

# Selective predation, parasitism, and trophic cascades in a bluegill–*Daphnia*–parasite system

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**Abstract** As disease incidence increases worldwide, there is increased interest in determining the factors controlling parasitism in natural populations. Recently, several studies have suggested a possible role of predation in reducing parasitism, but this idea has received little experimental attention. Here, I present the results of an experiment in which I manipulated predation rate in large field enclosures to test the effects of predation on parasitism using a bluegill predator–*Daphnia* host–yeast parasite system. Based on previous work showing high bluegill sunfish selectivity for infected over uninfected *Daphnia*, I anticipated that predators would reduce infection levels. Contrary to expectations, predation did not reduce infection prevalence. Instead, there were large epidemics in all treatments, followed by reductions of host density to very low levels. As *Daphnia* density decreased, phytoplankton abundance increased and water clarity decreased, suggesting a parasite-driven trophic cascade. Overall, these results suggest that selective predation does not always reduce infection prevalence, and that parasites have the

potential to drastically reduce host densities even in the presence of selective predators.

**Keywords** Indirect effects · *Metschnikowia bicuspidata* · *Daphnia dentifera* · *Lepomis macrochirus* · *Spirobacillus cienkowski*

## Introduction

Parasites are small and inconspicuous; as a result, there has been a tendency to overlook and ignore them (Ebert 2005; Lafferty et al. 2006; Mittelbach 2005). Yet, there is an increasing awareness that parasites are key members of food webs, both influenced by and influencing other members of the food web (e.g., Hatcher et al. 2006; Hudson et al. 1992a; Johnson and Chase 2004; Lafferty 2004; Ostfeld and Holt 2004). This suggests that understanding the causes and consequences of parasitism in natural populations will be accomplished only by integrating parasites into food webs, a task whose importance is increased by the worldwide increase in disease incidence (Harvell 2004; Lafferty et al. 2004; Ward and Lafferty 2004).

The idea that predation can reduce parasitism is pervasive, but has only recently received serious attention from ecologists. Most theoretical studies support this idea (Anderson and May 1981; Hall et al. 2005a; Packer et al. 2003), but a recent study found that, in some cases, predation can increase disease prevalence (Choisy and Rohani 2006). Several field studies support a role of predation in decreasing parasitism, based on correlative evidence for a negative relationship between infection prevalence and predation rate (Duffy et al. 2005; Hudson et al. 1992b; Johnson et al. 2006; Lafferty 2004). However, in at least

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one case, selective culling increased disease prevalence (Donnelly et al. 2003). Experimental studies on the effect of predation on parasitism have been limited (Packer et al. 2003). These experiments, all conducted on terrestrial parasitoid systems, have generally found decreased parasitism in the presence of predators (Colfer and Rosenheim 2001; Matsumoto et al. 2003; Snyder and Ives 2001, 2003; Stiling and Moon 2005), but in one case parasitism increased (Cardinale et al. 2003). Despite this limited and contradictory evidence, the ability of predation to “keep the herds healthy” has been suggested as a conservation strategy (Packer et al. 2003).

At its core, this healthy herds hypothesis relies on a trophic cascade involving an intraguild predator (Ostfeld and Holt 2004): predation should reduce parasitism, which, in turn, should increase host/prey density. Certainly, there is evidence that parasites can affect host population density (e.g., Anagnostakis 1987; Ebert et al. 2000; Hochachka and Dhondt 2000; Hudson et al. 1998; Lips et al. 2006). However, the full cascade—that is, predation decreasing parasitism and increasing host density—has only been studied with parasitoids, which may not show the same patterns as regular parasites (Lafferty et al. 2006). Even in these parasitoid systems, evidence for beneficial cascades is mixed. A recent meta-analysis found that, on average across all studied predator–parasitoid–host systems, the effect of predators on hosts was zero, with many studies finding a negative effect of predation on host density (Rosenheim and Harmon 2006).

