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RESEARCH ARTICLE

# Disease influences host population growth rates in a natural wild plant–pathogen association over a 30-year period

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## Abstract

1. The epidemiological and demographic dynamics of plant–pathogen interactions in natural environments are strongly affected by spatial and temporal influences. Here we assess the interaction between *Filipendula ulmaria* and its rust pathogen *Triphragmium ulmariae* by analysing a 30-year long dataset that has followed pathogen and plant population dynamics in a metapopulation of ~230 host patches growing on islands of the Skeppsvik archipelago in northern Sweden.
2. Over this period, the host metapopulation initially expanded in both number and size of individual patches before plateauing. In contrast, the pathogen metapopulation showed greater change. Disease incidence showed a convex pattern rising for the first decade before showing a marked decline in the last decade. At the same time, the prevalence of disease in infected populations showed a constant 30-year long decline.
3. At the individual host population level, each population was annually classified into one of four inter-year states: healthy, recolonization, extinction and diseased. Host populations that were healthy from 1 year to the next were significantly smaller than all other host population categories, while host populations in which disease was constantly present were significantly larger.
4. Host populations in which the pathogen underwent either an extinction or a recolonization event were of similar size and represented a measure of the host threshold size for long-term pathogen survival.
5. Host population growth rates declined as disease levels increased. The growth rate of host populations in which disease was continuously present was 75% lower than in populations that were free disease.
6. The sensitivity of the association to climate change as demonstrated through a decline in disease incidence and prevalence and an increase in drought damage to plant populations as temperatures rise has only become apparent through analysis of an extensive long-term dataset.
7. *Synthesis*. To date wild plant–pathogen studies have focused on the epidemiology of the pathogen and its effect on individual plant fitness. Here we have established a link to the impact of the pathogen on the long-term dynamics of host

populations. This has the potential to trigger a cascade of changes in the species composition and diversity of communities.

**KEYWORDS**
climate, epidemiology, extinction, host population growth rate, metapopulation, plant–pathogen interaction, recolonization

## 1 | INTRODUCTION

Interest in the impact of pathogens on host populations is at an all-time high. Patterns of epidemic development in human and animal populations are attracting increasing attention due to an upsurge in zoonoses (e.g. Hendra virus, SARS, MERS and Covid-19: Recht et al., 2020; Schmeller et al., 2020) or the spread of existing ones such as West Nile fever (Kilpatrick et al., 2007). In agriculture and forestry, novel diseases and ones spreading beyond well-established ranges are also exciting attention. This interest is driven by a concern about short-term human health and economic tolls, and by the potential for longer term consequences for the integrity and resilience of agricultural and natural ecosystems as climatic change alters evolutionary patterns, geographic ranges and potential infection severities of pathogens (Kocmánková et al., 2009; Velásquez et al., 2018; Wu et al., 2020; Yang et al., 2016; Zhan & McDonald, 2011). Despite this attention and the already extensive literature on host–pathogen interactions, there are still significant gaps in our broader knowledge. This is particularly so with respect to the epidemiological behaviour and demographic, ecological and evolutionary consequences of disease in wild systems.

In the literature assessing the impact of plant pathogens in non-agricultural systems, the potential for reductions in fecundity and longevity has been demonstrated extensively in controlled experiments but to a lesser extent in natural situations (Bernard-Capelle et al., 2006; Borer et al., 2007; Malmstrom, McCullough, et al., 2005). The quantum of such reductions in fecundity and longevity is highly variable being dependent on numerous factors mediating pathogen × host × environment interactions (disease triangles) including the timing of infection relative to host development, the intensity of disease and the type of disease involved (Burdon & Laine, 2019).

In the animal parasitology literature, there is a similar body of evidence that diseases induced by a range of pathogens including bacteria, viruses, fungi, oomycetes and other macro-parasites can affect individual host reproduction and survival. For example, long-term studies have shown the importance of nematode outbreaks on red grouse population sizes (Hudson et al., 1992), and mass mortalities of prairie dog populations caused by plague epidemics (Stapp et al., 2004). In studies of human disease, large and long-term datasets have underpinned an understanding of the importance of host population threshold sizes, the concept and validity of 'herd immunity', and the periodicity of diseases such as measles (Grenfell & Bolker, 1998; Keeley & Grenfell, 1997).

In contrast, a significant constraint to understanding the effects of plant disease in natural systems is a lack of long-term epidemiological studies. Indeed, insight into the demographic, ecological and evolutionary consequences of disease has largely been dependent on extrapolation from short-term (one or two seasons) observations of impacts on fecundity and/or longevity; on observations of patterns of resistance and susceptibility; and on inferences drawn from 'special case' situations involving the spread of exotic disease into new environments or the deliberate release of pathogens in biological control programs. The potential power of invasive pathogens to affect plant communities is demonstrated by significant host population declines associated with the loss of American chestnut to *Endothia parasitica* (Burke, 2012), multi-species losses caused by soil-borne pathogens such as *Phytophthora cinnamomi* in Australia (Shearer et al., 2012), wind and splash-borne *P. ramorum* ravaging tanoak populations in California (Rizzo et al., 2005) or recent epidemics of *Hymenoscyphus fraxineus* causing ash dieback in Europe (McMullan et al., 2018). In an analogous way, the outcomes of successful biological control programs demonstrate the potential for fungal pathogens to control the density and affect the spatial distribution of their weed hosts. Examples include the impact of *Puccinia chondrillina* on *Chondrilla juncea* in Australia (Burdon et al., 1981) and *Entyloma ageratinae* on *Ageratina riparia* in New Zealand (Barton et al., 2007).

