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Influence of predation on community resilience to disease

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Abstract

1. Outbreaks of generalist pathogens are influenced by host community structure, including population density and species diversity. Within host communities predation can influence pathogen transmission rates, prevalence and impacts. However, the influence of predation on community resilience to outbreaks of generalist pathogens is not fully understood.
2. The role of predation on host community resilience to disease was assessed using an epidemiological multi-host susceptible-exposed-infectious-recovered model. *Sphaerothecum destruens*, an emerging fungal-like generalist pathogen, was used as a model pathogen. Six cyprinid and salmonid fishes, including an asymptomatic carrier, were selected as model hosts that are known to be impacted by *S. destruens*, and they were used within a model host community.
3. Pathogen release into the host community was via introduction of the asymptomatic carrier. Mortality from infection, pathogen incubation rate, and host recovery rate were set to a range of evidence-based values in each species and were varied in secondary consumers to predict top-down effects of infection on the resilience of a host community. Predation pressure within the fish community was varied to test its effects on infection prevalence and host survival in the community.
4. Model predictions suggested that predation of the asymptomatic hosts by fishes in the host community was insufficient to eliminate *S. destruens*. *Sphaerothecum destruens* persisted in the community due to its rapid transmission from the asymptomatic host to susceptible host fishes. Following transmission, pathogen prevalence in the community was driven by transmission within and between susceptible host fishes, indicating low host community resilience. However, introducing low densities of a highly specific piscivorous fish into the community to pre-date asymptomatic hosts could limit pathogen prevalence in the host community, thus increasing resilience.
5. The model predictions indicate that whilst resilience to this generalist pathogen in the host community was low, this could be increased using management interventions. The results suggest that this model has high utility for predicting community resilience to disease and thus can be applied to other generalist parasites to determine risks of disease emergence.

KEYWORDS

aquatic, emerging infectious diseases, interspecies interactions, invasion, topmouth gudgeon

*Authors contributed equally.

1 | INTRODUCTION

Shifts in food web structure can occur due to pathogens altering the symmetry of competition between species and affecting density-dependent dynamics (Dobson, Lafferty, Kuris, Hechinger, & Jetz, 2008; Tompkins, Dunn, Smith, & Telfer, 2011). The consequences of infection by a particular pathogen vary according to its host specificity and the community structure of potential free-living hosts, and range from species-specific mortalities to community-wide disease outbreaks. Generalist pathogens can cause major shifts in biodiversity and food web structure by infecting a broad range of species across trophic levels (Fenton & Brockhurst, 2007; Peeler, Oidtmann, Midtlyng, Miossec, & Gozlan, 2011; Thompson, Lymbery, & Smith, 2010). Empirical work suggests that generalist pathogens emerge in some host communities but not in others, and with a range of lasting effects (Ercan et al., 2015; Fisher, Garner, & Walker, 2009; Olson et al., 2013), implying that host community structure may influence pathogen emergence and persistence. This could mean preventing emergence through disease resistance, or effectively recovering from it through community resilience. Here, community resilience is defined as the ability of populations to recover to pre-emergence abundance.

The introduction of a novel species to a community is in itself enough to affect food web structure (Amundsen et al., 2013; Britton, 2013; Gozlan, Britton, Cowx, & Copp, 2010), but that shift can be amplified when the introduced species acts as a host for a novel pathogen (Amundsen et al., 2009; Lafferty et al., 2008; Poulin & Leung 2011). For example, the introduction of rinderpest virus via farmed cattle to wild African ungulates in the Serengeti reduced populations of mid-trophic level species by 80% (Lafferty et al., 2008), affecting carnivore populations and changing the vegetation, which led to an increase in fires and habitat destruction (Hudson, Dobson, & Lafferty, 2006). Global trade in amphibians has largely contributed to the spread of the chytrid fungus *Batrochochytrium dendrobatidis* leading to local changes in primary production with direct consequences for insects, severely affecting ecosystem stability (Sime-Ngando, 2012; Whiles et al., 2006).

As seen with the increasing global incidence of *B. dendrobatidis* (Fisher et al., 2009; Olson et al., 2013), fungal and fungal-like pathogens are particularly threatening to biodiversity and ecosystem function (Ercan et al., 2015; Fisher et al., 2012; Gozlan et al., 2014). This is particularly true for fungal pathogens introduced via non-native hosts, as there may be additional community level changes caused by the introduction of the non-native species (Britton, 2013). One striking example is the introduction of the fungal-like generalist *Sphaerothecum destruens*, which can be transmitted to new locations and hosts via an invasive asymptomatic carrier, topmouth gudgeon *Pseudorasbora parva* (Gozlan, St-Hilaire, Feist, Martin, & Kent, 2005). *Sphaerothecum destruens* acts as a generalist pathogen with over ten known hosts (Andreou & Gozlan, 2016), and the infection has varying durations of incubation, recovery, and degrees of disease-induced mortality depending on host susceptibility (Andreou et al., 2011). These differences could also be related to the environment; chronic infections are expected in areas with low levels of free-living propagules (e.g., rivers) and more acute infections where there could be concentrated numbers of infectious propagules in a small area (e.g., aquaculture sites). Whether an infection has a chronic or acute effect is important, as it will affect responses of the host community. In chronic infections, susceptible host populations can be continuously depleted compared to acute infections which would be cleared from the population faster (Jolles, Cooper, & Levin, 2005). Conversely, chronic infections might allow the population to recover over time, as there are no severe peaks in mortality (Lachish, Knowles, Alves, Wood, & Sheldon, 2011). However, there are additional community interactions that can impact the prevalence of infection and mortality from disease, such as predation (Packer, Holt, Hudson, Lafferty, & Dobson, 2003; Searle, Mendelson, Green, & Duffy, 2013). Variation in the manifestation of infections by *S. destruens* makes this parasite an ideal model species for testing how predation can affect the transmission and emergence of different generalist pathogens.

