a similarly structured model, we tested for the effects of pathogen presence in July, year (a categorical variable), and host density on the host population growth from July to September in years 2012 and 2013. In both analyses, we used likelihood ratio tests to compare models with and without the pathogen × density, pathogen × year, and density × year interaction terms, and removed nonsignificant interaction terms from the models.

Spatial synchrony analysis

We analyzed the extent of spatial synchrony in disease occurrence among clusters of populations across the study region (Fig. 1a,b). Host populations were classified into semi-independent networks (SINs) using a hierarchical cluster analysis (HCA; Sneath & Sokal, 1973) in the software SPOMSIM (Moilanen, 2004). This involved building a tree structure of the populations using HCA, and then cutting the tree at a specific level to yield a network classification (Moilanen, 2004). The HCA tree structure was based on an index of host population connectivity, S_i^{L} (index L for landscape connectivity), which takes into account the sizes and spatial locations of host populations. This index is defined as:

$$S_i^{\mathrm{L}} = \sum \exp(-\alpha d_{ij})\sqrt{A_j}, \qquad \text{Eqn 2}$$

where d_{ij} is the Euclidian distance between patches i and j and α is the parameter of the negative exponential dispersal kernel, which was set to $1\ \mathrm{km}^{-1}$ based on the degree of aggregation of infection observed in the field (Laine & Hanski, 2006). A_j is the area of habitat patch j, which was square root transformed because this approximates the scaling of host population size with patch area, and because the emigration rate of the mildew can be assumed to be proportional to the host population size (Laine & Hanski, 2006). To study how the degree of synchrony estimated for the metapopulation depended on the size of the networks used in the analysis, we constructed three identical trees of all patches in the metapopulation, and cut each tree at a different level to produce 40, 89 or 117 SINs. In addition, we classified the populations into the 15 municipalities of Åland to serve as the largest spatial units (Fig. S2). Synchrony in extinction–colonization dynamics of the pathogen in 2001–2013 at the region-wide scale, its spatial structure and its temporal variation, were analyzed using extensions of the synchrony statistic of Loreau & de Mazancourt (2008) and semi-variograms (Chilès & Delfiner, 1999; Stein, 1999). The synchrony statistic φ was introduced by Loreau & de Mazancourt (2008) as a measure of community-wide synchrony that circumvents a drawback from the average correlation coefficient commonly used in synchrony studies (Bjørnstad *et al.*, 1999). For each of the classifications of 40, 89 and 117 SINs, or municipalities $i \in \{1, \ldots, I\}$, at each year $t \in \{2001, \ldots, 2013\}$, one observes the proportion $x_i(t)$ of patches occupied by powdery mildew among a set of $n_i(t)$

observed patches that depends on the SIN and the year. Let $x(t) = \sum_{i=1}^{I} x_i(t)$ be the sum over the whole region of these proportions. When there is perfect asynchrony between SINs (or no fluctuations), the temporal variance σ^2 of $x(2001), \ldots x(2013)$ is 0. When there is perfect synchrony, σ^2 is maximal and equal to $\left(\sum_{i=1}^{I} \sigma_i\right)^2$ where σ_i is the temporal SD of $x_i(2001), \ldots, x_i(2013)$. The region-wide synchrony statistic is defined by $\varphi = \sigma^2 / \left(\sum_{i=1}^{I} \sigma_i\right)^2$, and varies between 0 (perfect asynchrony) and 1 (perfect synchrony).

To study whether region-wide synchrony has changed during the study period, we computed φ separately with the 2001–2007 data and with the 2007–2013 data. To assess the significance of the difference between these time periods, we applied a randomization technique (Manly, 1997): we independently drew 10 000 sets of 7 yr among 2001–2013, we computed for each set of years the corresponding synchrony statistic, and we then compared the observed φ values with the distribution obtained by randomization.