In many cases, we are also interested in the next step of a potential cascade: the effect of host density on a lower trophic level, generally primary producers. The potential for parasite-driven trophic cascades is the idea underlying many biological control programs (Polis et al. 2000), and there is some experimental support for trophic cascades in parasitoid–herbivore–plant food chains in terrestrial systems (e.g., Colfer and Rosenheim 2001; Matsumoto et al. 2003; Stiling and Moon 2005). There is also evidence from stream communities for parasite-driven trophic cascades (Kohler and Hoiland 2001; Kohler and Wiley 1997; Whiles et al. 2006). To my knowledge, the potential for parasite-driven cascades in lake populations remains unstudied; this is somewhat surprising given the centrality of trophic cascade theory in lake ecology (Carpenter et al. 1987; Polis et al. 2000).

Here, I present the results of a study in which I tested the effects of predators on parasitism in a bluegill–*Daphnia*–microparasite system. This system is one in which the healthy herds effect should be especially strong. The healthy herds hypothesis relies on the indirect effect of predation outweighing the direct effect, which is more likely to occur when predation is selective on infected prey and the parasite is virulent (Packer et al.

2003). Bluegill sunfish are extremely selective on infected *Daphnia* (Duffy 2006; Duffy et al. 2005; Johnson et al. 2006). In addition, the parasite, *Metschnikowia*, is highly virulent, reducing fecundity and lifespan, even in the absence of predation (Duffy 2006; Ebert et al. 2000; Hall et al. 2006). Thus, I predicted that increased predation would decrease infection prevalence and increase *Daphnia* density. Because *Daphnia* are important herbivores in lake food webs and are often key links in trophic cascades (Lampert and Sommer 1997; Wetzel 2001), I was also interested in asking whether any parasite-driven changes in *Daphnia* density cascaded to the phytoplankton. I predicted that any parasite-driven decreases in *Daphnia* density should lead to increased phytoplankton abundance.

## Materials and methods

I set up an enclosure experiment, using a replicated regression approach (Cottingham et al. 2005), to test the effects of selective predation by bluegill sunfish (*Lepomis macrochirus*) on the dynamics of the yeast pathogen *Metschnikowia bicuspidata* and its host, *Daphnia dentifera*. *Daphnia dentifera* is a common species in the plankton of temperate lakes in North America (Hebert 1995). *Metschnikowia* is highly virulent, reducing fecundity by 25–50% and decreasing lifespan by up to 80% (Duffy 2006; Ebert et al. 2000; Hall et al. 2006). *Metschnikowia* is horizontally transmitted; uninfected *Daphnia* become infected after ingesting the needle-shaped ascospores, which puncture the gut wall (Ebert 2005; Green 1974). Infected hosts do not recover. Upon death, they release asci into the environment (Ebert 2005; Green 1974). There is no evidence for behavioral changes in *Metschnikowia*-infected *Daphnia*—infected *Daphnia* still undergo diel vertical migration and there is no spatial patchiness to infections in lakes (Hall et al. 2005b). *Metschnikowia* is a common parasite of *D. dentifera* populations in southwest Michigan (Cáceres et al. 2006). Epidemics are defined, as in previous studies (Cáceres et al. 2006; Duffy et al. 2005), as occurring when infection prevalence surpasses 1%.

Bluegill sunfish are the dominant vertebrate predators of zooplankton in lakes in southwest Michigan (Tessier and Woodruff 2002). In previous studies, we have shown that they prey highly selectively on infected *D. dentifera*, including *D. dentifera* infected with *Metschnikowia* (Duffy 2006; Duffy et al. 2005). Previously, I measured selectivity of bluegill predation on *Metschnikowia*-infected *D. dentifera* in multiple lakes and multiple years. I found that Chesson’s alpha, a measure of selectivity (Chesson 1983), ranged from 0.84–0.95 (Duffy 2006). This is vastly

different from neutral selectivity ( $\alpha = 0.5$ ) and it approaches the theoretical maximum ( $\alpha = 1.0$ ).