While such epidemics illustrate the potential power of pathogens in natural systems, they are in many ways artificial and leave the less intrusive consequences of disease unresolved. Reductions in seed production do not necessarily translate into a change in the size of future plant populations. Most plant species produce an overabundance of seed so that losses during seedling or juvenile phases may not affect the size of the adult plant population (Harper, 1977). Even in annual plants, a full assessment of the potential fitness effects of disease may be fraught with multi-year considerations. For example, a study of mildew on wheat found effects that carried over two post-epidemic generations involving a reduction in the number of individuals and their competitive ability (Jarosz et al., 1989). Notwithstanding this, our understanding of host plant–pathogen interactions inherently recognizes a tight link between evolutionary and ecological dynamics (Penczykowski et al., 2016). The best documented evidence that a pathogen has an on-going effect on its host is often found in studies showing that the level of disease resistance present in target host populations changes as spatial (Burdon & Jarosz, 1991; Montes et al., 2019) and temporal (Thrall et al., 2012) scales vary.

Demonstrating that the impact of endemic pathogens goes beyond changes in the genetic structure of host populations to an actual effect on population size, and through this the structure of

communities is far less well documented. For a small handful of endemic associations characterized by large long-lived hosts, the spread of a killing disease from well-defined infection foci creates the opportunity to track host deaths and compare community structures in the pre- and post-death phases. Situations involving tree pathogens such as *Phellinus weirii* (Cook et al., 1989) or *Armillaria luteobubalina* (Shearer et al., 1997) provide a clear picture of the role of at least some pathogens in natural communities. Another possibility to track host deaths is provided by clonal evergreen dwarf shrubs such as *Empterum hermaphroditum* (Olofsson et al., 2011, 2013) where diseased host tissue remains in situ enabling both pathogen identification and quantification. However, most host individuals rapidly disappear after death, leaving no signs of the pathogen responsible.

What role, if any, do the less obvious debilitating rusts, mildews and necrotrophic blotches have on the place of such hosts in plant communities? Determining an answer to this question for relatively short-lived species with high fecundity requires a longer term view of the epidemiological and ecological consequences of the waxing and waning of disease through time. However, although long-term temporal datasets have proved very powerful in uncovering underlying trends in as diverse areas of biology as agriculture (Silvertown et al., 2006), biodiversity research (Magurran et al., 2010) and human health (Roseboom et al., 2006), the number of datasets that link plant and pathogen dynamics in multiple naturally occurring populations through time is strictly limited (Burdon & Thrall, 2014). These interactions have variously demonstrated general aspects of the unpredictability of disease incidence and severity, the importance of environmental variation and even aspects of the evolutionary interaction between host resistance and pathogen infectivity (Thrall et al., 2012), but to date none have made a link between pathogen activity and host population size.

Here we report on an analysis of a 30-year long dataset covering epidemiological patterns in the rust pathogen *Triphragmium ulmariae* and population demographics in a large metapopulation of its host, *Filipendula ulmaria*, growing in up to 239 patches on 78 islands in the Skeppsvik archipelago of northern Sweden. We use this large dataset to explore:

1. Temporal patterns in disease incidence, prevalence and severity in the entire metapopulation;
2. Relationships between patterns of disease dynamics and the size of host populations; and
3. The impact of disease on host population growth rates.

In this way we extend our knowledge of the impact of pathogens in natural systems beyond their short-term effect on general measures of fitness and the population dynamics of their hosts, to their long-term effects on the growth rate of host populations and through this their potential to influence community structure.

## 2 | MATERIALS AND METHODS

### 2.1 | Study area

The Skeppsvik archipelago was created through an on-going process of isostatic land uplift of a series of parallel glacially lateral moraines covers more than 70 islands in the Gulf of Bothnia off the north-eastern coast of Sweden (63°44–48′N, 20°34–40′E). In this area, the dynamics of *Filipendula ulmaria* L. (Rosaceae) and its host-specific rust pathogen *Triphragmium ulmariae* (DC.) Link (Sphaerophragmiaceae) has been followed in a long-term (30-year) study of a series of separate populations (increasing from 179 in 1990 to 232 in 2019). Small islands were occupied by single host populations, while larger islands are home to multiple populations. The location of each host population was located on the detailed Ekonomisk Karta över Sverige, Västerbottens län; scale 1:10,000 map of the area using the grid point RT90 7080000/1740000 on the map sheet 20K 6h Tärnögen as the N/E 0/0 co-ordinate position. This corresponds to 63.7455°N, 20.6692°E.

### 2.2 | Host–pathogen biology

The *F. ulmaria–T. ulmariae* host–pathogen interaction involves an herbaceous perennial host that reproduces by seed and rhizatomous growth and a full cycle autoecious, host-specific rust pathogen (Wilson & Henderson, 1966). In the study area, the host shoots from underground rootstock every spring, flowering in summer before dying back in mid-autumn. The pathogen is similarly constrained, surviving the winter as dormant teliospores that germinate at the beginning of the growing season (June), and then undergoes sexual recombination before generating aecia and subsequently uredia. A few cycles of asexual reproduction occur before falling temperatures trigger the formation of new telia. Full descriptions of the life cycles of both host and pathogen have been published elsewhere (Burdon et al., 1995).

Dispersal of the pathogen between individual plants within a population or between populations across the archipelago may occur through aerially dispersed basidiospores, aeciospores or urediospores although basidiospores and aeciospores typically only travel a few metres. Rust urediospores have the reputation of travelling great distances but in the current association evidence suggests that population-to-population dispersal is limited. Instead, dispersal appears to depend heavily on the movement of telia and teliospores in winter storm generated flotsam that is washed around the islands of the archipelago (Burdon et al., 1995; Smith et al., 2003).