To study synchrony as a function of geographic distance between SINs or municipalities, we transformed the synchrony statistic φ measured over the whole archipelago into a pairwise synchrony statistic. For each pair (i,j) of SINs or municipalities, we computed the pairwise synchrony statistic $\varphi_{ij} = \tau_{ij}^2 / (\sigma_i + \sigma_j)^2$, where τ_{ij}^2 is the temporal variance of $x_i(t) + x_j(t)$, and plotted it against the distance between the centroids of i and j. To study the temporal variation of the spatial structure of the synchrony, we separately computed the pairwise synchrony statistics with the 2001–2007 data and with the 2007–2013 data, and plotted them against geographic distances.

Spatial autocovariance (or autocorrelation) is commonly used to measure spatial dependence in variables observed across space. It has been used specifically to describe the spatial structure of synchrony in ecological studies (e.g. see Bjørnstad *et al.*, 1999; Williams & Liebhold, 2000). For a spatial process whose mean is not constant, the semi-variogram used in geostatistics is an alternative and more appropriate tool to measure spatial dependence (Chilès & Delfiner, 1999; Chapter 2). To relax assumptions about the mean of the proportions $x_i(t)$ of patches occupied by powdery mildew, we used semi-variograms instead of autocovariance functions. We computed the semi-variograms for each year of the study period by following the approach of Walker *et al.* (2008; based on Monestiez *et al.*, 2006) who propose an estimation of semi-variograms for empirical proportions.

Theoretical model of spatial synchrony over time

We built a simple theoretical model to qualitatively investigate the conditions under which the synchrony pattern of a pathogen may evolve in the same way as in our case study. Our model is a stochastic patch occupancy model (SPOM; Ovaskainen & Hanski, 2004) that represents the spatially explicit dynamics of a metapopulation, including an offseason where pathogen extinction may occur and a growing season where colonization of uninfected patches (i.e. host populations) may occur. Full details of the model and methods for simulation and analysis are provided in Methods S1. Briefly, the model incorporates three key elements: spatial heterogeneity of pathogen survival during the offseason (i.e. there are places of the study area where survival is more likely; Soubeyrand *et al.*, 2009); homogeneous dispersal during the growing season (i.e. dispersal of pathogen particles released from infected patches does not depend on patch locations); temporal variation in survival probabilities and infection strengths of pathogen populations (i.e. yearly environmental effects are applied as multiplicative factors to survival probabilities and infection strengths of all pathogen populations in the study area).

At the beginning of the model simulation (year $t = 0$), 2000 uniformly distributed host patches are randomly infected with equal probability across space. Survival of a pathogen population between growing seasons depends on the location of the patch and an offseason environmental effect. Colonization of uninfected patches during growing seasons depends on the distance to patches where the pathogen has survived and on a growing-season environmental effect. The offseason effect is the sum of a temporal trend and a yearly random effect. Its expected value is constant for the first 20 yr of the simulation, but after year $t = 20$ the environmental effect increases linearly such that conditions for pathogen survival are increasingly favorable over time. The growing-season effect is a simple yearly random effect without trend. Figure S3 shows, for one simulation, the states of the metapopulation at years 10 and 30, after the offseason and after the growing season. It also shows the temporal variation in the yearly environmental effects.

We performed 200 simulations of the model. For each run, we simulated the model up to year $t = 30$. The states of the metapopulation from year 0 to 9 were discarded (burn-in) since the simulations were started at year 0 from an arbitrary state. Then, we separately considered the period from year 10 to 20 and the period from year 20 to 30 to assess the evolution of synchrony. The region-wide synchrony statistic φ and the pairwise synchrony statistics φ_{ij} were defined in the Spatial synchrony analysis section (above) at the resolution of clusters of patches (e.g. SINs or municipalities in our case study). In the simulation study, these clusters were defined by partitioning the spatial domain into 100 cells, and assigning the 2000 patches to the cells in which they were located (see Fig. S3). Thus, we defined 100 cells of patches, and the synchrony statistics were computed using the proportions of infected patches after the epidemic seasons in each of the 100 cells.