In highly acute infections, predators targeting infected prey can lower the level of infection in the host population (Packer et al., 2003). If a pathogen is trophically transmitted and infected hosts are predated on by a resistant species, then transmission to new hosts could be limited, as the predator presents a transmission 'dead-end' for the pathogen (Barber, Hoare, & Krause, 2000; Roche, Dobson, Guégan, & Rohani, 2012). While predation of infected hosts is one mechanism which influences disease dynamics, predation of parasites themselves reflects another pathway, especially for pathogens with free-living infectious stages (Johnson & Thieltges, 2010). Increased predation pressure on pathogens can be beneficial, with zoospore predation by *Daphnia* spp. capable of reducing infection levels of *B. dendrobatidis* in tadpoles under certain conditions (Searle et al., 2013). Removing such predators can have a detrimental effect on disease-susceptible prey populations with acute infections (Packer et al., 2003), but more work is needed on the effects of chronic infections, or where the predators themselves are also susceptible to infection. Thus, predator manipulation could be important in disease management, including use of selective restocking that increases the proportion of a non-susceptible species that limits pathogen transmission via dilution effects, by predating on infected hosts or on infectious propagules (Keesing et al., 2010; Roche et al., 2012).

Here, through the use of a multi-host susceptible-exposed-infectious-recovered (SEIR) model, this study tested how host community composition and predation influenced the resilience of a community to an introduced generalist pathogen. The model species were *S. destruens* and its asymptomatic carrier *P. parva*. An asymptomatic carrier was defined as a species that can transmit the pathogen to other hosts at no cost to its own health. Several fish species of the families Cyprinidae (e.g., *Abramis brama*, *Cyprinus carpio*, *Rutilus rutilus*) and Salmonidae (e.g., *Salmo salar*, *Oncorhynchus mykiss*, *Salmo trutta*) have experienced elevated mortality as a result of infection with *S. destruens* (Andreou, Arkush, Guégan, & Gozlan, 2012; Arkush, Frasca, & Hedrick, 1998; Paley, Andreou, Bateman, & Feist, 2012). A subset of known host species were selected to model a theoretical species community, which included species in direct competition with the introduced host and species which pre-dated on the introduced host

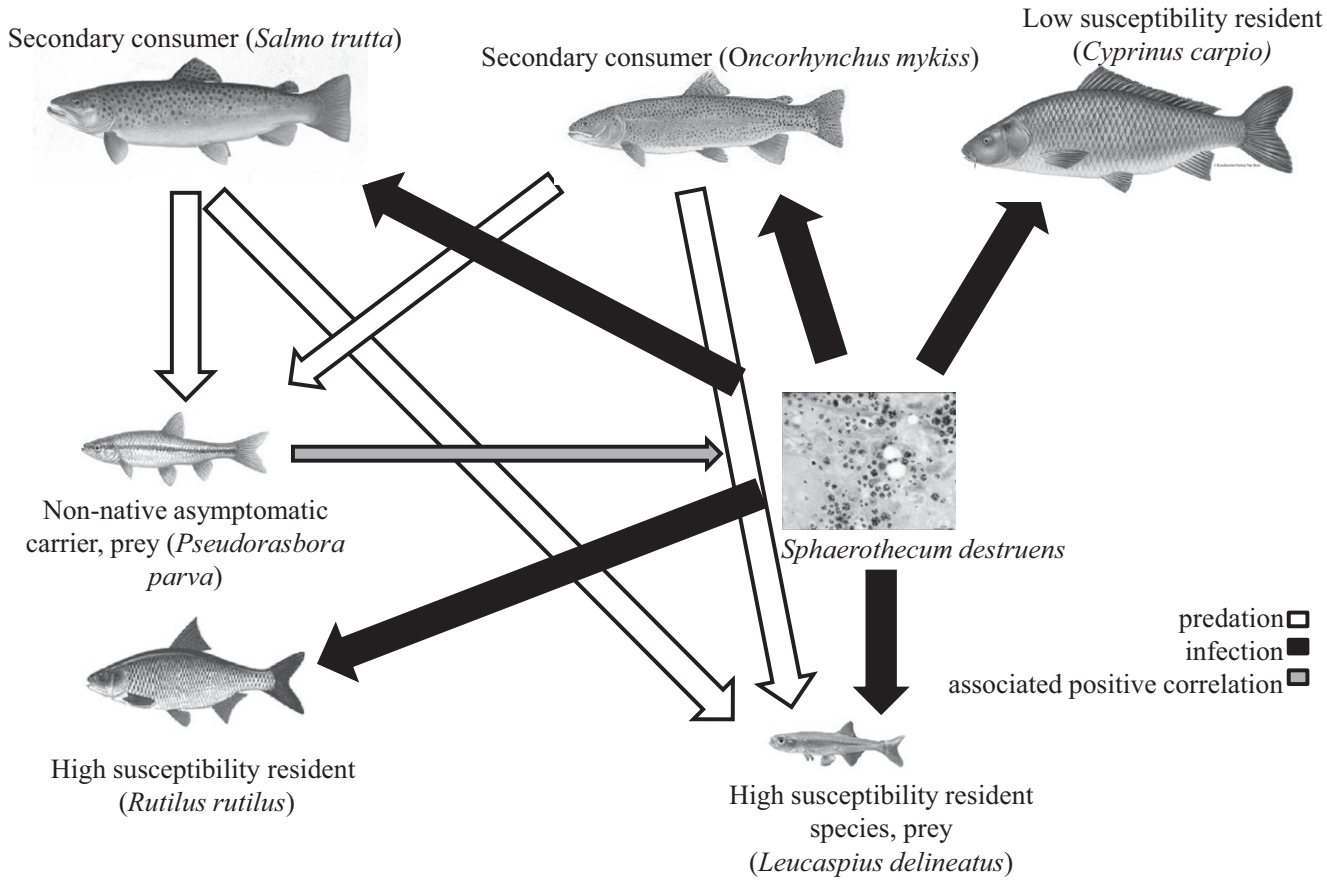

FIGURE 1 The interspecies interactions included in the community models. The grey arrow indicates a correlated increase in infectious propagules as the asymptomatic carrier density increases (as they introduce the pathogen). Black arrows indicate an inverse relationship between infectious propagules and species density due to infection. Similarly, and white arrows indicate predation between the connected species. The thickness of the arrows represents the strength of the interaction (c = 0.5 for thick arrows, 0.1 for thin arrows). Predation (p) was varied in model scenarios

(competing resident species and secondary consumers, respectively). This enabled the inclusion of resource competition within the model although looking at the specific effects of competition was not the primary focus of the study. Including resource competition within the overall model's framework ensured a more ecologically realistic community simulation. Objectives were to: (1) predict the effects of generalist pathogen introduction on host survival in a community comprising several susceptible fish species, and (2) test how predation of the introduced host affects the fish community's resilience and species diversity to the emergence of *S. destruens* at different degrees of predator infection susceptibility. Varying the epidemiology of the pathogen in the model allowed the exploration of a wide range of conditions beyond the empirical studies, and the predictions have implications for disease outbreak management.