### 2.3 | Host population data collection

Individual host populations were operationally defined as discreet groups of plants separated from others by water barriers (i.e. on different islands) or, when on the same island, by a combination of

shore-line distance and thickness of intervening vegetation (Zhan et al., 2018). In 1990, a comprehensive search of 78 islands identified 179 populations ranging in size from a single plant to a deme of 3,800 individuals. Depending on the size and physical diversity of an island, anywhere between 1 and 12 populations were found to be present. The number of populations slowly rose before reaching a peak of 232 populations in 2014, since then three populations have disappeared. During the 30-year period, a total of 239 populations were identified, but over that time 10 small populations fused with their nearest neighbours. The numerical size of each population was estimated by censusing representative areas and then adjusting those scores for the total area covered by the population. This approach was employed to cope with the patchy distribution of plants in many populations (for further details see Burdon et al., 1995; Ericson et al., 2002). Furthermore, the patchy nature of distribution within populations greatly limited the value of any attempt to estimate density at the population level. As a consequence, any reference to population size means the number of individuals present.

Over the 30 years of the study, we recorded five host population extinctions (out of >6,300 possibilities). All of these involved very small populations that reappeared within a year or two.

## 2.4 | Pathogen epidemiological data collection

Disease incidence (the presence/absence of disease), prevalence (% host individuals infected per population) and disease severity (% leaf area infected per population—this was averaged over both the infected and healthy individuals assessed) were all scored in each host population annually (1990 to 2019) at the mid-point in the epidemic cycle, in early-mid August. Populations found to be disease free at that time were revisited in early-mid September. In each population, disease prevalence was determined by examining a sample of haphazardly chosen plants (minimum number = 25) for the presence of pustules. Severity was scored on infected plants using a visual scale measuring percentage of leaf area infected; while incidence was simply determined by whether any disease was recorded. In very small host populations (<80 plants) all individuals were examined. In larger ones this was not practical, so a haphazardly chosen sample determined by the numerical size and the spatial distribution of the population was assessed (range of 50–200 plants assessed for disease per population). If disease was absent on these plants a further intensive search was made to determine whether the pathogen was present but at very low frequency. All epidemiological and host population assessments over the entire study time were carried out by Lars Ericson with the assistance of Jeremy Burdon.

## 2.5 | Drought effect data collection

The occurrence of drought damage to host plants was assessed in 19 medium–large *F. ulmaria* populations distributed through the Skeppsvik archipelago. Droughting was visually assessed in early August as score 1, if observed on dry sites, and as score 2, if observed on both dry and mesic sites, on the basic of withering of basal leaves important to the early seasonal increase in disease. A maximum index of 38 denotes extensive drought damages on dry and mesic sites in the archipelago.

## 2.6 | Mean temperature data collection

Gridded monthly mean temperature data for the period 1989–December 2015 inclusive was obtained from the Swedish Meteorological and Hydrological Institute, SMHI (http://www.smhi/se), for the Swedish grid point RT90 7084000, 1737000 located in the central part of the Skeppsvik archipelago. Post December 2015, temperature values are based on interpolations from three adjacent official meteorological stations as stated in Zhan et al., 2018.

## 2.7 | Data analysis

Metapopulation levels of disease—incidence, prevalence and severity—and host population size in each year were tabulated by averaging disease measures across all host populations. Host population growth rate was estimated for each of the populations using a logistic model based on the temporal change in size (plant number) of the population over the 30 years (used in Figure 4). Exploratory analysis by least squares indicated the logistic model performed as well as, or better than, other models for these populations. A further set of host population growth rates was estimated according to disease behaviour in the metapopulations, that is, host population size in each of four disease categories was estimated annually using the data pooled from all individual populations and then used to estimate growth rates according to their temporal change over the 30 years (used in Figure 5). In this analysis, disease dynamics in each host population in each year was assigned to one of four categories reflecting changes from the previous season: extinction (present–absent), recolonization (absent–present), continuing disease presence (present–present) and continuing disease absence (absent–absent). Host population size in each of these categories was estimated by averaging the number of host plants recorded in the corresponding category in Year $N$ – 1, generating 29 data points ($N$ – 1 = 30 – 1 = 29) for Year 1 to Year 29 for each of the four categories, which were then used to calculate growth rates of the disease categories. To test whether growth rates varied between categories, a bootstrapping analysis was conducted, in which disease duration category was randomized between populations in each year. The bootstrapping was repeated 100 times to generate 95% confidence intervals around the growth rates and population sizes for each disease duration category.

For consideration of the effect of host population size on the duration of disease (Figure 2), the persistence of disease was calculated by counting the years of disease absence or presence in individual

host populations. For example, if a population during a consecutive 6-year period has disease incidences of 100,111 (1 = disease present; 0 = disease absence) and a population size of 100, 150, 200, 250, 300 and 350, the population sizes of extinction (1st to 2nd year), continuing disease absence (2nd to 3rd year), recolonization (3rd to 4th year) and continuing disease presence (4th to 5th year and 5th to 6th year) are 100, 150, 200 and 275 (the average of 4th and 5th year) respectively (i.e. host population in the first year of the pair). And the population sizes supporting the 2-year consecutive disease absence (2nd to 3rd year) and 3-year consecutive disease presence (4th to 6th year) are 125 (the average of 1st and 2nd year) and 250 (the average of 3rd to 5th year) respectively. Because we were unable to determine the population size of $N - 1$ for the first year, disease persistence was counted starting from the second year of the survey, that is, 1991. These mean that in the persistence analysis, the maximum disease duration (continuous absence or presence) was 29 years and more data points were 'regenerated' from populations with a longer continuous disease status (presence or absence) than from those with a shorter similar status, even though each original data point (i.e. population size of host and incidence, prevalence and severity of disease in a year-deme combination) is only considered once. For example, only one data point was regenerated if the disease was continuous present/absent for the last 29 years in a population, but several data points were generated if there were a range of disease durations in the population.