Results

The high turnover rate of the pathogen is reflected in the frequency histogram of disease persistence, with the majority of local pathogen populations having persisted for only 1–2 yr (Fig. S4). At the metapopulation level, the prevalence of powdery mildew infection increased over the study period from 2001 to 2013 ($r = 0.79$, $P = 0.001$; Fig. 2a). This trend reflected a significant decrease in the pathogen extinction rate ($r = -0.64$, $P = 0.026$)

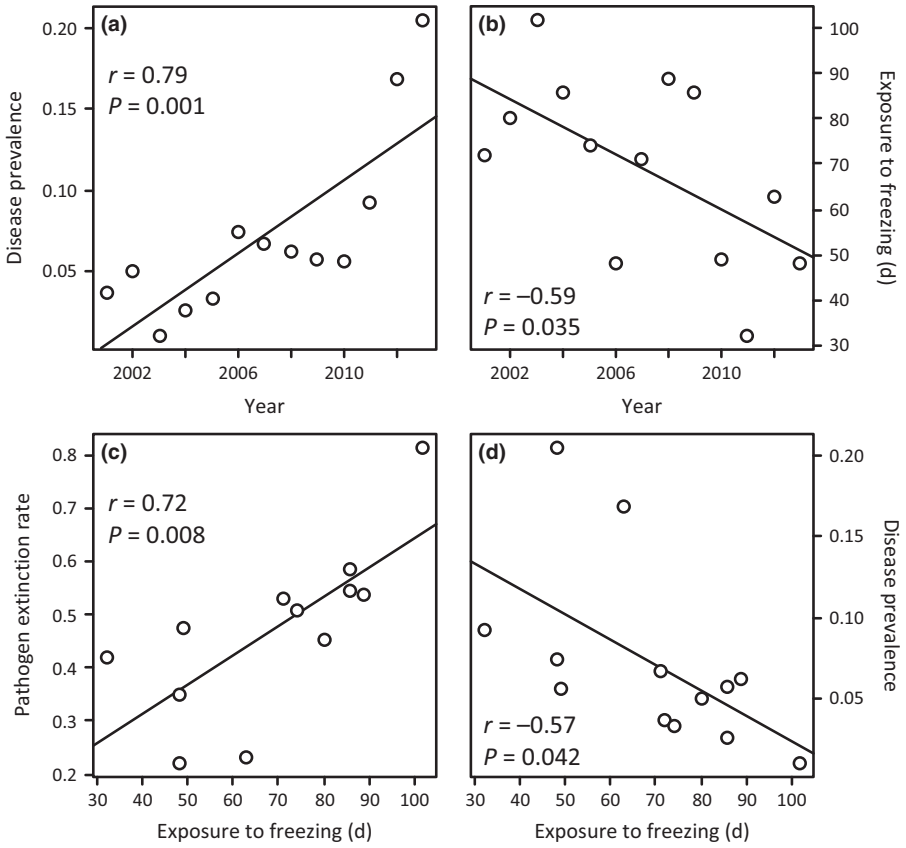

Fig. 2 Trends in disease prevalence and winter conditions at the metapopulation level. (a) Prevalence of *Podosphaera plantaginis* infection among *Plantago lanceolata* populations increased over the study period from 2001 to 2013. (b) At the same time, there was a decrease in pathogen exposure to freezing temperatures; that is, in the number of days per winter when temperatures are below freezing and the depth of the snow cover is < 5 cm. (c) Pathogen extinction rate was significantly higher following winters with greater exposure to freezing. (d) Disease prevalence was lower following winters with greater exposure to freezing.

and a significant increase in the pathogen colonization rate ($r = 0.75$, $P = 0.0005$) during the study period. Exposure to freezing conditions also declined substantially over the 13 yr ($r = -0.59$, $P = 0.035$; Fig. 2b); that is, there was a decrease in the number of days with freezing air temperatures but without a thick layer of snow. This metric of exposure to freezing was significantly correlated with higher rates of pathogen extinction from patches ($r = 0.72$, $P = 0.008$; Fig. 2c). Although exposure to freezing did not significantly correlate with the pathogen colonization rate ($r = -0.54$, $P = 0.072$), there was still a significant negative relationship between winter conditions and disease prevalence at the end of the growing season ($r = -0.57$, $P = 0.042$; Fig. 2d). By contrast, when considered separately, neither the number of freezing days nor the number of snowless days was significantly correlated with pathogen extinction (freezing days: $r = 0.11$, $P = 0.74$; snowless days: $r = 0.40$, $P = 0.19$) or prevalence (freezing days: $r = 0.05$, $P = 0.88$; snowless days: $r = -0.43$, $P = 0.14$).

