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SPECIAL ISSUE: PARASITES IN AQUATIC ECOLOGY

Epidemics in native species influence the outcome of a species invasion

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Abstract

Invasive species can have large effects on native communities. When native and invasive species share parasites, an epidemic in a native species could facilitate or inhibit the invasion. We sought to understand how the incidence and timing of epidemics in native species caused by a generalist parasite influenced the success and impact of an invasive species. We focused on North American native and invasive species of zooplankton (*Daphnia dentifera* and *Daphnia lumholtzi*, respectively), that can both become infected with a fungal parasite (*Metschnikowia bicuspidata*). In a laboratory microcosm experiment, we exposed the native species to varying parasite inocula (none, low, high) and two invasive species introduction times (before or during an epidemic in the native species). We found that the invasive species density in treatments with the parasite was higher compared to uninfected treatments, though only the early invasion, low-parasite and uninfected treatments exhibited significant pairwise differences. However, invasive resting eggs were only found in the uninfected treatments. The density of the native species was lowest with a combination of the parasite present, and the invasive species introduced during the epidemic. Native infection prevalence in these treatments (late invasion, parasite present) was also higher than prevalence in treatments where the invasive species was introduced before the epidemic. Therefore, the timing of an invasion relative to an epidemic can affect both the native and invasive species. Our results suggest that the occurrence and timing of epidemics in native species can influence the impacts of a species invasion.

Keywords *Daphnia dentifera* · *Daphnia lumholtzi* · Invasive species · *Metschnikowia bicuspidata* · Disease outbreak

Introduction

Invasive species play a major role in altering native community structure and can reduce the abundance of native species (Prenter et al. 2004; Pimentel et al. 2005; Searle et al. 2018). The consequences of successful biological invasions include the reduction of native biodiversity, loss of community structure, and alterations of ecosystem processes (Novak 2007). Core research on invasion ecology focuses on which communities are susceptible to invasions, what consequences occur as a result of invasive species, and what factors determine whether a population or ecosystem will resist or succumb to invasions (Novak 2007; Gill et al. 2021). When considering factors that affect the successful establishment of non-indigenous organisms, natural enemies such as parasites and predators are a productive area of research (Prenter et al. 2004; Dunn and Hatcher 2015; Faillace et al. 2017; Searle et al. 2018).

The success and effects of biological invasions can depend on biotic properties such as parasitism (Price et al. 1988; Prenter et al. 2004; Searle et al. 2018). For example, invasive species may benefit from the lack of evolutionary history in the range of native species where the invasion is occurring via the absence of parasites that can infect invasive species (i.e., enemy release; Keane and Crawley 2002; Colautti et al. 2004) or co-introduction of parasites that infect both the native and invasive species (i.e., novel

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weapons; Callaway and Ridenour 2004). Additionally, when native and invasive species share parasites, the less susceptible host species can experience a competitive advantage in the presence of the parasite, facilitating or inhibiting the invasion (Price et al. 1988; Settle and Wilson 1990; Prenter et al. 2004; Searle et al. 2018). Parasites can, therefore, directly alter native species vulnerability to invasive species, and indirectly affect native–invasive interactions (Price et al. 1988; Prenter et al. 2004; Dunn et al. 2012; Dunn and Hatcher 2015).

Infectious disease can also influence invasions via changes to native host density. For example, an epidemic in native species can reduce native abundance and result in an increase in available resources, making it easier for the invasive species to establish (Prenter et al. 2004; Havel et al. 2015; Searle et al. 2018). However, the size of an epidemic can affect how the native species density changes (Hudson and Dobson 1989; Hall et al. 2011; Searle et al. 2018) and this can potentially alter the resources available for the invasive species. For example, if large epidemics in native species result in very low native population abundance, then large epidemics may lead to high invasion success (Settle and Wilson 1990; Tuttle et al. 2017). Additionally, if an invasive species is introduced to a native community at the beginning of an epidemic, before the parasite has substantially reduced native host density, then the effect of the parasite on invasion success may be lower than if the invasion occurs later in the epidemic.

Epidemics in native species may also alter invasions by impacting the chances that an invasive species becomes infected with shared parasites (Price et al. 1988; Knevel et al. 2004). For example, if an invasive species arrives at a new community while the density of infected native individuals is high, then the invasive species may be likely to become infected with the causal parasite (Elton 1958; Price et al. 1988; Knevel et al. 2004). Thus, an invasion that occurs immediately before or during a large epidemic could lead to reduced invasion success if the invasive species is highly susceptible to the parasite. For both mechanisms by which epidemics in native species can alter invasion success (reduced native abundance and chances of invasive species becoming infected), the size of an epidemic and the timing of the invasion during this epidemic (i.e., before, during, or after the epidemic) could have large effects on invasion success.

We sought to understand how the size and timing of epidemics in native species influence the ability of an invasive species to successfully establish. Toward this goal, we asked 1) does the severity of an epidemic in a native species influence the success of an invasive species and 2) does the timing of the introduction of an invasive species during an epidemic influence its success and effects? We performed a microcosm experimental trial manipulating parasite infection and invasive species introduction times during a simulated invasion.

Materials and methods

Study system

We used a model host–parasite system involving native and invasive freshwater crustaceans (*Daphnia dentifera and Daphnia lumholtzi,* respectively), and a fungal parasite (*Metschnikowia bicuspidata*). *Daphnia dentifer*a (hereafter: "the native species") are native to North America and are dominant grazers found in lakes and ponds in Indiana, USA (Midwest; Hebert 1995). *Daphnia lumholtzi* (hereafter: "the invasive species") are native to lakes in Africa, Australia, and Asia and are invasive competitors of the native species (Hebert 1995; Kolar et al. 1997). The invasive species has spread throughout much of the USA and can alter the community structure of native zooplankton (Benzie 1988; Havel and Hebert 1993; Kolar et al. 1997). Both the native and invasive species can be reared in asexual isofemale lines and can be infected by *M. bicuspidata* as a result of incidental ingestion. Infection causes reduced feeding rates and mortality (Ebert et al. 2000; Searle et al. 2016, 2018). Infected *Daphnia* release spores of the parasite into the environment upon death (Ebert et al. 2000).