Associations between pathogen epidemiological (incidence, prevalence, severity and persistence) and host population parameters (size and growth rate) or sampling time, were fitted to linear or nonlinear models using least squares (Borwein & Erdelyi, 1995; Lin, 1989). Exploratory analysis using Moran's *I* test (Moran, 1950) and the Durbin–Watson statistic (Durbin & Watson, 1950) indicated disease data were not spatially or temporally autocorrelated or skewed and hence no corrections were made. For Moran's *I* test for spatial autocorrelation, populations within the same island were considered as the neighbours. Mixed-effect linear regression models, with islands as a random intercept, was used to assess the effects of pathogen epidemiological parameters (incidence, prevalence and severity) on the host growth rate. The use of random effects was determined by comparing the Akaike information criterion (AIC, McElreath, 2016) between the mixed-effect models and base-line models without random intercepts. In this evaluation, models with a smaller AIC value are better but are not considered to be statistically different if the difference is less than two units. Both the exponential and the mixed-effect model analyses were carried out in R statistical software using the function NLS and NLME packages respectively (R Core Team, 2021). Analysis of variance for the growth rate of *F. ulmaria* was performed using the general linear model (GLM) procedure implemented in SAS 9.1.3. Least significant difference (LSD) was used to compare population size and growth rate of *F. ulmaria* among the four disease categories (Ott, 1992). A contrast analysis in a stepwise procedure was also performed to compare the growth rates of recombination, extinction and disease–disease categories with disease-free category using 'R'.

## 3 | RESULTS

Over the 30-year period of this study, the number and size of *F. ulmaria* host plant populations comprising the Skeppsvik metapopulation have varied. Although size variation occurred between populations in any given year (Figure S1), mean host population size also increased consistently through the period (1990–2000) after which it flattened. The temporal pattern of population size fitted well to an exponential model ($y = 773.35\ (1 - e^{-0.27x})$, $R^2 = 0.66$, $p < 0.0001$), a quadratic polynomial model ($y = -1.37x^2 + 52.87x - 306.29$; $R^2 = 0.64$, $p < 0.0001$) and a linear model ($y = 10.48x + 531.50$; $R^2 = 0.32$, $p = 0.0012$) but the exponential model was superior to other two as indicated by the AIC test (Figure 1a; Tables S1 and S2).

The incidence of disease (presence/absence) caused by the rust *T. ulmariae* across the Skeppsvik host metapopulation changed through time. Over the first 10 years or so of the study, the proportion of host populations infected initially rose before plateauing and subsequently showed a consistent decline (Figure 1b). The tightly fitting convex curve pattern underlying the changing dynamics of disease incidence in the metapopulation ($y = -0.001x^2 + 0.03x - 0.30$; $R^2 = 0.58$; $p < 0.0001$) contrasts with the consistent decline in mean disease prevalence (number of individual plants infected per population; Figure 1c). Although highly significant, the latter epidemiological parameter showed greater year-to-year variability ($y = -0.05x + 2.28$; $R^2 = 0.37$; $p = 0.0004$). The general patterns displayed by the severity data were similar to those of the prevalence data (Figure 1d) but with greater year-to-year variability especially in the earlier years as indicated by standard errors of the parameters (Table S2).

Earlier versions of these basic host and pathogen metapopulation-level data—of mean host population size, and disease incidence and prevalence (Figure 1a–c respectively) covering shorter periods of time have been published previously (Smith et al., 2011; Zhan et al., 2018). However, the extended dataset presented here provides a complete 30-year record of patterns of change in host and pathogen population sizes. These changes are fundamental to the core of the current paper and to an understanding the role of disease in affecting wild host populations.

Beneath these gross metapopulation level patterns there was considerable variability in the temporal persistence of disease in individual host populations. As host population sizes increased, there was a greater probability that disease was present for longer consecutive stretches of years. On the other hand, as host population sizes got smaller the incidence of disease fell and the number of continuous disease-free years stretched out (Figure 2). The data fit both linear ($y = 0.03x - 18.12$; $R^2 = 0.85$; $p < 0.0001$) and nonlinear ($y = 13.75\ \ln(x) - 83.49$; $R^2 = 0.86$; $p < 0.0001$) relationships (Table S2). Even though, the latter is a slightly better fit than the former, they do not differ statistically (Table S1) when compared using the AIC. As host populations became smaller and smaller, disease incidence also declined asymptotically towards a never-present scenario. On the other hand, as mean host population sizes continued to increase, the long-term persistence of disease also rose but never exceeded the total number of years assessed. Such differences in

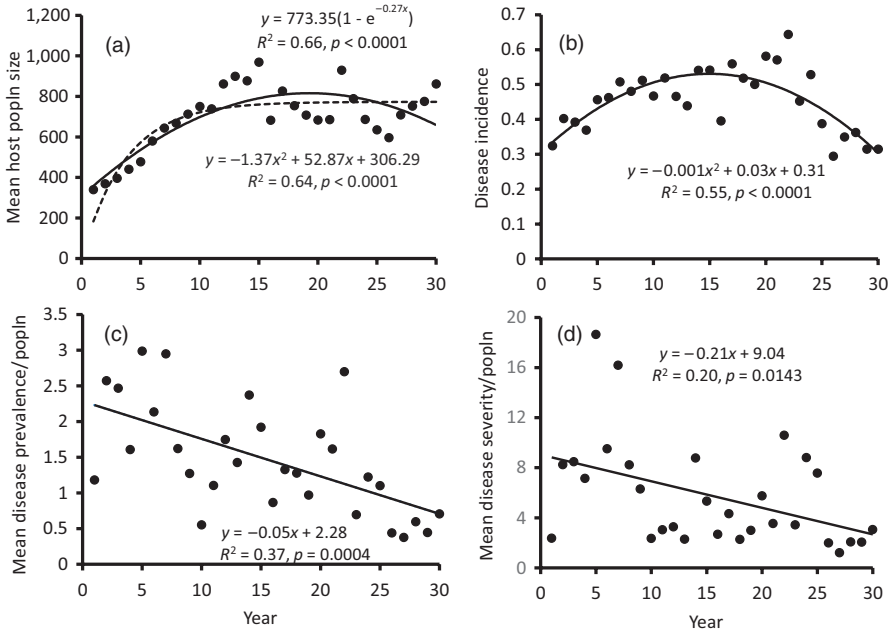


**FIGURE 1** Demographic changes in the *Filipendula ulmaria* (host)–*Triphragmium ulmariae* (pathogen) metapopulation over the 30-year study period. (a) Changes in mean host population size (average of all populations for any given year) through time; (b) changes in disease incidence frequency across the entire metapopulation; (c) changes in mean disease prevalence through time; and (d) changes in severity through time