Across the study system, spatial synchrony of pathogen prevalence among SINs of populations was higher over the latter half of the study period in 2007–2013 than over the first half in 2001–2007 (Fig. 3a). This measure of region-wide synchrony decreased with increased spatial resolution of the analysis (Fig. 3a). The index of pairwise synchrony decreased with distance between SINs, and this decrease was steeper during the period 2001–2007 than during the period 2007–2013 (illustrated for 117 SINs in Fig. 3b; similar graphs at lower spatial resolutions are provided in Fig. S5), corroborating the analysis of the region-wide synchrony (Fig. 3a). The spatial structure of synchrony and its variation in time was also analyzed with semi-variograms presented in Fig. S6. However, because the variances of the proportions of occupied populations fluctuated over time, these semi-variograms (Fig. S6) cannot be compared over time periods without bias.

Simulations of the theoretical model showed that a temporal increase in a large-scale environmental driver promoting host overwintering success could drive a qualitatively similar pattern to our empirical synchrony results. In these simulations, the region-wide synchrony statistic (φ) was much greater when computed for the second half compared with the first half of the study period (years 20–30 vs 10–20; Fig. 4a). In addition, at a given distance separating clusters of populations, pairwise synchrony statistics (φ_{ij}) were greater in the second compared with the first half of the study period (Fig. 4b). Qualitatively, Fig. 4 presents patterns of synchrony statistics that are similar to those obtained in the case study (Fig. 3). Thus, these modeling results show that an increase in the favorability of the environmental conditions for offseason pathogen survival (e.g. the decreased exposure to freezing observed in our field study) may induce an increase in the synchrony of disease dynamics.

Disease significantly decreased the relative growth rate of host populations, quantified as a change in log-transformed *P. lanceolata* coverage (m^2), both between and within years (Fig. 5). Overall, the annual population growth rates decreased through time and were significantly lower in infected populations (Table 1; Fig. 5a). Growth rates were also significantly lower for