This experiment was run in highly productive Wintergreen Lake (Kalamazoo County, MI, USA) in July–August 2005. The enclosures were 1-m-diameter cylindrical polyethylene bags that were sealed at the bottom, suspended from wooden frames, and buoyed by Styrofoam floats. Enclosures were 5 m deep, yielding a volume of 5,000 L. This type of enclosure has been used frequently to study interactions between *Daphnia* and bluegill (e.g., González and Tessier 1997; Mittelbach and Osenberg 1993). Ten enclosures were set up in the deep part of Wintergreen Lake, arranged in two blocks with five enclosures each. On June 30, the enclosures were filled with water from the lake. The water was pumped through an 80  $\mu\text{m}$  mesh net to remove zooplankton. While *Metschnikowia*-infected *D. dentifera* were occasionally observed in Wintergreen Lake in 2004, no infections were observed in 2005, so it is unlikely that *Metschnikowia* spores were added to enclosures from lake water. On July 1 and 5, the bags were stocked with *D. dentifera*; because it was not possible to collect *D. dentifera* from Wintergreen without also collecting many other species, the bags were stocked with *D. dentifera* from nearby Warner Lake. On July 8, lab-reared *Metschnikowia* spores were added to the bags at a concentration of 1,900 spores/L. This is lower than spore densities measured in lake populations at the start of epidemics ( $\sim 5,000$  spores/L; S.R. Hall, unpubl. data). Bags were mixed daily with a Secchi disk for the first week after spores were added to ensure they remained suspended in the water column.

Bluegill from Warner Lake were added to the enclosures on July 15. Predation rate was regulated by restricting the area of the enclosures in which the fish could feed, as well as the time they spent in the enclosure. This yielded five predation treatments, covering a range from 0–1 bluegill/m<sup>2</sup>. Bluegill densities in Wintergreen Lake in the past decade have ranged from 0–0.67 bluegill/m<sup>2</sup> (Mittelbach et al. 2006), so the experimental predation treatments cover an ecologically relevant range of predation. There were two replicates per predation treatment, one replicate in each block of enclosures. To restrict the area in which fish could feed, fish were confined in 20 mm mesh monofilament cages of either 0.25 or 0.5 m diameter. Cages were 3.5 m long and were suspended from the top of the water column in the center of the enclosure. The higher predation treatments had one or two fish in a 0.5 m diameter cage, yielding fish densities of 0.5 and 1 m<sup>-2</sup>; bluegill densities were determined by multiplying the number of bluegill in a cage by the surface area of the cage (e.g., 2 bluegill  $\times$  0.5 m<sup>-2</sup> cage = 1 bluegill m<sup>-2</sup>). The two lower predation treatments (0.25 and 0.175 m<sup>-2</sup> fish density) had one

fish in a 0.25-m-diameter cage in the enclosure every day (0.25 m<sup>-2</sup> fish density) or every other day (0.175 m<sup>-2</sup> fish density). One of the 0.175 m<sup>-2</sup> treatment enclosures failed; *D. dentifera* collapsed in this bag as soon as spores were added, and so this bag was excluded from analysis. There was also a no-predation treatment. This treatment contained a fish in a 0.25-m-diameter cage, to control for indirect effects of fish caused by kairomones (Lass and Spaak 2003), but the fish was enclosed in a smaller cage made of bridal veil, which did not allow *Daphnia* to pass through. The two no-predation treatment fish were replaced every three days to prevent mortality from starvation. The mean ( $\pm 1$  SE) standard length of fish used in the experiment was 58.0 mm ( $\pm 1.1$ ). Any fish that died (a total of 11 fish over the entire experiment) were replaced within 24 h.