2 | MATERIALS AND METHODS

2.1 | Host-pathogen model system

Sphaerothecum destruens is a unicellular, intracellular generalist pathogen that is transmitted to over 10 known susceptible host species through infectious spores (Arkush et al., 1998). Spores build up within host organs and are shed through the gut epithelium, skin, bile or urine, where they can survive and zoosporulate in freshwater for several days (Andreou, Gozlan, & Paley, 2009; Arkush, Leonel, Adkison, & Hedrick, 2003). Phylogenetically, *S. destruens* is on the boundary between animals and fungi in the Mesomycetozoea class, with several other known pathogens of animals and humans (Mendoza, Taylor, & Ajello, 2002). It has caused up to 90% mortality in salmon farms in the United States (Arkush et al., 1998) and has been found at prevalence ranges from 25% to 100% in fish species in Turkey (Ercan et al., 2015).

2.2 | Species assemblage

The species assemblage was selected to include hosts with a range of disease resistance to *S. destruens*, with an underlying framework of resource competition incorporated within the model structure. Model parameters for each species were based on existing data and expert knowledge on interspecific interactions and susceptibility to *S. destruens* infection, including previous single host models of epidemiology, species data from Fishbase (www.fishbase.org), and

published literature (see Supporting Information for full details). The theoretical assemblage included two secondary consumer species, brown trout *S. trutta* and rainbow trout *O. mykiss* (Arkush et al., 2003; Beyer, Copp, & Gozlan, 2007), two prey species *Leucaspius delineatus* and *P. parva* (which was the introduced asymptomatic carrier) (Pinder & Gozlan, 2003) and two additional species with high and low disease resistance *C. carpio* and *R. rutilus*, respectively (Andreou et al., 2012). The latter two species were not considered as prey or predators but were initially abundant species in the community and were susceptible to *S. destruens* (Figure 1). Species that were present in the community before the introduction of *P. parva* were referred to as resident species. Such an assemblage structure mimicked wild communities that could become infected with *S. destruens* (Ercan et al., 2015; Gozlan et al., 2005). Note that although found in some lowland European rivers, this specific species assemblage is not widespread, especially the coexistence of the salmonid species with species such as *C. carpio*. However, this community structure was useful for this study, as the parameter estimates and interactions were supported by empirical data to create general conclusions.

2.3 | Multi-host ecological model

An SEIR model was used to simulate a pathogen outbreak in host communities, where a asymptomatic carrier and the generalist pathogen were introduced. The model predicted the timeline and level of population decline by providing a temporal perspective of community changes. The model categorised each section of a host population based on their infection status (susceptible [S], exposed [E], infectious [I] and recovered [R]). Differential equations described the rate of change in a host population from one category to the next. In addition to epidemiological parameters, a Lotka–Volterra competition coefficient (*c*) between each Species *i* and *j* was incorporated, which represented interspecies resource competition (see Supporting Information). Density-dependent birth (*b*), incorporating the effects of resource competition, and natural mortality (*m*) of the population were based on species data from www.fishbase.org. Predation (*p*) was included separately from resource competition, as individuals could be removed from any category of infection. Infected individuals can be preyed on proportionally more than uninfected individuals (Moore, 2002; Schaller, 1972). However, there is no available data for this aspect in the model system, especially as there are two types of infection considered (acute vs chronic). To minimise assumptions and keep the model evidence-driven, this effect was not considered in the model, only in the discussion. Susceptible individuals of Species *i* could become infected through direct contact with an infectious individual (β_i) or by ingesting infectious propagules (*Z*) via environmental transmission at rate $\varepsilon_i\left(\frac{Z}{Z+k_{ei}}\right)$.

$$\frac{dS_i}{dt}=b_iN_i\left(1-\frac{N_i-\sum_{j=1}^{n}c_{ij}N_j}{K_i}\right)-S_i\sum_{i}^{n}\beta_{ji}I_j-\varepsilon_i\left(\frac{Z}{Z+k_{ei}}\right)S_i-\sum_{j=1}p_{ij}S_iN_j-m_iS_i$$

When susceptible individuals ingested enough spores, they moved into the exposed (*E*) category (Equation 2) and remained in this category for the incubation period of the pathogen $\frac{1}{\sigma_i\left(\frac{Z}{Z+k_{si}}\right)}$. The saturation functions included in the equations represent the dose-dependent progression of infection (Al-Shorbaji, Gozlan, Roche, Robert Britton, & Andreou, 2015). Each *k* value refers to the threshold density of pathogen propagules needed to be ingested by the host for a 50% probability of progressing to the next stage of infection (Anttila et al., 2016; Codeço 2001, Roche et al., 2009). These functions were included to better demonstrate how varying conditions can change the progression of infection (i.e., high *S. destruens* density can lead to more rapidly progressing infections, and vice versa).

$$\frac{dE_i}{dt}=S_i\sum_{i=1}^{n}\beta_{ji}I_j+\varepsilon_i\left(\frac{Z}{Z+k_{\varepsilon i}}\right)S_i-\sigma_i\left(\frac{Z}{Z+k_{si}}\right)E_i-\sum_{j=1}p_{ij}E_iN_j-m_iE_i \quad (2)$$

Following the incubation period, individuals became infectious (I) and released spores. Within the infectious category, individuals either experienced mortality from the disease at rate $\alpha_i\left(\frac{Z}{Z+k_{ai}}\right)$ or recovered at rate $\gamma_i\left(\frac{Z}{Z+k_{gi}}\right)$ (Equation 3). Recovered individuals could not be re-infected (Equation 4).

$$\frac{dI_i}{dt}=\sigma_i\left(\frac{Z}{Z+k_{s_i}}\right)E_i-\gamma_i\left(\frac{Z}{Z+k_{gi}}\right)I_i-\alpha_i\left(\frac{Z}{Z+k_{ai}}\right)I_i-\sum_{j=1}p_{ij}I_iN_j-m_iI_i \quad (3)$$

$$\frac{dR_i}{dt}=\gamma_i\left(\frac{Z}{Z+k_{g_i}}\right)I_i-\sum_{j=1}p_{ij}R_iN_j-m_iR_i \quad (4)$$

The number of infectious propagules in the environment (*Z*) was dependent on the rate of release of spores from each infected host per day (ϕ_i), and the mortality rate of the spores μ (Equation 5). This mortality rate was based on experimental work on the longevity of *S. destruens* spores and zoospores (Andreou et al., 2009), and varies according to temperature.

$$\frac{dZ}{dt}=\sum_{1=i}^{n}\phi_iI_i-\mu Z \quad (5)$$

A population carrying capacity for each species (*K*) was introduced under the assumptions that a) equilibrium population sizes of species are dependent on body size (Dunham & Vinyard, 1997), and thus smaller species were more abundant than larger species (Froese & Pauly, 2015). Values were based on average body masses for each species from Fishbase (www.fishbase.org). This represented intraspecific competition within the community, as birth rates declined after reaching carrying capacity (Britton, Davies, & Brazier, 2008).