Experimental setup and design

Our experiment was a 3×2 design with three levels of parasite inocula (none, low: 75 spores/mL added, and high: 150 spores/mL added) and two introduction times of the invasive species (early, late) for a total of six treatments. Each treatment was replicated 10 times for a total of 60 experimental units (microcosms). The experiment was then divided into two blocks so that sampling could be conducted over 2 days, with half of the replicates from each treatment in each block.

To begin the experiment, we filled 1L beakers with 800 mL well water and introduced 3 native adults from each of 5 isofemale lines (Online Resource Table S1; day 1), for a total of 15 native animals per beaker. *Daphnia* were fed 20.0×10^6 cells of *Ankistrodesmus falcatus* algae per beaker per day for the duration of the experiment. Native populations were allowed to establish prior to the parasite treatment exposure. Parasite spores were obtained by blending previously infected animals and added on day 7 for block 1 and day 8 for block 2. For our invasion treatments, two invasive individuals from a single clone (age 10–12 days: Online Resource Table S1) were added to each "early invasion" beaker on day 17 for block 1 and day 18 for block 2, and to each "late invasion" beaker on day 31 for block 1 and day 32 for block 2. These times were chosen to represent

an invasion occurring before the native species experienced a large increase in infection (early invasion) or at the peak of infection during the epidemic (late invasion; based on timing from Searle et al. 2018). To estimate resource availability, 2 days after the addition of the invasive species into a beaker, we estimated chlorophyll levels in the beaker's water by taking a 1 mL sample from 25 mm below the water's surface and recording raw fluorescence units with a fluorometer (Turner Trilogy) using an in vivo module. These values were converted to concentration of *Ankistrodesmus falcatus* cells using a standard curve created by solutions with known concentrations of this algae.

Beakers were maintained under a 16:8 light–dark cycle. All beakers received a full water change weekly beginning on day 3. The experiment was ended 7 weeks (49 days) after the introduction of the invasive species; days 66–67 for the early invasion and days 80–81 for the late invasion.

A population census was conducted weekly, immediately before water changes, beginning on day 10. For the census, after homogenizing the contents of each beaker, a 100 mL subsample was removed and viewed under a stereomicroscope. Animals were enumerated according to species (native or invasive), age (juvenile or adult), sex (female or male), and infection status (infected or uninfected) before being returned to their beaker. Infection can be identified in live animals because the spores of *M. bicuspidata* cause the hemolymph to appear opaque, while healthy individuals have hemolymph that is clear (Duffy and Hall 2008). At the end of the experiment, beakers were filtered completely through 333 µmm mesh and the number of *D. lumholtzi* ephippia were counted as another measure of invasion success. Ephippia contain diapausing eggs that Cladocera produce under some environmental conditions (Cáceres 1998).

Statistical analysis

All data were analyzed in R (version 4.3.0; R Core Team 2022), and the data and code needed to reproduce the figures and statistical tests can be found on the repository osf.io (https://osf.io/n69r7/). For each of the following *Daphnia* models, we used data collected from the first 7 weeks after the early (weeks 3–9) and late invasion (weeks 5–11) treatments (Figs. 1, 2). To estimate the effect of parasite and invasion timing treatments on both the total and infected native densities and to account for overdispersion of the data, we used a negative binomial generalized linear mixed effect model (GLMM; package: *lme4*, function: *glmer.nb*; Bates et al. 2015) with parasite treatment (i.e., uninfected, low, or high), invasion timing (i.e., early or late), and their interactions as predictor variables. We included beaker identity as a random effect to account for the correlation between the experimental week and the densities of *Daphnia* in each experimental group. Though we attempted to include experimental week as a fixed effect in each of the models in this study (see code on osf.io), to keep the terms as consistent as possible among our models, we mapped experimental week as a random effect as its inclusion as fixed effects either caused models to fail to converge or did not qualitatively alter our findings. We used similar models to those described above for the native species to test the treatment effects on the total and infected invasive densities, but instead used a zero-inflated Poisson GLMM (package: *glmmTMB*, function: *glmmTMB*, ziformula = ~; Brooks et al. 2017) to account for the high frequency of zero counts in the data.

Our models for infection prevalence of each species were similar to those used for density, except we used a binomial distribution with a logit link function. Models were tested for over and under dispersion using a simulation-based test (package: *DHARMa*, function: *testDispersion*; Hartig 2022), and every model but one—the native density model which was significantly under dispersed ($p = 0.048$) and showed signs of singular fit—exhibited no evidence of under or over dispersion. When we removed beaker identity as a random effect from the native density model, we no longer observed a significant effect of under dispersion ($p = 0.064$) or singular fit. However, as the inclusion of beaker identity did not qualitatively alter our findings, we retained beaker identity in the model for more direct comparison with our other models. All models met the assumption that the random effect was normally distributed.

To compare ephippia counts and native density at the time of invasion across treatments, we used quassi-Poisson generalized linear models (GLM) with invasion timing, parasite treatment, and their interactions as predictor variables. Models with the same parameters were used to determine if there were significant differences among treatments in native density on the day of the invasion. Finally, we used two-way ANOVAs with parasite treatment, invasion timing, and their interactions to determine if chlorophyll levels differed between each experimental group on the dates of early and late invasion. In addition to presenting the untransformed β coefficients, standard errors, and p values in our models, we also present the untransformed effect sizes as estimated marginal means (EMM) and 95% confidence intervals (package: *emmeans*, function *emmean*; Lenth 2022) for the relevant comparisons that returned a multiple-comparison adjusted (Tukey method) p-value < 0.05 (package: *emmeans*, function: *pairs*).