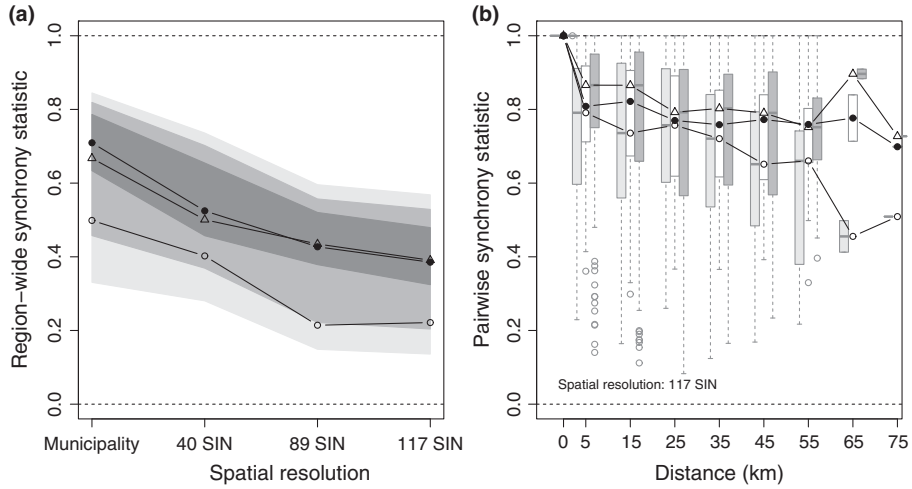

Fig. 3 Spatial synchrony of *Podosphaera plantaginis* infection in the *Plantago lanceolata* metapopulation over the 13-yr study period. (a) Region-wide synchrony statistic as a function of the period (2001–2013, closed circles; 2001–2007, open circles; 2007–2013, triangles) and the spatial resolution from the lowest (15 municipalities) to the highest (117 semi-independent networks, SINs) resolution taken into account. The gray palette gives quantiles of the distribution of the region-wide synchrony statistic computed over 7 yr uniformly randomly chosen (the quantiles are, from bottom to top, 0.025, 0.10, 0.25, 0.75, 0.90 and 0.975). (b) Distributions (boxplots) of the pairwise synchrony statistic as a function of geographic distance between SIN centroids (distances d are grouped in classes of distances: $d = 0, 0 < d \leq 10, 10 < d \leq 20, \ldots, 70 < d \leq 80$). The pairwise synchrony statistic is computed over the periods 2001–2013 (white boxplots), 2001–2007 (light gray boxplots) and 2007–2013 (dark gray boxplots). Whiskers extend to the most extreme data point which is ≤ 1.5 times the interquartile range from the box. Black dashed lines show the evolution of the median of the pairwise synchrony statistic for each data period.

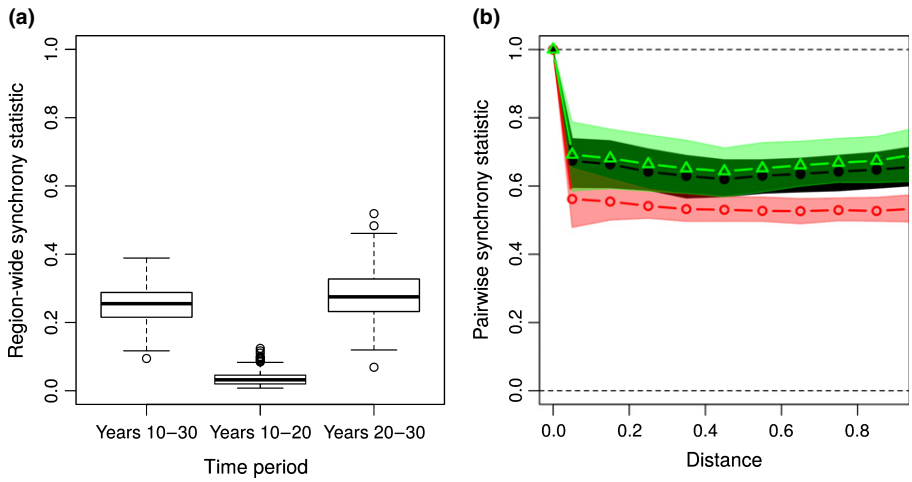

Fig. 4 Distributions of synchrony statistics obtained from 200 simulations of the metapopulation model. (a) Distributions (boxplots) of the region-wide synchrony statistic φ, when φ was computed from years 10 to 30, from years 10 to 20, and from years 20 to 30. Whiskers extend to the most extreme data point which is ≤ 1.5 times the interquartile range from the box. (b) 90% envelopes and medians of the pairwise synchrony statistics φ_{ij} against the distance separating the populations of host patches, for years 10–30 (black envelope and black line broken by closed circles), for years 10–20 (red, transparent envelope and red line broken by open circles), and for years 20–30 (green, transparent envelope and green line broken by triangles).

populations with greater host density, and the effect of density varied over time (Table 1). However, there were no significant interactions between the pathogen presence and either density or time. In a given population, a decreased growth rate in 1 yr tended to be followed by an increased growth rate the following year, and *vice versa* (significant negative autocorrelation parameter; Table 1). In 2012 and 2013, the host population growth from July to September was significantly lower in populations with greater host density and those that were infected in July (Table 2; Fig. 5b). There were no significant interactive effects of pathogen presence, host density or year on host population growth during the growing season. The random effect of population explained very little variance in host population growth during the two growing seasons (Table 2).