2.4 | Parameter estimation

Epidemiological parameter values for all species were estimated in Al-Shorbaji et al. (2015), based on empirical data (Table 1). The number of spores released by infected hosts per day was reduced to 20% of the values estimated from the single host models, to account for water current and attachment to sediments in natural

TABLE 1 Parameter ranges used in single host epidemiological SEIR simulations

Parameter (per day rate)	*Abramis brama*	*Cyprinus carpio*	*Rutilus rutilus*	*Leucaspius delineatus*	*Salmo salar*
Direct transmission (β)	0.15–0.99	0.0155–0.017	0.08–0.1	0.015–0.02	0.008–0.016
Mortality from infection (α)	0.165–0.18	0.017–0.025	0.129–0.133	0.22–0.3	0.06–0.08
Environmental transmission (ε)	0.12–0.7	0.0008–0.0012	0.003–0.007	0.0074–0.008	0.004–0.008
Incubation rate (σ)	0.09–0.16	0.013–0.0805	0.095–0.11	0.23–0.233	0.04–0.11
Recovery rate (γ)	0.095–0.105	0.065–0.072	0.099–0.101	0.14–0.17	0.02–0.04
Spore release (ϕ)	0–10000	350–650	0–4000	330–350	45–100
Spore mortality (μ)	0.15–0.3	0.18–0.205	0.18–0.21	0.48–0.53	0.48–0.55
Ke*	$0–1*10^8$	$0–1*10^6$	$0–6*10^7$	500–50000	$0–1*10^7$
Ks*	0–45000	5000–11000	0–15000	6800–7200	$2000–3*10^6$
Ka*	0–25000	8000–12000	6000–11000	3000–10000	$10000–1*10^6$
Kg*	0–50000	4000–10000	6000–15000	6500–10000	$5000–1*10^6$

systems (Schmeller et al., 2013; Searle et al., 2013), compared to the closed tank systems in the experimental trials. Parameter estimations represented a range of species that varied in disease resistance and in trophic interactions. The parameters may be numerically uncertain, but they fit the purpose of the model to simulate a theoretical community and answer questions about community resilience.

The parameter ranges for mortality from infection (i.e., virulence), incubation rate and recovery rate were examined between species for consistent values or patterns. Median parameter values for each species were used as data points to fit standardised major axis (SMA) regression models using the *smatr* R package (Warton, Duursma, Falster, & Taskinen, 2012). The corresponding minimum and maximum values of each parameter range were incorporated into the figures as uncertainty. Values outside these ranges led to a model's mean squared error (MSE) being at least 5% higher than the minimum MSE. These ranges were selected as a representation of uncertainty as values of standard error and confidence interval may be skewed for five data points.

A positive correlation between mortality from infection (α) and incubation rate (σ) was predicted, under the assumption that a shorter incubation time (therefore, a larger σ) led to more rapidly acting infections and thus higher mortalities (Carey et al., 2006; Daszak et al., 1999). Incubation and recovery rate (ɣ) were highly sensitive parameters in the single host models (Al-Shorbaji et al., 2015) and a potential correlation between them was tested to uncover any additional insights about the epidemiology and progression of infection in this model system. Relationships between parameters are often hard to determine for generalist pathogens, as each host can respond differently and species-specific data are limited in most cases. Establishing these correlations here was a novel step in this field and created an avenue for expanding the conclusions made by this model to similar generalist pathogens and other host species. SMA regression models were used to estimate relationships between parameters across species in the model.

2.5 | Predation scenarios tested using the model

The focus of the model was on how predation within a resident community affects its response to introduced species (an asymptomatic carrier) and infection. Both predation and infection were varied in the model to examine a range of possible outcomes. The long-term effects of acute versus chronic infections on community survival were observed, by examining the community Shannon diversity index (*H*) 10 years after the introduction of the asymptomatic carrier and pathogen. Here, acute refers to high virulence, short-term infections (several days) while chronic refers to low virulence, relatively long-term infections (several weeks or longer). Ten years was selected as an appropriate timeframe to detect long-term impacts. *Pseudorasbora parva* can become the dominant species in a community within 3 years of introduction (Britton, Davies, & Harrod, 2010), and so extending this period to 10 years was considered sufficient to detect long-term and lagging effects. The model did not incorporate long-term changes in conditions such as temperature or salinity, or stochastic effects, which would be expected to reduce the model's predictive power when long-term impacts (e.g., >10 years) would be modelled.

Beyond the epidemiology of the infection, the host community was varied. The effect of increased predation pressure on the survival of the introduced host and on pathogen persistence was tested in two ways: altering the predation coefficient (*p*) between secondary consumer and prey species, and altering the secondary consumer population density within the community. Species' abundance in the model was monitored to examine long-term population trends. All models were run in R (Version 2.15.1) and the Shannon diversity index (H) of the different fish communities was calculated in R in the *vegan* package (Oksanen et al., 2011). This metric was chosen to explore the community equilibrium in terms of both species richness and evenness following the introduction of the asymptomatic carrier. It allowed the comparison of the community before and after a pertubation in a summary form, in addition to the model outputs on species' abundance over time. Interspecific interactions were modelled in (1) the absence of the introduced asymptomatic carrier; (2) the absence of the pathogen (i.e.,

0% prevalence) and (3) the presence of both asymptomatic carrier and pathogen, incorporating various levels of host disease resistance.

3 | RESULTS

3.1 | Correlations between epidemiological model parameters

The epidemiological parameter ranges based on the empirical infection data (Andreou et al. 2011) were explored to examine possible correlations between them. The data supported a significant positive correlation between mortality from infection (α) and incubation rate (σ) (Figure 2) and between incubation and recovery rate (γ) (Figure 3). These estimated parameter relationships were used to test the role of host susceptibility in community resilience. Based on the *S. destruens* data, the median parameter values across the susceptible species also supported the presence of a positive correlation between recovery rate and virulence (SMA: $\gamma = 0.49\alpha + 0.026$, $p = .056$, $R^2 = 0.75$). Here, a positive correlation between mortality from infection, incubation rate, and recovery rate was assumed to explore the effects of acute versus chronic infections on community resilience. This provided a more complete perspective on pathogen dynamics in this particular system.