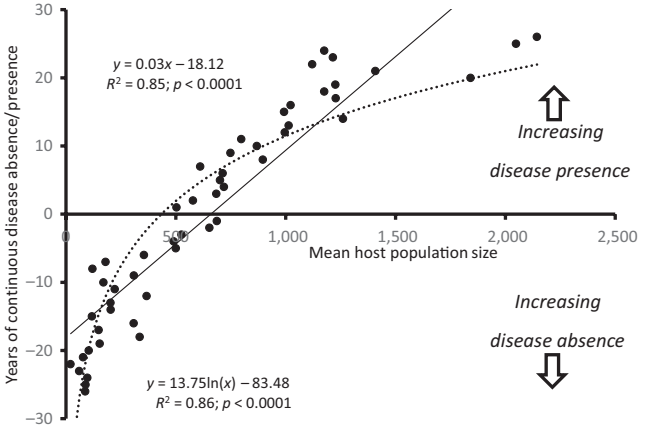


**FIGURE 2** The effect of host population size on the duration of continuous disease presence or absence. The mean host population size was calculated by taking the mean size of each population covering each disease duration period and subsequently averaging all population estimates for each duration period

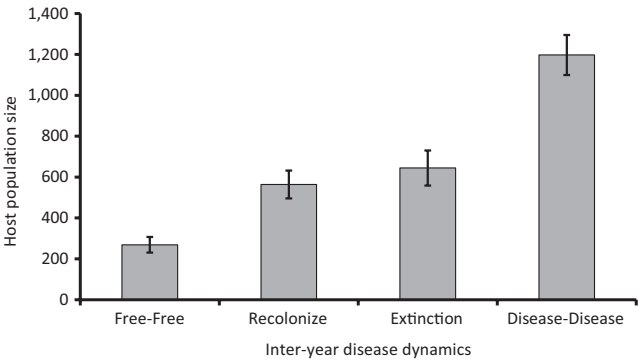


**FIGURE 3** The impact of host population size on disease status. The mean host population size was calculated by taking the mean size of each population covering each disease duration period and subsequently averaging all population estimates for each duration period

the duration of the pathogen's presence in different host populations sets the scene for potential differences in the impact on host populations and the possibility of differential selective pressures.

Disease in individual host populations flicker from presence to absence and vice versa over time. Consequently, the different individual host–pathogen population associations can be allocated into four groups reflecting the dynamics of those associations [disease absence (absent–absent); recolonization (absent–present); extinction (present–absent); continuing disease presence (present–present] based on the prior year (Yn − 1) to current year (Yn) disease transition status (Figure 3). When this view of disease incidence is related to host population size, not surprisingly, host in which disease was present from 1 year to the next (D-D) had the largest population size. The host population group that remained disease free (F-F) had the smallest population size, while there was no significant difference in host population size between disease recolonization (F-D) and extinction (D-F) situations. Contrast comparison also indicated the population sizes of recolonization, extinction and disease–disease were also significantly higher than that in disease-free group (Table 1; Table S3). Although

**TABLE 1** Parameter estimates and their associated standard errors and *p*-values in the comparisons of population size between three disease classes, that is, extinction, recolonization and disease–disease, and the reference classes (disease free)

| Coefficients | Estimate | *SE* | *p*-value |
|---|---|---|---|
| Intercept | 269.95 | 38.92 | <0.0001 |
| Recolonization | 276.45 | 53.67 | <0.0001 |
| Extinction | 395.98 | 54.61 | <0.0001 |
| Disease–disease | 927.24 | 55.04 | <0.0001 |

there was variation in population size within each disease group, all groups met the assumption of homoscedasticity (data not shown).

We are interested in understanding the relationship between disease and plant performance. An earlier study (Ericson et al., 2002) demonstrated that the *Triphragmium* rust could affect the probability of survival of young *F. ulmaria* plants. Here we are interested in determining the pathogen's impact on plant demography at a population level. First, we examined the relationship between the three disease parameters (incidence, prevalence and severity) and host population size using the full 30-year dataset. Use of mixed models was justified by their lower AIC values than those of the models with fixed effect (Table S1) with an exception in the association between the disease prevalence and host growth rate in which random intercept has a slightly higher AIC value. Even then, we used the mixed model over the fixed model for consistency. In all cases host population growth rate declined as the disease parameters in question increased as revealed by the mixed models (Figure 4; Table S4). The tightest relationship occurred with respect to disease prevalence (Figure 4b) but those with disease incidence (Figure 4a) and severity (Figure 4c) were also both significant. It is noted that if the single extreme outlying data point seen in these figures was ignored the strength of the association increases markedly.

As noted earlier, the dynamics of disease in different populations can be categorized to reflect changing patterns of occurrence from 1 year to the next. With this in mind, we investigated the relationship between the four host population disease transition groups (Free-Free, Free-Diseased, Diseased-Free and Diseased-Diseased) and host population growth rate for each transition pair averaged across all populations for all years (Figure 5). This showed a clear growth rate advantage to host populations in which disease was absent in the current year [Free-Free and Diseased-Free (extinction)] over populations in which disease was present in the current year [Diseased-Diseased and Free-Diseased (recolonized)]. Moreover, host populations in which disease was present in both years had a significantly lower growth rate than those just recolonized.