Discussion

To date, the role of pathogens in regulating the dynamics of their wild host plant populations has remained poorly understood. The highly dynamic and ephemeral nature of local pathogen populations, combined with the perennial lifestyle of many plant species and potential genotype × (genotype×) environment

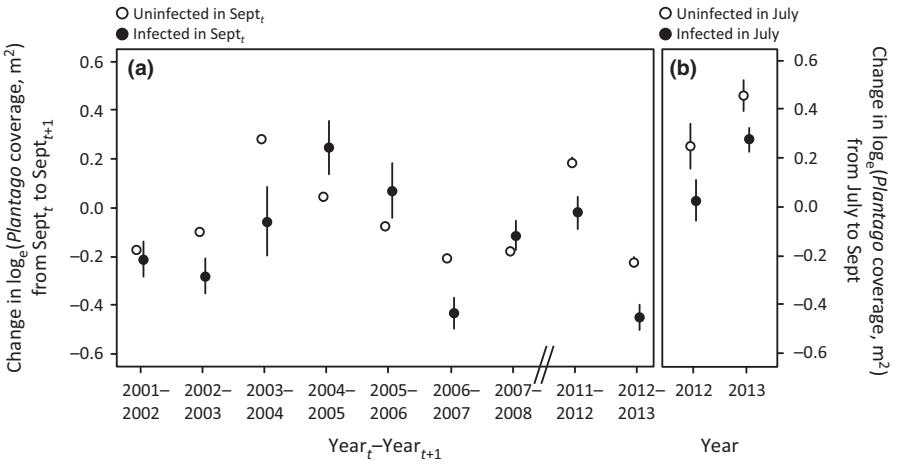

Fig. 5 Change in $\log_e$-transformed coverage of the host plant, *Plantago lanceolata*, in infected (closed circles) and uninfected (open circles) populations. (a) Annual host population growth rate decreased over the time series, and was lower in populations infected by *Podosphaera plantaginis*. (b) Host population growth from July to September was lower in infected than uninfected populations in both years. Error bars give ± 1 SE (the SE are very small for the > 3000 uninfected populations in (a)).

Table 1 Consequences of *Podosphaera plantaginis* infection for *Plantago lanceolata* population growth between sequential years from 2001 to 2008 and from 2011 to 2013, analyzed using a linear mixed effects model with first order autoregressive error structure

Source$_{ndf,ddf}$	Estimate ± SE	*F*-statistic	*P*-value
Intercept$_{1,23257}$	−0.46 ± 0.03	240.11	**< 0.0001**
Pathogen presence$_{1,23257}$	−0.15 ± 0.017	86.10	**< 0.0001**
Host density$_{1,23257}$	−0.088 ± 0.006	1940.33	**< 0.0001**
Year$_{1,23257}$	−0.053 ± 0.005	26.00	**< 0.0001**
Density × year$_{1,23257}$	−0.0092 ± 0.0009	94.75	**< 0.0001**
	SD or φ	LRT	*P*-value
Population (random effect)	SD = 0.000029	2917.61[1]	**< 0.0001**
temporal autocorrelation	φ = −0.42	2536.17[2]	**< 0.0001**

Population growth was modeled as the change in $\log_e$-transformed coverage of the host plant between years. Significant *P*-values are highlighted in bold.
LRT: [1]Statistic for exact likelihood ratio test, based on 10 000 simulated values from the sample distribution, testing the null hypothesis that the variance explained by the random effect is equal to zero (Scheipl *et al.*, 2008); [2]Statistic for likelihood ratio test comparing mixed effects models with and without autoregressive error structure.

Table 2 Consequences of *Podosphaera plantaginis* infection for *Plantago lanceolata* population growth over the growing seasons in 2012 and 2013, analyzed with a linear mixed effects model

Source$_{ndf,ddf}$	Estimate ± SE	*F*-statistic	*P*-value
Intercept$_{1,665}$	−1.64 ± 0.17	82.71	**< 0.0001**
Pathogen presence in July$_{1,201}$	−0.14 ± 0.06	8.61	**0.0037**
Host density in July$_{1,201}$	−0.34 ± 0.03	158.91	**< 0.0001**
Year$_{1,201}$	0.07 ± 0.05	2.36	0.13
	SD	LRT[1]	*P*-value
Population (random effect)	0.00033	0.00	0.56