This experiment was designed to test the effects of selective fish predation on infection prevalence and *D. dentifera* density. I sampled the *D. dentifera* population every other day from July 4 to July 8, on July 12, and then every three days beginning July 17 (replicate 1) or July 18 (replicate 2). Samples were collected using whole water-column tows of a Wisconsin net, and two samples were collected per enclosure; overall, this represented a mortality of  $\sim 1$ –4% of the population every three days (or, on average, 0.003–0.01 day<sup>-1</sup>), which is unlikely to have had substantial effects on *D. dentifera* population dynamics. Because *D. dentifera* tend to aggregate, enclosures were mixed well with a Secchi disk prior to sampling to homogenize the *D. dentifera* distribution. I examined these samples to quantify infection prevalence and *D. dentifera* density. On early sampling dates (before July 21), only one sample was examined for infection prevalence, as densities were high and an accurate estimate could be obtained from a single sample. *D. dentifera* were examined live under a stereomicroscope at 25–50 $\times$  magnification. *Daphnia* are normally transparent. However, after infection with *Metschnikowia*, they become opaque and asci are visible inside their bodies, allowing infections to be detected without dissection.

This experiment also provided the opportunity to measure cascading effects of predation and parasitism. Thus, I measured Secchi depth, which indicates water clarity (Lampert and Sommer 1997) every sampling day, and measured chlorophyll *a*, which indicates phytoplankton abundance, every third sampling day. Secchi depth was measured prior to mixing with the Secchi disk. Chlorophyll *a* samples were also collected prior to mixing with the Secchi disk using a 3-m-long tube that collected an integrated sample of the epilimnion. Chlorophyll *a* samples were kept cool and in the dark and processed immediately upon their return to the lab. Samples were filtered onto GF/F Whatman glass microfiber filters,

extracted in 95% ethanol, and measured with narrow-band fluorimetry.

### Data analysis

I tested for effects of predation on infection prevalence and host density using a repeated measures ANOVA, with predation modeled as a continuous variable. The analysis was done using Proc Mixed in SAS 9.1, following methods in Littell et al. (2006). Arcsine square root-transformed proportion infected or log *D. dentifera* density was used as the dependent variable. Prior to analysis, densities of zero were replaced with  $0.001 \text{ L}^{-1}$ , which is one-half the lowest nonzero value measured (and, therefore, provides an estimate of the minimum sampling resolution). I ran the analysis twice, once with arcsine square root-transformed overall proportion infected (i.e., no. infected *D. dentifera*/total *D. dentifera* in sample) as the dependent variable, and once with arcsine square root-transformed proportion infected adults (i.e., no. infected adult *D. dentifera*/total adult *D. dentifera* in sample). Infections are primarily observed in adults, so patterns in infection prevalence might be clearer when only adults are considered. The model included time, predation treatment, and their interaction as fixed effects, enclosure as the repeated measures subject, and an autoregressive (1) covariance structure. This analysis was repeated with the data split into two groups: with predation (composed of the seven enclosures with predators) and no predation (composed of the two no-predation enclosures). I also tested for a correlation between predation level and overall infection prevalence, adult infection prevalence or host density during the peak in infection prevalence (July 29–30; see “Results”), using Spearman rank correlations in SAS 9.1.

Based on the results of these analyses, I wanted to ensure that fish had been effective predators. Thus, I tested for a correlation between predation treatment and log *D. dentifera* density on July 21–22; at this point, fish had been in the enclosures for one week, but the parasite should not have begun driving host dynamics. I carried out a Spearman rank correlation using SAS 9.1.

I used multivariate autoregression, MAR(1), to assess the interactions between infection prevalence, *D. dentifera* density and Secchi depth, and to determine the impact of predation on each of these variables (Ives et al. 2003). Secchi depth was used instead of chlorophyll *a* because it was measured every sampling day, whereas chlorophyll *a* was measured only every third sampling day. Secchi depth and chlorophyll *a* were strongly and significantly negatively correlated ( $r = -0.67$ ,  $P < 0.001$ ). This approach assumes that the dynamics of *D. dentifera*, phytoplankton (Secchi depth), and infection prevalence are given by the model:

$$x_1(t) = b_{10} + b_{11}x_1(t-1) + b_{13}x_3(t-1) + c_1u_{\text{trt}} + E_1(t) \quad (1)$$

$$x_2(t) = b_{20} + b_{12}x_2(t-1) + b_{22}x_2(t-1) + c_2u_{\text{trt}} + E_2(t) \quad (2)$$