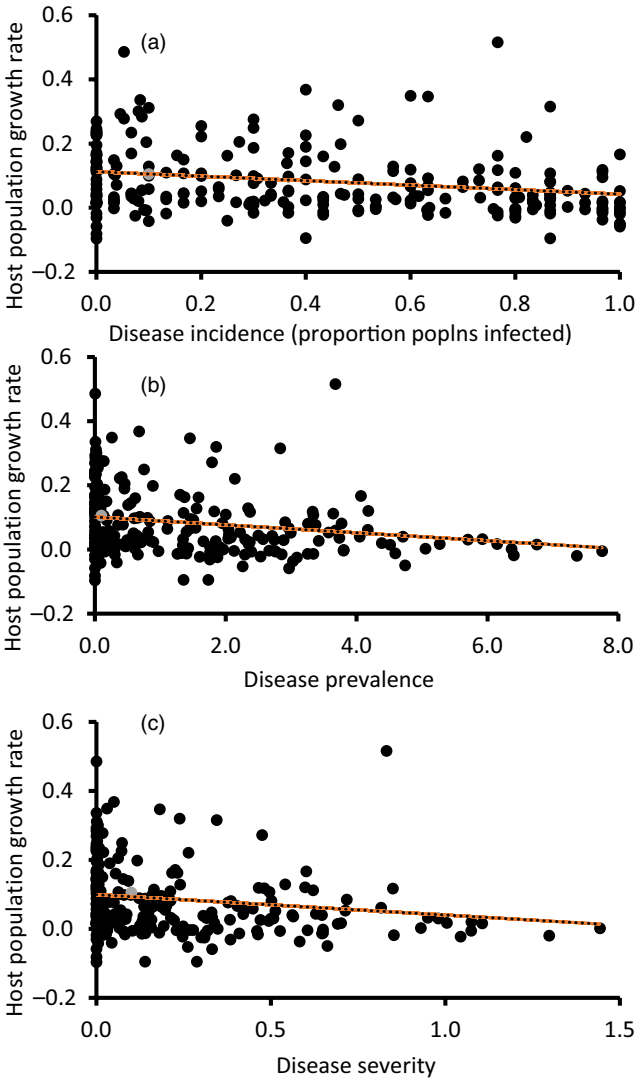


**FIGURE 4** The relationship between disease parameters and host population growth rate. (a) The effect of disease incidence ($y = -0.0698x + 0.1122$); (b) the effect of disease prevalence ($y = -0.0123x + 0.1017$); and (c) the effect of disease severity ($y = -0.0591x + 0.0992$). Host population growth rates were estimated using a logistic model based on the size of populations over the entire 30-year study period

## 4 | DISCUSSION

Studies of the role of pathogens in wild plant populations and communities have taken two distinct approaches. The first of these—the pathology–genetics–co-evolution axis—places emphasis on pathogen epidemiology, changes in the population genetics of both host and pathogen, and any consequential co-evolutionary interactions between host and pathogen (Laine, 2006; Thrall et al., 2012). In contrast, the second approach—the ecology–biodiversity–conservation axis—places more emphasis on the host, its effect on the pathogen and potential changes in the diversity of plant communities (Knops et al., 1999; Roscher et al., 2007). These two approaches are highly complementary although individually they provide incomplete views of the full ramifications of the interactions occurring between pathogens and plants in natural plant communities (Burdon & Laine, 2019).

In the current study we took a different approach to assess interactions occurring between pathogen and host plant populations at a spatiotemporal scale. By following long-term (30 year) host and pathogen population dynamics in 180–239 populations, we have shown that a period of host population number and mean population size increase in the 1990s was followed by a period of fluctuation in size around a near-steady (asymptotic exponential distribution) or very slowly decreasing mean (quadratic distribution; Figure 1a). Even though both distributions fit the data well, the exponential model performed better than the quadratic one by AIC test, indicating that the temporal dynamics of *F. ulmaria* follows the general pattern of population growth in many species (Van Bael & Pruett-Jones, 1996). Over the same period the pathogen

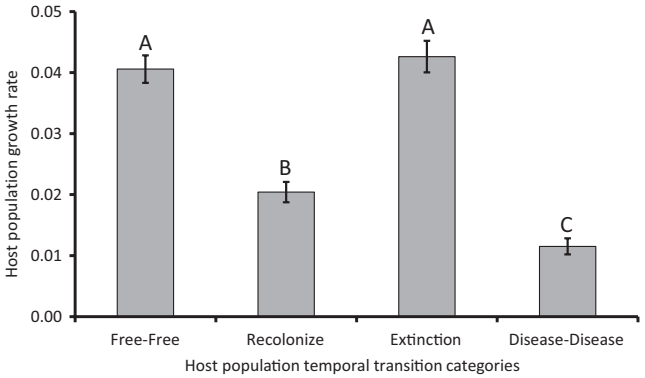


**FIGURE 5** Differences in the growth rate of *Filipendula ulmaria* populations according to one of four annual (Year *N* –1 to Year *N*) temporal transition categories (F-F, F-D, D-F and D-D) of the pathogen *Triphragmium ulmariae*. 5% LSD is indicated by different letters atop of the columns. For each transition pair (F-F, F-D, D-F and D-D) the size of each host population was taken as an average of that of each individual Year *N* – 1 to Year *N* transition averaged across all populations for all years to obtain a mean growth rate

metapopulation has shown a convex rise and subsequent fall in the proportion of host populations infected (Figure 1b) while mean disease prevalence (Figure 1c) and mean disease severity (Figure 1d) per population have shown a steady decline over the full 30-year period. While such summary statistics provide a broad view of temporal changes in the interaction, they can disguise the unpredictability of the occurrence of disease from one season to the next. Although even large host populations may vary in size from year to year, when their core size remains reliably over a thousand individuals the pathogen can remain continuously present for periods of 20 years or more (Figure 2). In contrast, host populations of less than a few hundred in size essentially fall below the threshold size for reliable pathogen re-establishment from year to year (Figure 2). While many small populations do occasionally become infected, the probability that the pathogen then persists locally into following seasons is often very low.