Population growth was modeled as the change in $\log_e$-transformed coverage of the host plant from July to September. Significant *P*-values are highlighted in bold.
LRT, [1]Statistic for exact likelihood ratio test, based on 10 000 simulated values from the sample distribution, testing the null hypothesis that the variance explained by the random effect is equal to zero (Scheipl *et al.*, 2008).

interactions, has presented a challenge for quantifying the selection intensity that pathogens impose on their hosts in the wild (Laine, 2009; Wolinska & King, 2009; Burdon & Thrall, 2014). Our study spanning several thousand host populations and multiple years confirms the key role of environmental conditions in determining pathogen persistence over the winter offseason. As winter conditions changed through time, we observed a trend for disease dynamics to become increasingly synchronous across the metapopulation. In addition, our study confirms that pathogens can impact the growth of their host populations.

A major obstacle in quantifying selection intensity imposed by pathogens on their hosts is the scarcity of data on infection frequency spanning multiple populations and years. Some of the challenges in measuring the rates of infection may be purely methodological, as detecting pathogen occurrence reliably may be difficult, if not impossible, for diseases that lack conspicuous infection stages. The clear spatial configuration of the *P. lanceolata* population network in the Åland archipelago and the conspicuous symptoms caused by *P. plantaginis* make this pathosystem highly amenable to large-scale epidemiological studies. A high extinction rate of local pathogen populations during winter is responsible for keeping metapopulation-level infection prevalence low, yet factors generating variation in overwintering success have not been identified previously (Soubeyrand *et al.*, 2009; Jousimo *et al.*, 2014). Here, we show that successful overwintering at the metapopulation level correlates significantly with how mild the ambient winter temperatures are, or, when temperatures are below freezing, whether there is snow cover to protect the pathogen resting stages. As there is no negative effect of exposure to freezing on the host populations (see Fig. S7, Table S1), we assume that pathogen resting structures are directly affected by abiotic conditions. Pathogen resting spore structures are highly sensitive to abiotic variation (Tack & Laine, 2014), and may be damaged by freezing temperatures. Snow is likely to provide protection from exposure to freezing temperatures, resulting in higher overwintering success (Peterson *et al.*, 2009; Kumar *et al.*, 2013). The strong effect of winter conditions on pathogen

extinction rate was still evident at the end of the next season's epidemic, with a lower metapopulation-level disease prevalence in years with more days of exposure to subzero temperatures. To our knowledge, this is the first time that climatic conditions during pathogen dormancy have been identified as potentially critical for the prevalence of disease in the next season. Recently, both low and variable overwinter survival have also been documented for other pathogens (Montarry *et al.*, 2007; Desprez-Loustau *et al.*, 2014), suggesting that dynamics during the offseason may play a key role in the epidemiology of a wide range of pathogens.

We also found that disease occurrence patterns across the 50 × 70 km archipelago have become more synchronous in the second half of the 2001–2013 study period. Hence, while previously an increase in disease incidence in some parts of the metapopulation could coincide with a decrease in other parts, in recent years the increase has become more simultaneous across the pathosystem. While this may seem like a trivial side effect of increased disease incidence overall, it should be noted that even at its highest, the metapopulation level disease prevalence is only 20%. Hence, the host network is far from being saturated by infection, and could in theory support spatially asynchronous disease occurrence patterns. This increase in spatial synchrony means that there are larger clusters of infected populations within the archipelago, and that the ecological and evolutionary selection imposed by the pathogen on its host populations is likely becoming more homogeneous across the system. In a previous study, we found that spatial autocorrelation of *P. plantaginis* population extinctions occured at a much larger spatial scale than that of colonizations, suggesting that large-scale weather patterns may play a critical role for extinction dynamics (Jousimo *et al.*, 2014). The increase in synchrony we present here corresponds with years of mild winter temperatures and/or long-lasting snow cover in the Åland archipelago. Indeed, our model confirmed that a qualitative change in offseason conditions favoring pathogen survival generates disease synchrony patterns at the metapopulation level similar to that observed in our data. Overall, these results highlight that predictions regarding how changing climate impacts epidemiological dynamics need to shift focus from the transmission stage to the entire lifecycle of the pathogens (Garrett *et al.*, 2006). In many pathogens the overwintering stage is also the sexual stage (Agrios, 2005; Billiard *et al.*, 2012), and hence, successful survival of the offseason may not only increase the severity of disease epidemics but also their evolutionary potential (Gautam *et al.*, 2013). Future studies should investigate whether harsh winter conditions reduce pathogen diversity in our focal pathosystem, as would be expected based on the epidemiological data.