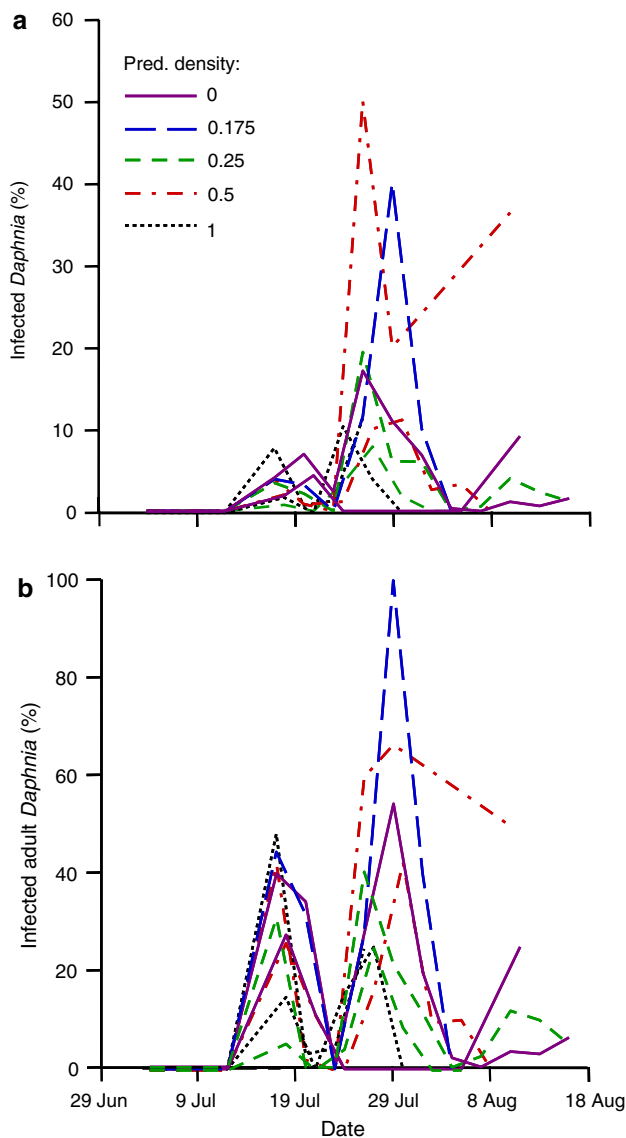
$$x_3(t) = b_{30} + b_{33}x_3(t-1) + c_3u_{\text{trt}} + E_3(t) \quad (3)$$

where  $x_1(t)$  is the log-transformed *D. dentifera* density at sample  $t$ ,  $x_2(t)$  is the Secchi depth,  $x_3(t)$  is the proportion infected, and  $u_{\text{trt}}$  is the predation level for each of the treatments. Because I was interested in comparing the magnitudes of effects among the different variables, I standardized each variable by dividing by its standard deviation taken across all enclosures and all samples. This standardization makes the coefficients  $b_{ij}$  ( $i, j = 1, 2, 3$ ) and  $c_i$  measure interactions as effect sizes among the interacting variables. Thus, the effect of  $x_j$  (e.g., infection prevalence) on the change in  $x_i$  (e.g., *D. dentifera* density) between samples is given by  $b_{ij}$ , and the effect of predation treatment on  $x_i$  is given by  $c_i$ .  $E(t)$  is a vector of process errors that has a multivariate normal distribution with mean vector  $\mathbf{0}$  and covariance matrix  $\Sigma$ . Parameters in all three equations were estimated simultaneously, using the data from all nine enclosures. These conditional least squares (CLS) estimates minimize the sum of squared differences between the value observed at time  $t$  and those predicted by the MAR(1) model given the observed state of the system at time  $t-1$ . Confidence intervals of 95% were obtained by bootstrapping (Ives et al. 2003). For bootstrapping, the initial values for the three variables were set to the observed values in each of the nine enclosures. Data sets of 2,000 bootstraps were then simulated by iterating Eqs. 1–3, with values of  $E_i(t)$  sampled with replacement from the CLS residuals; to maintain the correlation structure in the covariance matrix  $\Sigma$ , triplets of residuals taken from the same time in the same enclosure were sampled as a set.