Following the dynamics of both host and pathogen in multiple populations over multiple years allows a detailed examination of the threshold effect. In essence, our data allow a sub-division of the demographic interaction between host and pathogen that reflects the annual survival challenge faced by the pathogen at the individual population level, consistent with host–pathogen interactions in other systems (Garnett & Lewis, 2007; Geard et al., 2015). From 1 year to the next, a host population may be continuously infected, continuously free of disease, become re-infected after a period of no infection, or the pathogen may become locally extinct. The probability of the first and second of these outcomes—the pathogen remaining present or remaining absent—is strongly correlated with the size of the host plant population. Disease rarely dies out in large host populations while it is rarely ever present in very small ones (Figure 3). In contrast, there was no significant difference in the size of host populations in which the pathogen population became extinct or in which recolonization had occurred. It is this host population size at which pathogen survival flickers unpredictable on-and-off that essentially sets the host threshold size for its continued presence.

The evolutionary and ecological implications of the reliable long-term presence of a pathogen are considerable. Clearly, the presence of a pathogen is required to drive selection for an increase in the frequency and diversity of resistance within given host populations relative to others (Burdon & Laine, 2019). In many cases, variation in the level of disease resistance among host populations has been linked to differences in the environment (Dinoor, 1970; Laine et al., 2011). However, the preciseness of this interaction is most convincingly illustrated within single host populations where selection has driven the accumulation of a higher frequency of resistance in microenvironments particularly favourable for pathogen growth and survival. For example, in the interaction between *Plantago lanceolata* and its mildew *Podosphaera plantaginis* higher levels of resistance were found in areas of host populations in which microclimatic differences favoured a greater level of pathogen activity (Laine, 2006). In many of the larger *F. ulmaria* populations we have followed in the current study, the spatial distribution of the pathogen also appears to be non-random with disease being encountered more often in certain parts of the population than others. Examples of such favourable areas are: (a) more shady sites often of north-western aspect; (b) accumulation bays where flotsam is regularly deposited; and (c) boulder-rich sites where litter is trapped even under high sea levels. The first-mentioned site is particularly favourable for build-up of the asexual uredinial stage, while the two latter sites are important for over-winter survival. Such favourable sites often make up only a minor fraction of the shore, so it has not been feasible to include such detailed resolution in our annual disease assessments and could contribute to the conservation of no spatial autocorrelation. Despite this, it is clear from earlier studies that *T. ulmariae* may cause mortality with the survival of individual *F. ulmaria* seedlings over a 5-year period being tightly correlated with the severity of disease suffered (Ericson et al., 2002).

The long-term presence of a pathogen may also have significant ecological implications, but in contrast to their role in shaping the genetic structure of wild plant populations (Burdon, 1987; Burdon & Laine, 2019), their impact on the temporal dynamics of host populations and through this on the structure of whole plant communities has received surprisingly little attention. Indeed, the most compelling cases involve a few examples of the impact of root rots that kill dominant species in forest and woodland communities. In these cases, the consequence of multi-year passage of disease caused by *Armillaria* (Shearer et al., 1997), *Heterobasidion* (Rizzo et al., 2000) or *Phellinus* (Hansen & Goheen, 2000; Holah et al., 1997) species through the forest can be assessed at single points in time. Other examples are disease caused by various snow blight fungi, *Arwidssonia* and *Eupropolella*, on evergreen ericaceous dwarf shrubs (Olofsson et al., 2011, 2013).

Logistic difficulties in following the fate of individual plants in large numbers of plots; in accurately ascribing whether mortality has biotic or abiotic causes; and in identifying the causal organism, have together curtailed attempts to assess the impact of most diseases

in herbaceous plant communities. Certainly, multiple examples exist that demonstrate the impact that host diversity has on the size, impact and infectivity of pathogen populations (Mitchell et al., 2002; Rottstock et al., 2014; Sommerhalder et al., 2011; Yang et al., 2019; Zhan et al., 2002) but these provide only circumstantial support for a direct role of pathogens driving community diversity by reducing the size of individual plant numbers. A further step in that direction may be achieved through the use of fungicides to demonstrate that pathogens can suppress dominant grasses (Allan et al., 2010; Kohli et al., 2021) or that the depredations of nematodes and soil-borne fungi help speed the process of succession on sand dunes (van der Putten et al., 1993).

A more direct way of assessing the role of pathogens in influencing plant community composition is to determine whether identified impacts on the fecundity or longevity of individual plants exceed the 'built-in' resilience provided by excess propagule production and to result in changes in host population growth rates. Population growth rate is frequently used in assessments of the impact of diseases on animal populations (Holmes, 1982; Lachish et al., 2007), but is little known in studies of the effect of diseases on wild plant communities, and even then, are based on indirect rather than direct measures. For example, yellow mosaic virus is a common pathogen in wild populations of *Curcurbita pepo* where infection prevalence ranges from 0% to 100% across years and various host species. A common garden experiment was used to estimate the effect of viral infection on flower, fruit and seed production, and the resultant data then used to parameterize a deterministic matrix model (Prendeville et al., 2014) to show that the effect of viral infection on population growth rate of host plants varied among virus treatments and different *C. pepo* populations.