In this study, we focused on the relationship between winter climatic conditions and pathogen extinction dynamics. However, temporal change in other unmeasured environmental or ecological variables may have contributed to the observed trends in extinction rate and regional synchrony. For example, increased nitrogen deposition has been found to promote fungal infection in the dwarf-shrub *Vaccinium myrtillus* in Sweden (Strengbom *et al.*, 2002). Future studies could explore whether nitrogen levels in Åland are increasing through time, and whether this promotes within-season growth or overwinter survival of powdery mildew populations in our system. Another exciting future venue of research would be to measure whether ongoing climate change is altering plant diversity within these meadows, thereby increasing or decreasing *P. lanceolata* density to the degree that infection dynamics also change.

Despite the highly ephemeral and dynamic nature of disease occurrence in the *Plantago–Podopshaera* pathosystem, our results show that disease has a significant negative effect on the growth of wild host plant populations. We find that infected host populations decrease in size or grow more slowly than healthy host populations within a single growing season. Moreover, we find that infection in the previous year has a negative effect on population growth the following year. For a perennial such as *P. lanceolata*, the fitness effects of infection may be delayed (Jarosz & Burdon, 1992; Shefferson & Roach, 2012), appearing only in the subsequent seasons following infections. However, our results also indicate that host populations tend to rebound after a year of decreased growth. Given the ephemeral disease occurrence patterns, and how strongly the effects of infection may be mediated by the biotic (Hood, 2003; Laine, 2011; Clément *et al.*, 2012) or abiotic environment (Laine, 2007; Jorgensen, 2012; Mas & Verdu, 2014), it is not surprising that the negative effect of infection is not apparent in all years, and that there is considerable variation in the extent to which the pathogen impacts its host populations. Although our data at the population level does not allow us to tease apart which host life-history stages are most affected by infection, previous results and field observations confirm that increased mortality of infected hosts is likely to explain much of the observed decrease in growth of diseased populations (Laine, 2004, 2006).

These results add to the growing body of literature suggesting that selection does not need to be persistent over space and time to generate rapid ecological and evolutionary change in nature (Thompson, 2013). Understanding changes in selective pressures imposed by infection is a dilemma that lies at the very heart of disease biology. As evidenced by our results here, understanding the factors governing a successful completion of the resting stage may be needed to solve this dilemma, although it has traditionally been ignored in the ecological, evolutionary and epidemiological studies of pathogens. In particular, our study confirms that consideration of the offseason should be an essential component of successful disease control efforts. The increase in synchrony of disease dynamics across the pathosystem during our study period highlights the potential for changing climate to alter epidemiological dynamics, resulting in the greater spatial extent of epidemics within regions.

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Supporting Information

Additional supporting information may be found in the online version of this article.

Fig. S1 Photographs of the study system.

Fig. S2 Maps of the metapopulation and smaller networks of host populations.

Fig. S3 Metapopulation model simulation with temporal variation in environmental effect.

Fig. S4 Histogram of the total number of years powdery mildew was present in each population.

Fig. S5 Pairwise synchrony statistic as a function of distance between population clusters.

Fig. S6 Semi-variograms of the empirical proportions of powdery mildew presence.

Fig. S7 Relationship between exposure to freezing temperatures during the offseason and annual growth rate of host populations.

Table S1 Effects of infection, exposure to freezing, and host density on annual growth rate of host populations

Methods S1 Details of the theoretical model of spatial synchrony over time.

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