3.2 | Introduction and establishment of the asymptomatic carrier in the community

The resident community remained at a stable equilibrium without the introduced species (Figure 4a). Following their introduction and in the absence of *S. destruens*, *P. parva* was predicted to establish a stable population within 2 to 3 years (Figure 4b). Neither secondary consumer species showed any significant changes in population size in the absence of infection, but they limited the population growth of *P. parva* to 77% of its population size in the absence of predation. The remaining resident species demonstrated minor population declines (Figure 4).

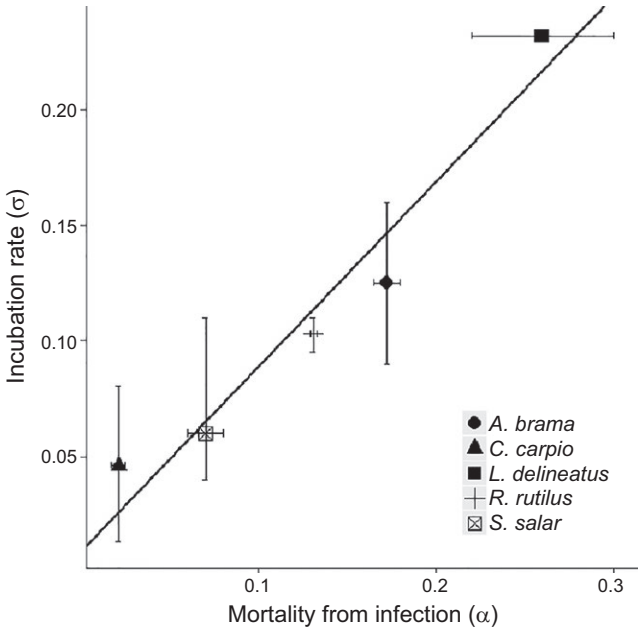

FIGURE 2 The median values of incubation rate for each species (σ) plotted against the median values of mortality from infection (α) for each species, with the minimum and maximum values of each range incorporated as uncertainty. There was a significant positive linear correlation between mortality (α) and incubation rate (σ) (SMA: $\sigma = 0.79\alpha + 0.009$, $p < .001$, $R^2 = 0.94$)

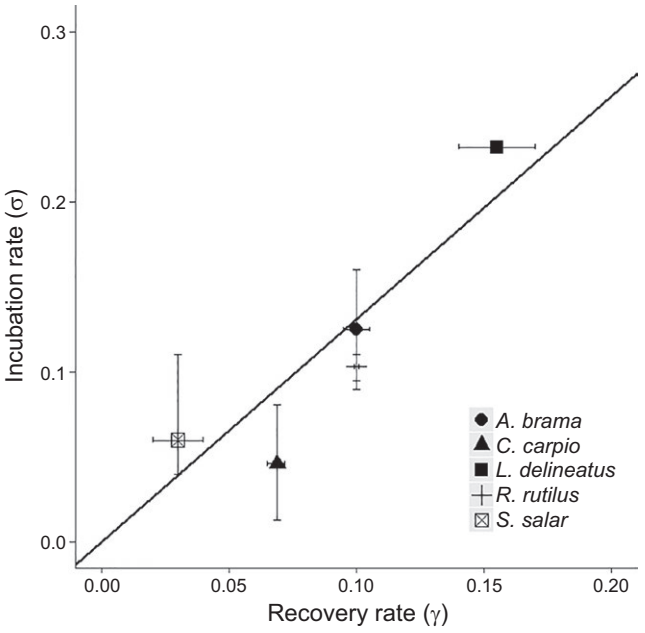

FIGURE 3 The median values of incubation rate for each species (σ) plotted against the median values of recovery rate (γ) for each species, with the minimum and maximum values of each range incorporated as uncertainty. There was a significant linear correlation between recovery rate and incubation rate (SMA: $\sigma = 1.31\gamma$, $p < .001$, $R^2 = 0.95$)

3.3 | Impact of Sphaerothecum destruens on community structure

The model simulations then predicted how *S. destruens* would impact community structure by modelling chronic and acute infections on the secondary consumers. Chronic infections were correlated with higher community diversity than acute infections ($H = 1.4$ vs $H = 1.1$, compared with an initial value of 1.43). Fish abundance was lowest when mortality from infection was high (with a short incubation rate) and recovery rate was low (i.e., a long infectious period) (Figure 5). There was a sharp decline in species abundance when the consumers' mortality from infection surpassed a threshold ($\alpha = 0.1$), and all species experienced a decline within 1 year of introduction of the asymptomatic host (*L. delineatus*: −68%; *R. rutilus*: −16%; *C. carpio*: −2%; *O. mykiss*: −66%; *S. trutta*: −15%). Beyond this threshold, species richness did not recover to initial levels after 10 years, across all parameter combinations.

3.4 | Impact of predation pressure on the asymptomatic carrier and pathogen emergence

It was predicted that high predation pressure was required on *P. parva* to prevent their population from surviving (Figure 6a). Uninfected consumer