Results

Our low and high parasite inocula treatments were essentially the same in terms of host densities and infected host densities (Figs. 1, 2) indicating that both of our

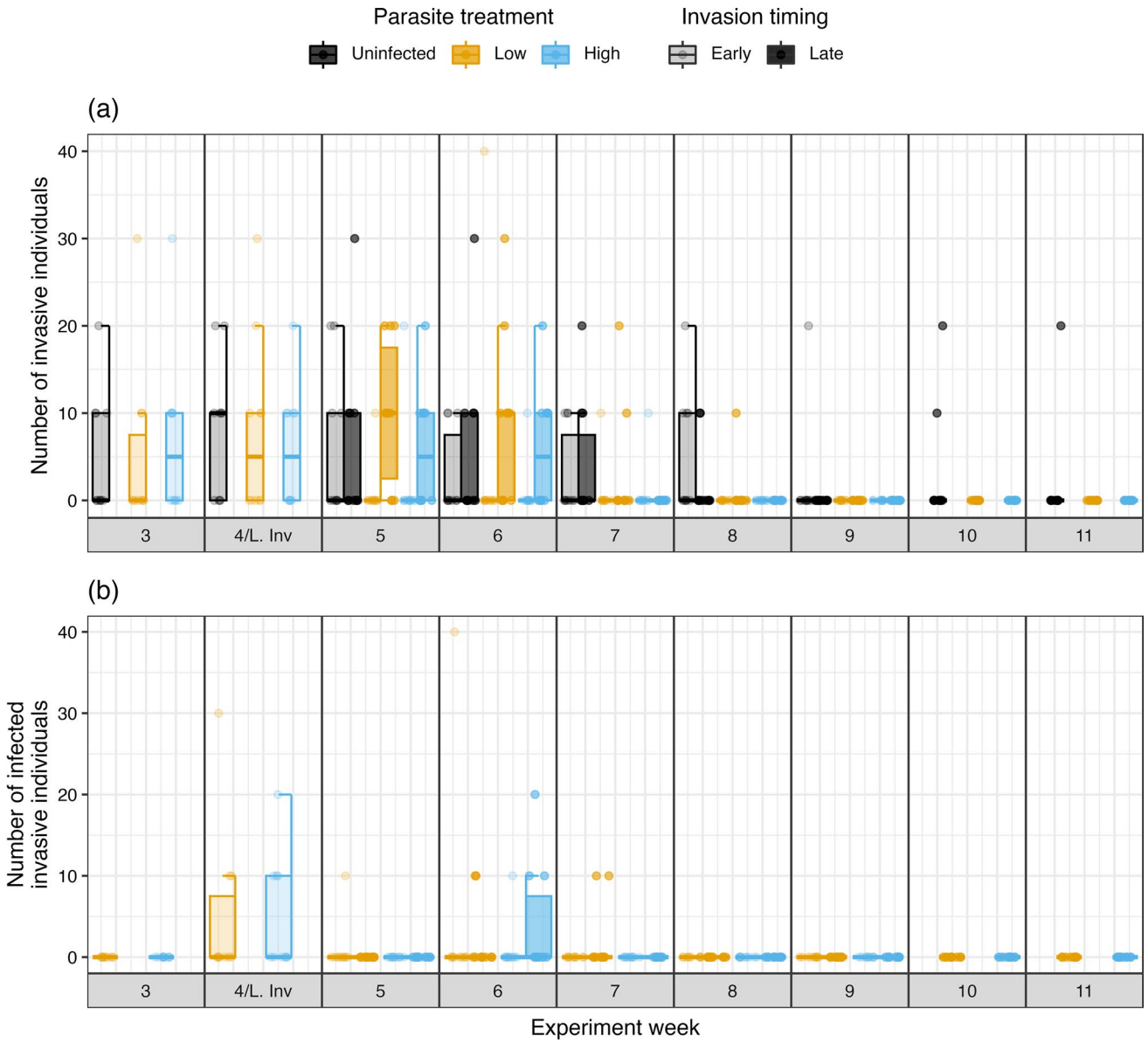

Fig. 1 The (**a**) number of invasive and (**b**) infected invasive individuals throughout the experiment, distinguished by parasite treatment and timing of invasion. Invasion treatments are separated into early invasion (left-hatching with transparent points and shading) and late invasion (right-hatching with opaque points and shading), while parasite treatment is indicated by color. Data from the first two weeks of the experiment are omitted as invasive individuals were not counted until one week after each invasion; early occurring in week 2 ('2/E. Inv–Early invasion') on the x-axis) and late in week 4 ('4/L. Inv–Late invasion') on the x-axis) of the experiment. The points represent individual observations, and the error bars represent standard error (±SE). There were 10 replicates for each treatment

parasite-exposed treatments led to a similar proportion of infected individuals. The two invasion times occurred when we planned, where the early invasion occurred when infected native densities were low, and the late invasion occurred close to the peak of infected native densities (Fig. 2).