Here we assessed the impact of disease on host population demography by examining the relationship between the growth rate of individual host populations and their relationship to disease incidence, prevalence and severity (Figure 4). The strong correlation, particularly between disease incidence or prevalence and host population growth rate (Figure 4a,b), demonstrates the important influence the pathogen has on the growth rate of *F. ulmaria* populations. On average, host population size increased over the study time (Figure 1a). Negative density-dependent population growth could interact with disease parameters to shape the observed patterns of association. However, when we performed a multiple-regression analysis with population size and disease as independent variables and host population growth rate as dependent variable, we found that population size did not contribute to the growth rate in this case ($p = 0.6848$), confirming our argument that the disease is a constraining factor regulating host growth.

When this relationship is further deconstructed by considering the growth rate of populations from each of the four temporal transition categories (Free-Free, Free-Diseased, Diseased-Free and Diseased-Diseased; Figure 5) the impact of the disease and its implications for early season recruitment are emphasized. Growth rates were greatest for host populations in which no disease was present in either year/*N* or Year *N* (F-F) or in populations in which disease became extinct (D-F). For both these population categories no seedling losses to rust would have occurred (seedlings are particularly vulnerable to rust-induced mortality; Ericson et al., 2002). Also note that high mortality among diseased seedlings/juveniles is often common following longer periods of drought (unpublished data; see also discussion about adult plants further below). In contrast, host populations in which disease successfully recolonized (F-D) or in which disease was constantly present (D-D) were exposed to the early development of damaging aecial infections some weeks before our annual survey period. Furthermore, the category of populations showing the lowest growth rate (D-D) was that in which the pathogen had successfully carried over from the previous season and might reasonably be expected to have the most disease present early in the season.

Factors affecting the growth rate of individual components of natural plant communities have the potential to initiate cascades of changes in the species composition and diversity of communities and the broader networks of other species they sustain. An earlier analysis of the Skeppsvik *Filipendula–Triphragmium* dataset detected a positive association between a long-term trend of increasing extinction rates in individual pathogen populations of the metapopulation and increasing temperature (Zhan et al., 2018). Indeed, over the 30 years of this study average mean April–November temperatures have shown a slow but inexorable rise (1.6°C between 1990 and 2020; Figure S2). Over the same time period we have seen the number of plants suffering from droughting to be strongly related to mean July temperatures (Figure S3). One likely explanation for the increased extinction rates is that disease on adult plants normally starts to increase on basal leaf rosettes. Following drought these leaves start to wither before the build-up of the asexual uredinial stage, thus drastically reducing the amount of inoculum.

Such extensive withering of basal leaf rosettes has increased during the study period and the subsequent negative effect on the pathogen population is amplified as diseased leaves are more sensitive to drought than healthy leaves (unpubl. data). The increased sensitivity of plants to drought as a consequence of rust infection that breaches the integrity of the outer cuticle has been seen on a number of occasions—for example, in epidemics of rust on *Senecio vulgaris* (Paul & Ayres, 1984). The observed association between disease persistence with temperature may also be attributed to reduced survival and reproduction of the pathogen at higher air temperatures—a phenomenon that has been documented for many pathogens (Sabburg et al., 2015; Yang et al., 2016).

Temperatures in northern Fennoscandia have risen significantly in the past 25 years and are expected to continue to rise (IPCC, 2014; Figure S2) with regional climate simulation models predicting increases of between 1.6–3.2 and 3.7–5.2°C by 2,100 (Jacob et al., 2014). The consequences of such temperature changes are hard to predict but for *T. ulmariae* an increasing restriction to more benign, moist and shady, microsites as drier shore habitats prevail in the archipelago, would likely result in a continued slow decline in its incidence, prevalence and severity

(Figure 1b–d). Provided these changes are restricted to the pathogen, this might be expected to remove some constraints on a greater expansion of *F. ulmaria* populations on the Skeppsvik islands. Other work in the archipelago has addressed the impact of leaf-chewing beetles on *Filipendula ulmaria* and how they may relate to local variations in plant community composition (Hambäck et al., 2006; Stenberg et al., 2006, 2007, 2008). The 'knock-on' consequences for these beetles of a possible expansion of *F. ulmaria* following a release from *T. ulmariae*, as well as for the wider range of its other specialist enemies (a blotch fungus, two mildews, a handful of sawfly species, two aphids and two leafhoppers) and a large number of more polyphagous species, are hard to tell especially when the links these species also have to other plants in the community are taken into account. Given the important role plant pathogens have in shaping the structure of communities, changes in the epidemiology of pathogens have potentially far-reaching impacts on ecological and evolutionary processes that we are yet to fully understand.

Our long-term data clearly show the ability of pathogens to affect the growth rate of their host populations. However, of potentially greater significance is the cascade of events that may occur as climatic conditions continue to change. Demographic and genetic interactions between hosts and pathogens are sensitive to environmental changes that alter the fitness of either or both host and pathogen. In the Skeppsvik archipelago, these interactions between *F. ulmaria* and its rust pathogen *T. ulmariae* are coming under increasing pressure as growing season temperature increases of 1.6°C over just 30 years (Figure S2) are affecting plant performance and droughting (Figure S3). The *T. ulmariae*–*F. ulmaria* association illustrates how sensitive pathogen–host interactions are to climate change (this study and Zhan et al., 2018) and that the outcome of these fine-tuned interactions on the disease cycle is a gradual process impossible to grasp without accurate data (cf. Shaw & Osborne, 2011). As pathogens are generally neglected in long-term studies this will hamper our understanding of the mechanisms underlying observed and future changes in natural and man-made biota.

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## CONFLICT OF INTEREST

The authors have no conflict of interest to report.

## AUTHORS' CONTRIBUTIONS

L.E. and J.J.B. conceived the study and gathered all the epidemiological and demographic data; J.Z. and J.G.-J. conducted the data analyses; all authors contributed to writing the manuscript.

## DATA AVAILABILITY STATEMENT

Data from this study are available from the Dryad Digital Repository https://doi.org/10.5061/dryad.2bvq83br7 (Zhan, 2021).

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