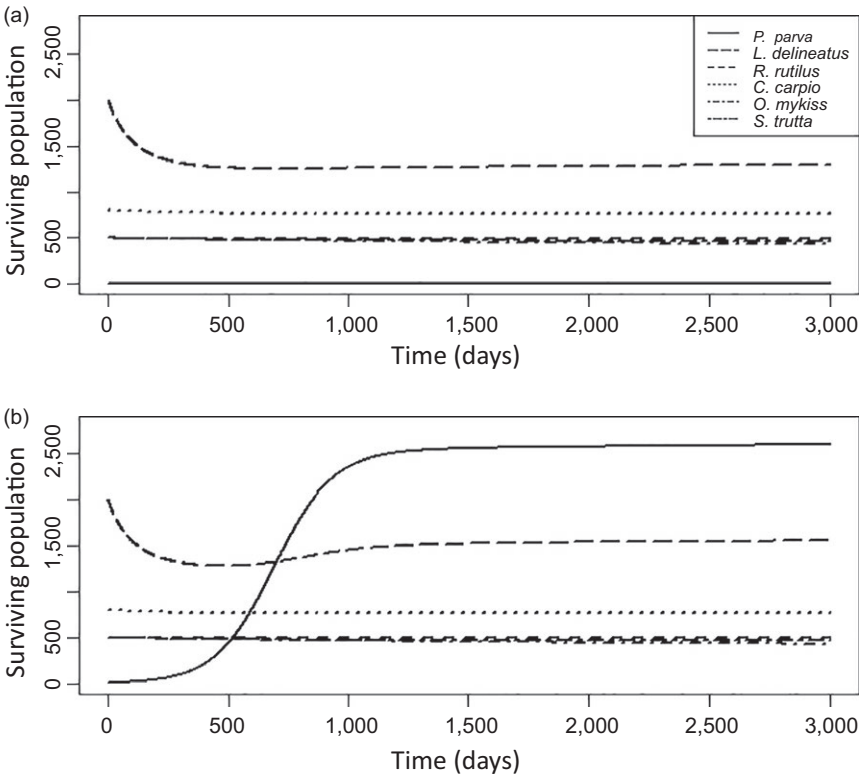

FIGURE 4 Species abundance in a community without any disturbance (a) and in a community where topmouth gudgeon *Pseudorasbora parva* was introduced, but not the pathogen (b). The community remained stable without any disturbances, but when the asymptomatic carrier was introduced, minor declines were observed in *Leucaspius delineatus*, *Rutilus rutilus*, and *Cyprinus carpio*

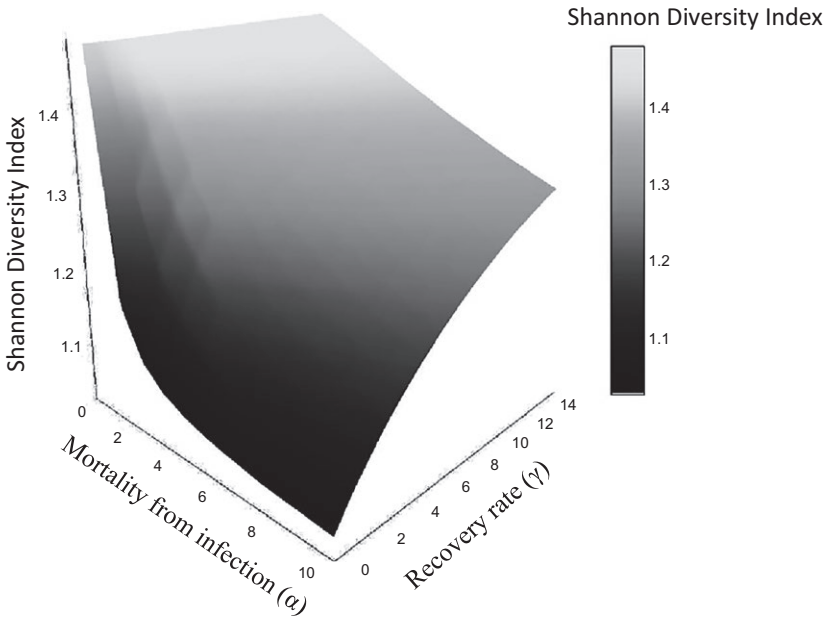

FIGURE 5 The Shannon diversity index of the resident fish community 10 years after the asymptomatic carrier introduction, across a range of infection parameter values for secondary consumer species (mortality from infection, recovery rate, and incubation rate). The initial Shannon diversity index before the introduction of the asymptomatic carrier was 1.43. Other species' parameters were kept constant. Low mortality from infection maintained high diversity across all values of recovery rate. Higher recovery rates in secondary consumers helped maintain high diversity. The model demonstrated that species introduction can have a long-term effect on community diversity, and resistant species at high trophic levels can sustain community diversity after a disturbance

populations were predicted to reduce the population of *P. parva* more than infected consumers, because their density was not reduced from infection. When predation was kept constant but the secondary consumer density was increased, *P. parva* populations decreased but still maintained a sustainable population (Figure 6b). In a realistic ecological setting, the consumers would also have predated on *L. delineatus* to the same degree, which was predicted to lead to its population crashing as predation pressure increased (at coefficients of 1×10^{-5} and 4×10^{-6},

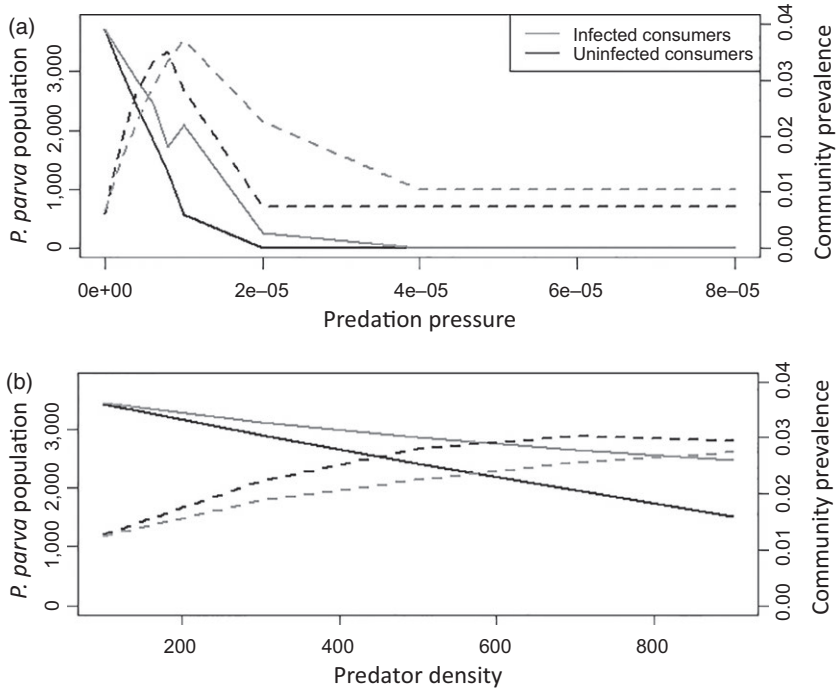

FIGURE 6 (a) *Pseudorasbora parva* abundance 10 years post-introduction under a range of predation pressures, while predator density was kept constant at 500. High predation pressure prevented the introduced species from surviving both in the presence of infection (grey) and the absence of infection (black). Uninfected consumers were able to reduce the population of *P. parva* more than infected consumers at equal predation pressures (due to higher survival). The average prevalence of infection in the community (dashed lines) demonstrates that high predation also decreases the prevalence of infection in the community; (b) Altering the population density of the secondary consumers (100–900) while keeping predation pressure constant ($p = 4 \times 10^{-6}$), also showed that high abundance of predators can reduce the asymptomatic carrier population. However, high predator density was not effective at lowering the prevalence of infection in the community, for both infected and uninfected consumers

for infected and uninfected consumers, respectively). Predating on *P. parva* controlled its population growth but did not prevent pathogen emergence, as the average disease prevalence in the community ranged from 0.9% under high predation pressure, to 3% under high predator density (Figure 6). At high levels of predation, the pathogen remained at lower levels in the environment compared to low predation (around 4,000 spores compared with >20,000, respectively).