Invasive and native species density

We found significant effects of the low ($\beta = 1.50$, SE = 0.61, $p = 0.015$) and high parasite treatment ($\beta = 1.22$, SE = 0.60, $p = 0.04$) on invasive density when compared to the uninfected group, though the only significant pairwise comparisons occurred between the uninfected and low-parasite treatment in the early invasion group (Uninfected: EMM = 1.43, [0.05–2.79]; Low: EMM = 1.93, [1.04–4.42]; $p = 0.04$; Fig. 3A), and the early and late invasion timing within the low-parasite treatment group (Early: EMM = 2.93, [1.43–4.42]; Late: EMM = 1.18, [− 0.19–2.55]; $p = 0.01$; Fig. 3A). We did not find significant effects of invasion timing, the interaction between

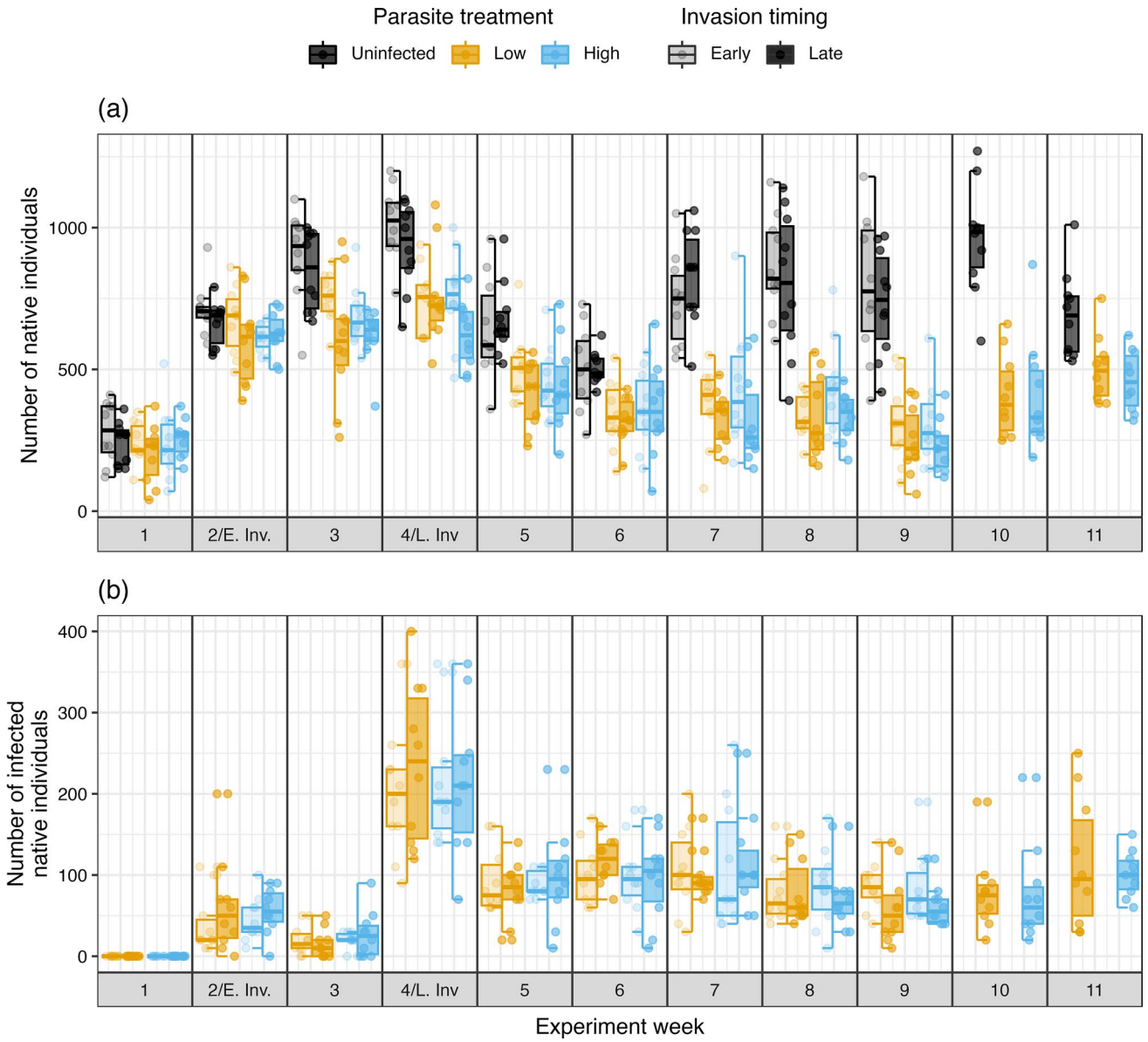

Fig. 2 The (**a**) number of native and (**b**) infected native individuals throughout the experiment, distinguished by parasite treatment and timing of invasion. Invasion treatments are separated into early invasion (left-hatching with transparent points and shading) and late invasion (right-hatching with opaque points and shading), while parasite treatment is indicated by color. Time of invasion is denoted on the x-axis labels with early occurring in week 2 ('2/E. Inv–Early invasion') and late in week 4 ('4/L. Inv–Late invasion') of the experiment. The points represent individual observations, and the error bars represent standard error (±SE). There were 10 replicates for each treatment

infection treatment and invasion timing, or in any other, single-treatment pairwise comparison on invasive density (Fig. 3A). For native density, both parasite treatments had a negative effect on density (Low: $\beta = 0.54$, SE = 0.06, $p < 0.001$; High: $\beta = 0.51$, SE = 0.06, $p < 0.001$; Fig. 3B) when compared to the uninfected group. Though we did not find an overall effect of invasion timing on native density ($p = 0.67$) or an effect within the uninfected group ($p = 0.67$), our pairwise comparisons of each parasite treatment found that late invasion had a negative effect on density in both the low (Early: EMM = 6.14, [5.97–6.31]; Late: EMM = 5.98, [5.81–6.15], $p = 0.01$; Fig. 3B) and high parasite treatment groups (Early: EMM = 6.17, [6.00–6.34]; Late: EMM = 5.98, [5.81–6.15], $p = 0.002$; Fig. 3B). Finally, we found that native densities on the day of invasion were significantly lower in the early treatments ($\beta = 0.27$, SE = 0.07, $p < 0.001$) and among each of the pairwise comparisons between the low, high, and uninfected treatment groups (Uninfected: EMM = 6.70, [6.63 – 6.77]; Low: EMM = 6.57, [6.50–6.64]; High:

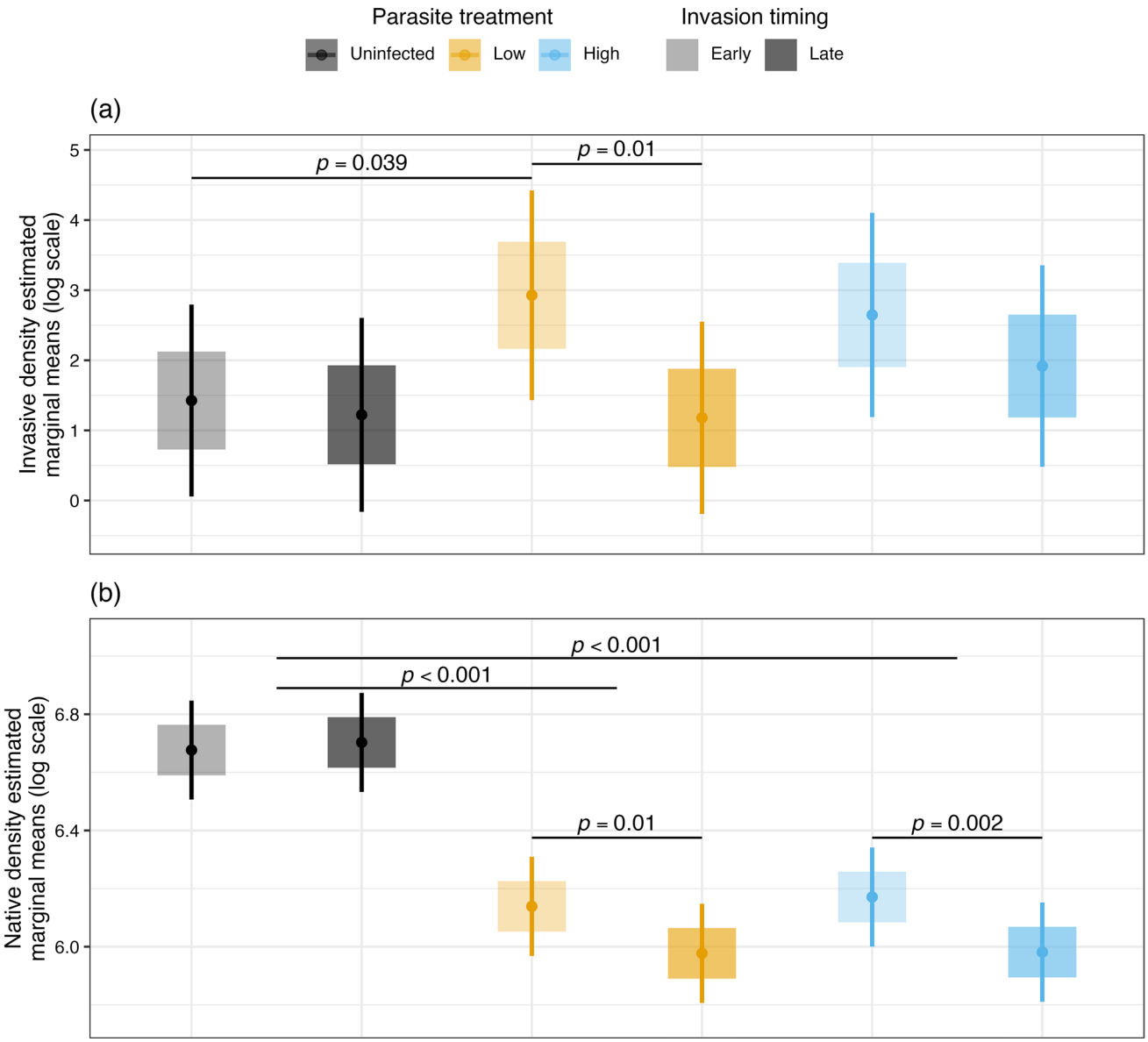

Fig. 3 Estimated marginal means of parasite treatment and invasion timing on the total density of (**a**) invasive and (**b**) native species. Our models found differences between the uninfected and low-parasite treatments during the early invasion and between the early and late invasion in the low-parasite treatment in the (**a**) invasive density. We found no other differences between the other treatments in the (**a**) invasive species density or between the (**b**) native density in the two invasion treatments in the uninfected group. However, the (**b**) native density in the late invasion group in each of the low and high parasite treatments had significantly fewer native individuals than their counterparts in the early invasion group. Log scale estimated marginal means (points), the standard error of these means (shaded boxes), and 95% confidence intervals (vertical lines) are presented for each parasite treatment (colors) and invasion timing (transparent vs. opaque shading). There were ten replicates for each treatment

EMM = 6.42, [6.35–6.50]; $p < 0.05$). However, only in the uninfected group did native density on the day of the invasion differ between early (EMM = 6.57, [6.46–6.67]) and late invasion (EMM = 6.84, [6.75–6.93]) treatments ($\beta = 0.27$, SE = 0.07, $p < 0.001$; Online Resource Fig. S1). Overall, the parasite treatments affected both invasive