### Results

There were large epidemics in all enclosures, with two peaks in infection prevalence (Fig. 1). Overall infection prevalence reached 1–8% (mean = 4%) in the first epidemic peak and 5–48% (mean = 31%) in the second epidemic peak; these infection prevalences match what is observed in lake populations (Cáceres et al. 2006). There was no significant effect of predation treatment on infection prevalence (overall infection prevalence:  $F_{(1,7)} = 1.09$ ,  $P = 0.33$ , Fig. 1a; adult infection prevalence:  $F_{(1,7)} = 0.20$ ,  $P = 0.68$ , Fig. 1b; see “Electronic Supplementary Material” for full ANOVA tables). There was a significant time  $\times$  treatment interaction (overall infection prevalence:  $F_{(12,57)} = 2.19$ ,  $P = 0.02$ ;





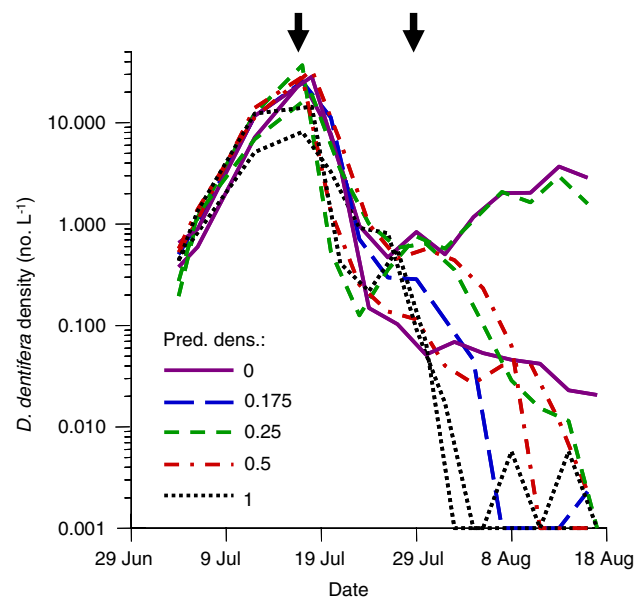
**Fig. 1a–b** Percent of **a** population and **b** adults of *D. dentifera* (prey) infected with *Metschnikowia* (yeast parasite) in lake enclosures with different bluegill (predator) densities (no. m<sup>-2</sup>) in Michigan, USA during July–August 2005. The five predation treatments (0, 0.175, 0.25, 0.5 and 1.0 m<sup>-2</sup>) are indicated with differently dashed lines (see legend); there are two replicates per predation treatment (except for 0.175 m<sup>-2</sup>, for which there is only one replicate; see “Methods”). Data from all replicates are shown

adult infection prevalence:  $F_{(12,57)} = 2.23$ ,  $P = 0.02$ ). The analysis with the treatments aggregated into predation and no-predation groups did not find a significant effect of predation (overall infection prevalence:  $F_{(1,7)} = 0.30$ ,  $P = 0.60$ ; adult infection prevalence:  $F_{(1,7)} = 0.06$ ,  $P = 0.81$ ; see “Electronic Supplementary Material” for full ANOVA tables). There was no significant time  $\times$  treatment interaction (overall infection prevalence:  $F_{(12,57)} = 0.53$ ,  $P = 0.88$ ; adult infection prevalence:  $F_{(12,57)} = 0.48$ ,  $P = 0.92$ ). The