Predator density and predation pressure were varied simultaneously to test the parameter space in which biocontrol of the infection was feasible, as indicated by changes in environmental zoospore level (Figure 7). When predation pressure was high, fewer resistant predators were required to reduce pathogen density in the environment.

4 | DISCUSSION

The SEIR model was used to investigate the role of predation on host community resilience to disease emergence of a generalist pathogen and predicted that secondary consumer species would inhibit the population growth of *P. parva*, the introduced asymptomatic carrier. However, low to intermediate levels of predation of the asymptomatic host would not prevent the transmission of *S. destruens* to the other fishes in the community Whilst high predation pressure on *P. parva* by secondary consumers did suppress its population growth and also contained the pathogen, it was also predicted that this could result in the extirpation of resident species that occupied similar trophic niches and had a high susceptibility to the pathogen (*L. delineatus*, in this community). Thus, predictions suggested that although biotic resistance through predation could alleviate the consequences of asymptomatic carrier introduction (Britton, 2013) and, to a certain degree, prevent pathogen spread, it would also impact community structure.

Community structure and diversity was also predicted to be influenced by the type of infection, with the model predicting that both chronically infected and resistant secondary consumers would help to maintain higher host population levels than acutely infected consumers. This could be a result of a dilution effect by resistant secondary consumers, as they acted as a dead end host for the pathogen (Keesing, Holt, & Ostfeld, 2006). The slow-growing nature of chronic infections is predicted to delay the onset of secondary infections, as they release infectious propagules after a longer incubation period compared to acutely infected hosts. However, some susceptible host populations were predicted to continuously experience chronic exposure to pathogen propagules in the environment. In practice, it is problematic to disentangle the respective effects of mortality from infection and recovery in modelling parameter relationships, as both are ways to end the period of infection. Virulent infections can act more rapidly than less virulent infections, with hosts either recovering or dying within a shorter time (van Baalen, 1998). For example, *B.*

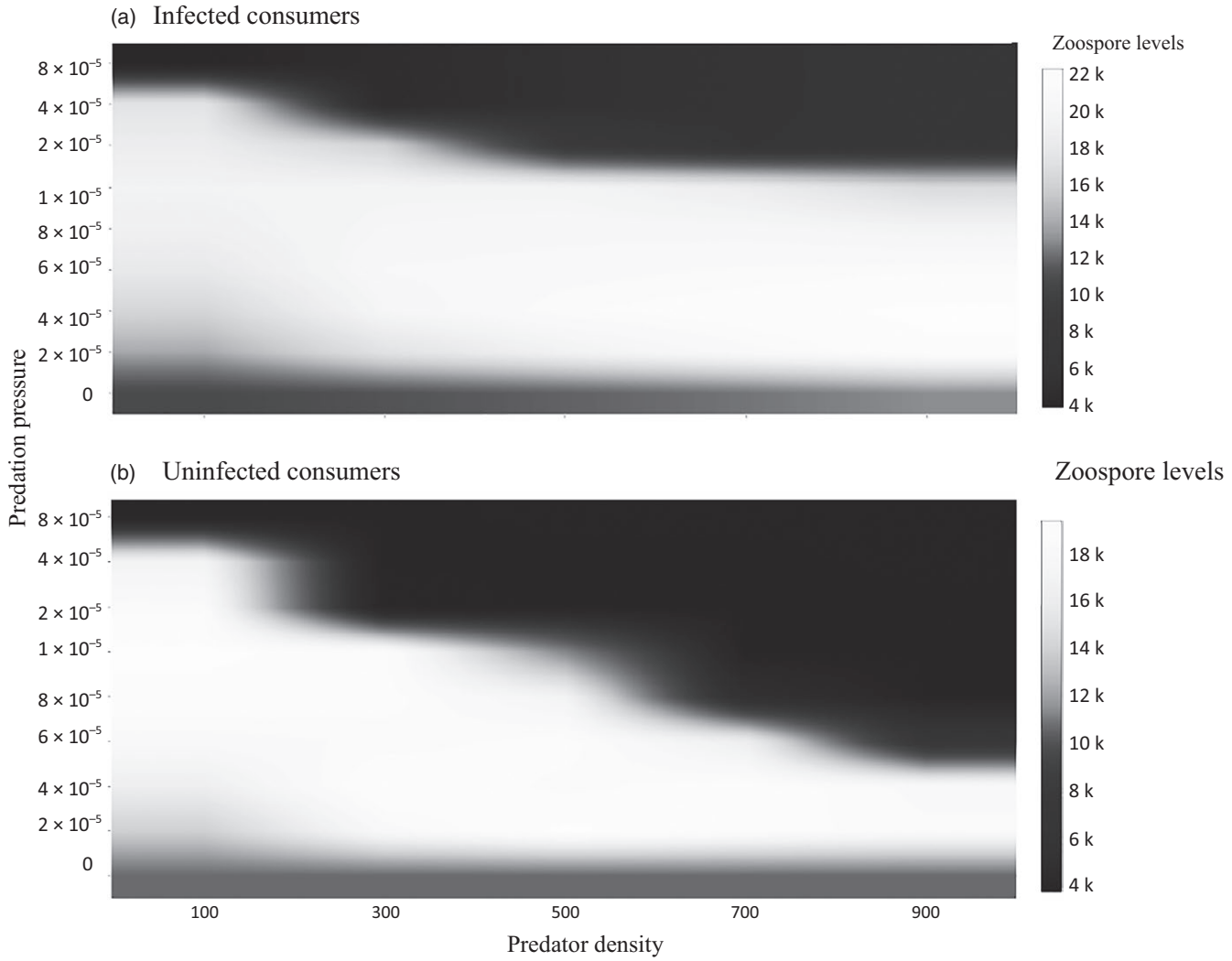

FIGURE 7 Environmental spore levels at different degrees of predation pressure and secondary consumer populations for both infected consumers (a) and uninfected consumers (b). The conditions under which infection control is possible through predation are indicated by the darker areas of the heatmap. Uninfected (or resistant) consumers can control the infectious propagules in the environment at lower initial densities and at lower predation pressure than infected (or susceptible) consumers

dendrobatidis, a relatively similar pathogen to *S. destruens*, has a short duration of infection (i.e., high recovery rate) and a high mortality rate (Daszak et al., 1999). Alternatively, other pathogen systems demonstrate an inverse relationship between mortality from infection and recovery rate (Anderson & May, 1982).