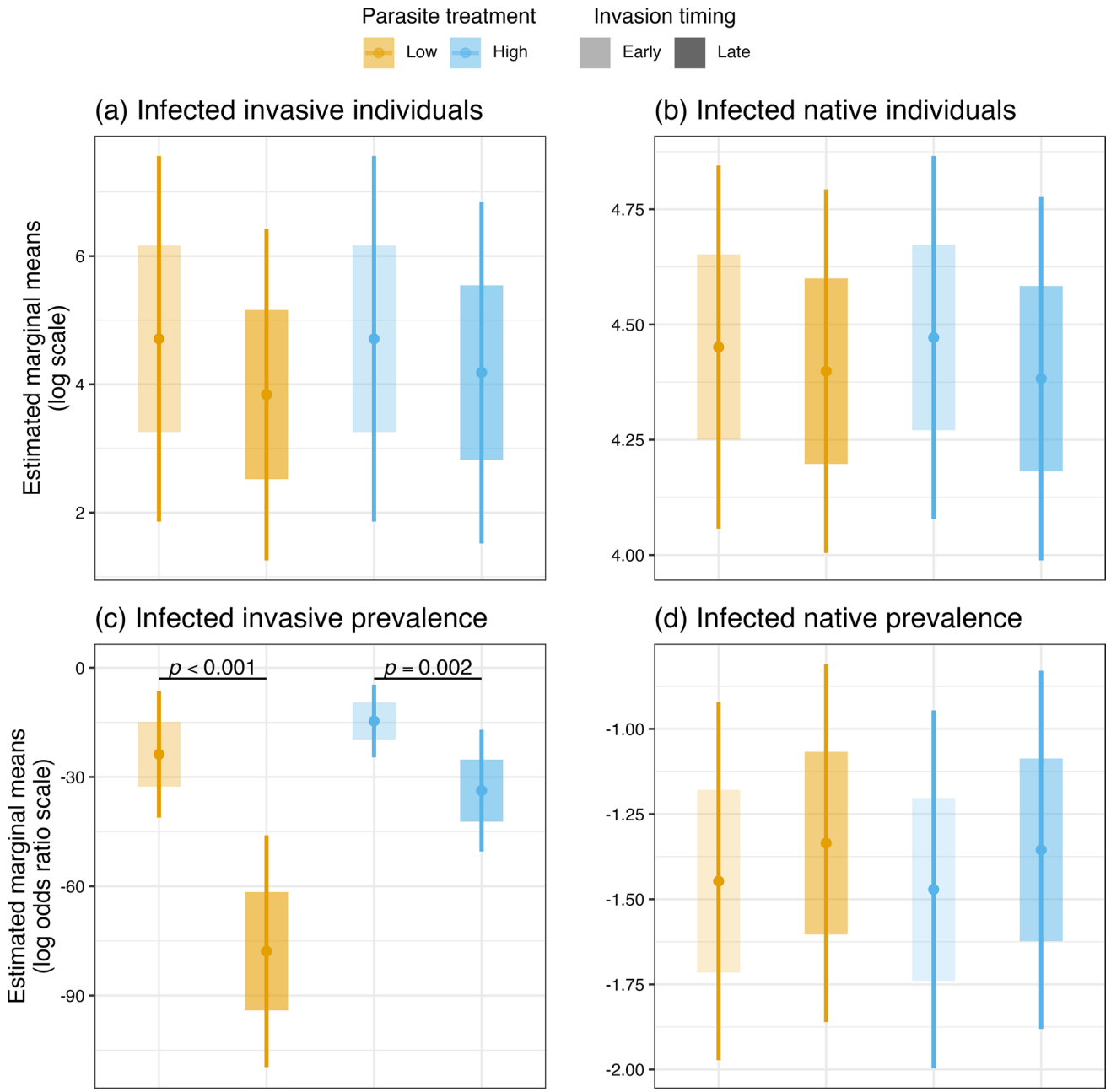

Fig. 4 Estimated marginal means of parasite treatment and invasion timing on the number of (**a**) invasive infected individuals, (**b**) native infected individuals, (**c**) invasive infection prevalence, and (**d**) native infection prevalence. Our models found no significant effect of the parasite and invasion treatments on (**a**) invasive infection density or (**b**) native infection density and (**d**) native infection prevalence. However, we did find that (**c**) invasive individuals in the early invasion treatment for both the low and high parasite treatments had a significantly higher infection prevalence than the late invasion treatment. Log scale (top panels–**a** , **b**) or log odds ratio scale (bottom panels–**c**, **d**) estimated marginal means (points), the standard error of these means (shaded boxes), and 95% confidence intervals (vertical lines) are presented for each parasite treatment (colors) and invasion timing (transparent vs. opaque shading). There were ten replicates for each treatment

and native densities, specifically the late invasion had a negative effect on native densities that were exposed to parasites.

Invasive and native species infected density and infection prevalence

None of the treatments or their interactions yielded significant effects on the density of infected individuals of either

species (Fig. 4A, B). We did find that the invasive infection prevalence was significantly higher in the early invasion treatment overall ($\beta = 54.1$, SE = 11.37, $p < 0.001$) and for both the low (Early: EMM = − 23.8, [− 41.2 to − 6.39]; Late: EMM = − 77.8, [− 109.6 to − 46.01]; $p < 0.001$; Fig. 4C) and high parasite treatment groups (Early: EMM = − 14.6, [− 24.6 to − 4.66]; Late: EMM = − 33.7, [− 50.4 to − 17.05]; $p = 0.002$; Fig. 4C). However, we did not find significant effects of treatments or their interactions on native infection prevalence (Fig. 4D). Overall, in the early invasion treatments, there was higher invasive infection prevalence in both parasite treatments, while no treatments affected the native infection prevalence or the number of native or invasive infected individuals or the invasive infection prevalence.

Ephippia counts

We identified all ephippia as being from the invasive species. The number of ephippia found at the end of the experiment in the uninfected parasite treatment differed significantly from both the low ($\beta = 2.51$, SE = 0.82, $p = 0.004$) and high parasite treatments ($\beta = 1.81$, SE = 0.60, $p = 0.004$), though we did not observe an effect of invasion time or the interactions between the treatments on ephippia counts (Fig. 5). Overall, there were more invasive ephippia in the uninfected treatments compared to the low and high parasite treatments.