Where the community is comprised of susceptible hosts, high host diversity can amplify the spread of generalist pathogen infection (Mihaljevic, Joseph, Orlofske, & Paull, 2014). However, increasing the diversity of a host community to include non-competent hosts has successfully limited the spread of Lyme disease (Ostfeld & Keesing, 2011) and rodent-borne haemorrhagic fevers (Mills, 2006). This dilution effect caused by resistant or dead-end hosts has been used to limit the spread of infection and create herd immunity (Johnson & Thieltges, 2010). In our study, resistant secondary consumers (dead end hosts) helped maintain the community diversity and reduced the levels of infectious propagules in the environment, compared to susceptible consumers. Previous work has demonstrated that where hosts (or the pathogen itself) are pre-dated on by other species, disease prevalence can decrease (Roy & Holt, 2008; Searle et al., 2013). For example, there is some evidence that *B. dendrobatidis* outbreaks are correlated with low levels of macro-invertebrates, which usually prey on the chytrid spores (Schmeller et al., 2013; Strauss & Smith, 2013). Here, *S. destruens* persisted in the environment and in susceptible host populations. Susceptible species in the community are expected to continue transmitting *S. destruens* independently of the asymptomatic carrier population, once the disease has established within their populations. As predator densities increased so did the prevalence of infection, even in communities with uninfected secondary consumers due to continuing secondary infections of susceptible hosts. For example, in model scenarios with high densities of resistant predators, mean community disease prevalence was 3%. Large populations at high trophic levels would also disrupt community function and food web structure (Eby, Roach, Crowder, & Stanford, 2006). This could explain why disease outbreaks are observed in some communities and not others,

highlighting the importance of looking at the specific community composition and interactions like predation, not only at biodiversity levels, when predicting whether a community can recover from an epidemic.

The effects of infection can lead to new or altered interactions between predators and prey, even extending to species that are not infected (Hatcher, Dick, & Dunn, 2006). Based on evidence from other host-pathogen systems, it is likely that infected individuals would be more heavily preyed on than uninfected individuals (Lafferty, 1992; Moore, 2002). Higher prevalence of an acanthocephalan parasite *Echinorhynchus truttae* in invasive *Gammarus pulex* compared to native *Gammarus duebeni celticus* decreased intraguild predation of the native amphipod, and slowed the process of species replacement (MacNeil et al., 2003). Quantifying this additional effect of infection in a community context is difficult, but important to understand. Future research questions in this system should examine the wider community effects of infection beyond the clinical aspects. First, this pathogen may have potential economic implications for fisheries and sport fishing lakes. *S. destruens* has been detected in farmed sea bass (a €400 million annual farming industry in the Mediterranean), with potentially severe consequences as they showed a prevalence of 67% (Ercan et al., 2015). In addition to the economic effects, *S. destruens* has been associated with population declines of endemic European fishes such as *Petroleuciscus smyrnaeus*, *Squalius fellowesii*, and *Oxynoemacheilus* sp. (Ercan et al., 2015). The risk *S. destruens* poses to conservation efforts of vulnerable species and ecosystems is significant, especially considering the wide host range and transmission ability of the pathogen.

A biocontrol approach through predation has already been tested in this model system, demonstrating that *P. parva* populations could be reduced by perch *Perca fluviatilis* (a secondary consumer species) (Davies & Britton, 2015). However, this could potentially also increase the risk of negative top-down effects on aspects of ecosystem functioning (Eby et al., 2006). Predation as a form of biocontrol has been explored in a number of systems (Maharaj, Appleton, & Miller, 1992; Snyder & Wise, 1999; Symondson, Sunderland, & Greenstone, 2002), but often there are unforeseen limitations and repercussions, such as non-target species declining due to direct predation or knock-on effects resulting from the shifting community structure (Knapp, Corn, & Schindler, 2001; Louda, Pemberton, Johnson, & Follett, 2003). In the *S. destruens* model, high predation was predicted to reduce the *P. parva* population, but was also predicted to increase predation of *L. delineatus* to the point of extirpation. Unwanted effects of species removal on the remaining species in the community have been observed in several systems (Bergstrom et al., 2009; Zavaleta, Hobbs, & Mooney, 2001), and can be difficult to control retrospectively. This includes the ecological release of species previously controlled by the removed species, such as the expansion of rat populations after eradicating feral cats in New Zealand (Zavaleta, 2002).

In the context of disease eradication, if the pathogen is already established in several host species or in the environment, removing one host species will not eliminate the pathogen (Wobeser, 2002), but could actually contribute to disease re-emergence. As shown by our work, this management approach may not be effective in mitigating the effects of the pathogen. If predator enhancement is selected as a preferred management approach to minimise the transmission and impact of the pathogen then the predator species should be a resistant specialist predator that will prey on the target species and then only a low number of other species and, ideally, none (Louda et al., 2003; Messing & Wright, 2006; Symondson et al., 2002). Based on our model results, high species-specific predation that does not require high densities of predators may be an effective method to counteract the effects of both introduced host and pathogen. In all cases, more data on the prevalence of infection in the wild will aid management efforts in deciding where and to what degree interventions are needed. The findings and methodology in this work can be applied to multiple systems to establish the risks of pathogen emergence and more importantly, predict a given community's resilience to infectious outbreaks.

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AUTHORS' CONTRIBUTIONS

F.A.-S., D.A. & R.G. conceived the ideas for the study. D.A. & R.G. collected the data. F.A.-S. & B.R. analysed the data and developed the model. F.A.-S., D.A., R.B., R.G. & B.R. wrote and revised the manuscript. All authors contributed critically to the drafts and gave final approval for publication.

DATA ACCESSIBILITY

Experimental data are available on figshare: https://doi.org/10.6084/m9.figshare.5082055.v1 (Al-Shorbaji, Andreou, & Gozlan, 2017).

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

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