Chlorophyll concentrations

Our two-way ANOVAs indicated that the chlorophyll/algae concentrations on the early invasion date did not differ between parasite treatment groups ($F_{2,54} = 0.03$, $p = 0.470$), but did differ between invasion timing treatments ($F_{1,54} = 6.99$, $p = 0.011$); pairwise comparisons indicated that this difference were driven largely by the low-parasite treatment group (Early: EMM = 1002, [535–1469]; Late: EMM = 1705, [1238–2172]; $p = 0.038$) and high parasite treatment group (Early: EMM = 994, [528 – 1461]; Late: EMM = 1627, [1160–2094]; $p = 0.060$) but not by the uninfected group (Early: EMM = 1211, [744–1678]; Late: EMM = 1383, [917–1850]; $p = 0.603$; Online Resource Fig.

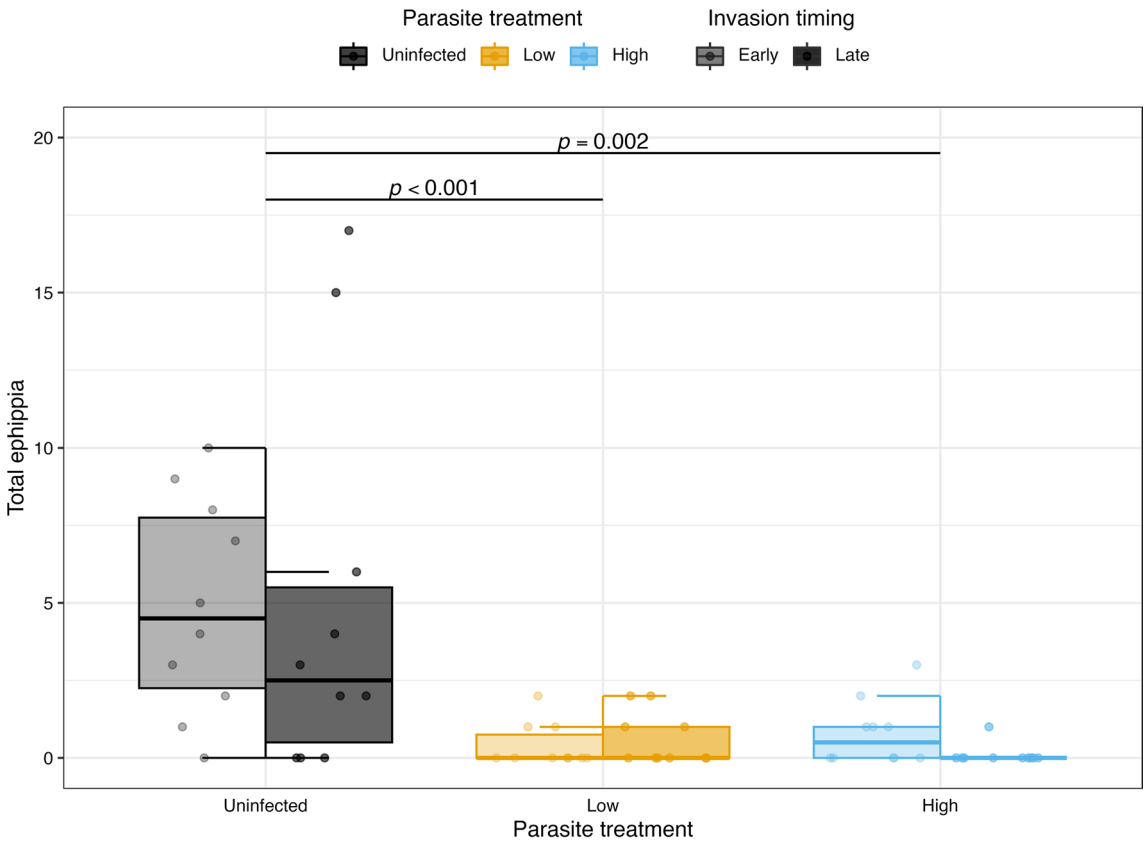

Fig. 5 Total ephippia counts from the invasive species in the uninfected group at the end of the experiment differed significantly from low and high parasite treatment groups, but we did not observe an effect of invasion timing on ephippia counts. Invasion treatments are separated into early invasion (left-hatching with transparent points and shading) and late invasion (right-hatching with opaque points and shading), while parasite treatment is indicated by color. The points represent individual observations, and the error bars represent standard error (± SE)

S2). We found no significant effect of parasite treatment, invasion timing, or their interactions on the concentration of algae during the late invasion date (Online Resource Fig. S3). Overall, in the early invasion time, there was more chlorophyll in the late invasion and low-parasite treatment.

Discussion

While there are many mechanisms by which disease can influence invasions (Prenter et al. 2004), relatively little is known regarding how epidemics in native species can influence invasions. In our laboratory microcosm experiment, we found that the invasive species density in treatments with the parasite was higher compared to uninfected treatments, but invasive resting eggs were only found in uninfected treatments (Figs. 3a, 5). The density of the native species was reduced by the presence of the parasite, which could have been caused by a reduction in native fecundity or an increase in mortality due to infection, which has been shown in other studies (Civitello et al. 2015; Searle et al. 2018). Additionally, the timing of the invasion impacted native density, where the late invasion treatments had lower native densities than the early invasion treatments when the parasite was present (Fig. 3). Additionally, invasive species infection prevalence was impacted by the timing of the introduction of the invasive species, where there was higher infection prevalence during the early invasion time in both parasite treatments (Fig. 4c). This interaction between the parasite and invasion timing on the native species density indicates that the invasive species could have aided in the overall decline in the native density by competing for resources, or the epidemic had already caused mass infection and death in the native species by the time the invasive species arrived. Together, these results imply that parasitism and the timing of a species invasion can interact to affect the success and impact of an invasion.

The parasite reduced the native species densities (Figs. 2a, 3b), which also led to a slight increase in the densities of the invasive species (Fig. 1, 3a). Therefore, our results indicate that when the parasite caused infection and decreased native density, there were more resources (e.g., space and supplied algae) for the invasive species to increase its densities (Fig, S2; Prenter et al. 2004; Searle et al. 2018). It should be noted that, while we found a main effect of both parasite treatments on the invasive species density, the effect sizes were relatively small for invasive density compared to the effects of treatment on native density. Specifically, invasive density in the low-parasite treatment was higher during the early invasion when compared to the late invasion treatment. In addition, during the early invasion, the invasive density was significantly higher in low-parasite treatment compared to the uninfected treatment. These results indicate that the invasive species was able to moderately increase their population size to utilize the space and the supplied food resources made available when the native species declined in density due to the parasite. However, the invasive species density showed a decline over time in the parasite treatments (Fig. 1a), suggesting that the long-term effects of the parasite on the invasive species may be negative or neutral. Throughout the course of the experiment, 10% of all the native species were infected, and 19% of all the invasive species were infected. Because the invasive species were becoming infected at a relatively high rate, they may be unable to benefit in the long term from the reduction in native densities caused by the parasite, resulting in both species suffering the effects of the epidemic together.

Measuring both ephippia and the invasive species density is useful for measuring the success of an invasion. Typically, with a higher invasive density, it can be assumed that the invasive species is establishing well in the new environment. Density represents immediate reproduction, as more individuals are created that utilize more resources, take up space, and diminish native densities. Because ephippia are dormant and can remain viable for years, they can be long-lived and extend the generation time of the invasive species (Panov et al. 2004), which can induce population perseverance in volatile environments, and demonstrates investment in future populations (Cáceres 1998). In this experiment, we found more ephippia from the invasive species in the uninfected treatments (Fig. 5), even though invasive species density was lower in these treatments compared to the infected treatments. This pattern of higher ephippia densities in the absence of the parasite may be caused by the higher densities of the native species in these treatments, which led to crowding and triggered production of resting eggs (Smith et al. 2009). Using both invasive species density and ephippia as estimates of invasion success in aquatic crustacean studies can be useful tools for predicting immediate and future invasion success.

There was a significant effect of the parasite on the native densities (Figs. 2a, 3b). In particular, the native densities were lower in the late invasion treatments compared to the early invasion treatments, but only when the parasite was present (Fig. 3b). Therefore, while the parasite alone reduced native densities, the parasite plus late invasion combination was particularly detrimental to native populations. Although not a significant effect, this interaction may be caused by the negative effects of both the parasite and the invasion occurring simultaneously. Native populations may have been able to mitigate the negative effects of the invasive species before the epidemic occurred but were unable to deal with the invasion when also experiencing the epidemic. In addition, the invaders may depress the host density more when resource density is lower and interspecific competition is harsher due to the lower algae densities during the late time point

compared to the early time point (Figs. S2 & S3). Therefore, the timing of an invasion during an epidemic in the native species may affect how it is influenced by the invasion.

Resources are often a key driver of invasion success (Prenter et al. 2004; Guo et al. 2015). In particular, resources are typically considered a crucial component to an invasive species' prosperity in a novel environment, because they help the species establish, reproduce quickly, and compete with the native species (Byers 2002; Guo et al. 2015). It is predicted that invasive species should establish and proliferate more readily in communities with more resources (McKenzie and Townsend 2007; Guo et al. 2015). In our study, we found that in the early invasion date in the low-parasite treatment, there was substantially more chlorophyll levels (a measure of algal food resources) in the late invasion compared to the early invasion (Online Resource Fig. S2). In our experimental design, the resource population was constantly supplied and can lead to detritus accumulation (animals were fed 20.0×10^6 algae cells daily), which may not occur in many natural settings. Since the parasite (*M. bicuspidata*) reduces feeding rates and causes death in infected individuals, it can be assumed that there was a higher algae concentration in the late invasion, because the *Daphnia* either were infected or dying in that treatment (shown in Figs. 1, 2). We did not find a detectable difference in chlorophyll levels across the other treatments in the late invasion date (Online Resource Figs. S2 & S3).

Laboratory controlled experiments can be powerful tools for manipulating epidemics and invasions, but present differences or limitations in comparison to natural systems. In our microcosm design, we were unable to test how the severity of an epidemic alters species invasions as we had planned due to the low and high parasite treatments having similar infection rates. We also supplied and kept the resources (algae) constant throughout the experiment which may have minimized the possibility of cascading effects across the food chain (Online Resource Figs. S2 & S3). In the natural environment, the concentration of resources would fluctuate on their own, potentially mediating species responses to competition and parasitism. However, future studies could investigate these complex interactions between resources, invasions, and disease and identifying the general mechanisms of these interactions for other systems (Walsman et al. 2022).

Disease is known to influence invasion success (Prenter et al. 2004); however, the ways in which parasites in native species and the timing of the invasion affects invasion success are relatively unknown. Our study suggests that native epidemics and the timing of an invasion may interact to affect both the native and invasive species and affect the success of an invasion (e.g., Fig. 3). Understanding how disease and invasive species interact to influence invasions will be helpful with determining whether certain populations will be at risk of invasion and how disease will impact invasions in aquatic communities.

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Author contribution statement CLS and KLJ conceived and designed the experiments. KLJ, PEB, BNH performed the experiments. BDH analyzed the data. PEB wrote the manuscript; all other authors provided editorial advice.

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Data availability The datasets used and/or analyzed during the current study are available on the repository osf.io (https://osf.io/n69r7).

Code availability The code developed during this study is available on the repository osf.io (https://osf.io/n69r7).

Declarations

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval Ethics approval was not required for this study.

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