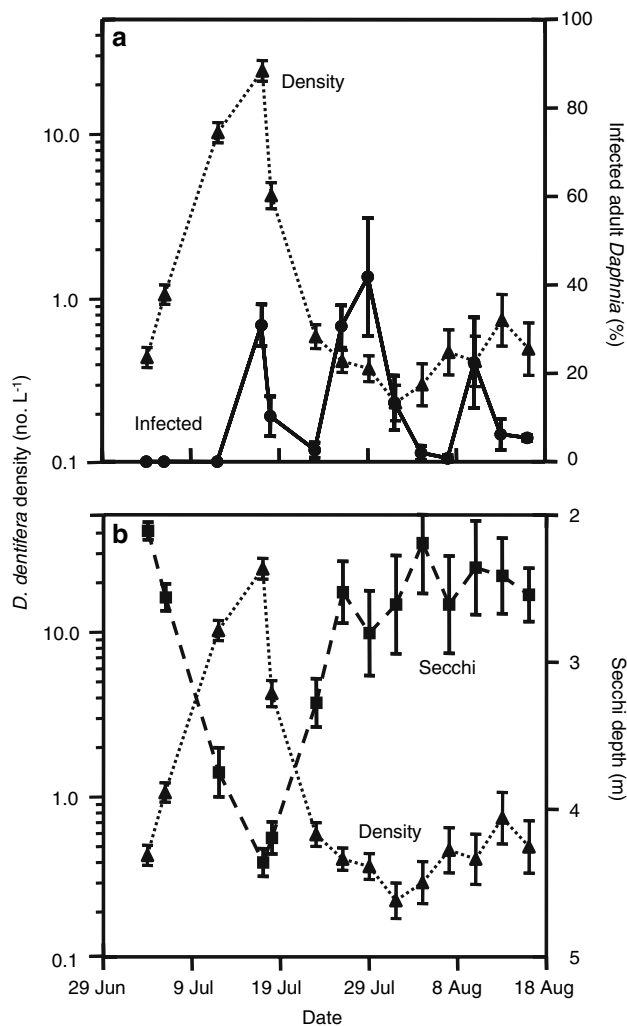
regressions also failed to find an effect of predation: the CLS estimate of the effect of fish predation on change in infection prevalence was not significant (CLS estimate: 0.01,  $P > 0.42$ ), and there was no significant correlation between predation treatment and peak infection prevalence ( $-0.49 < r < -0.33$ , all  $P > 0.26$ ).

There was also no significant main effect of predation on *D. dentifera* density ( $F_{(1,7)} = 3.14$ ,  $P = 0.12$ ; see “Electronic Supplementary Material” for full ANOVA table); there was a significant time  $\times$  predation treatment interaction ( $F_{(13,91)} = 2.49$ ,  $P = 0.01$ ). There was no significant effect of predation on density when predation was aggregated into two groups (predation and no predation; main effect  $F_{(1,7)} = 1.21$ ,  $P = 0.31$ ; interaction effect  $F_{(13,91)} = 0.92$ ,  $P = 0.54$ ). The regression also failed to find a significant effect of predation on host density (CLS estimate: 0.03,  $P > 0.40$ ). However, this is not because fish were ineffective predators: there was a significant decrease in *D. dentifera* density with increasing predation (Spearman’s rho =  $-0.64$ ,  $P = 0.03$ ) early in the experiment before the parasite had a chance to affect *D. dentifera* dynamics.

Instead, it appears that the effect of parasitism on host density dominated the effect of predation. Host density increased exponentially until the first epidemic peak, at which point *D. dentifera* declined greatly (Fig. 2). After the second peak in infection prevalence, densities in most



**Fig. 2** Time-series of *D. dentifera* density. The five predation treatments are indicated with differently dashed lines, as in Fig. 1 (see legend); there are two replicates per predation treatment (except for 0.175 m<sup>-2</sup>, for which there is only one replicate). The arrows above the figure indicate peaks in infection prevalence (see Fig. 1). Data from all replicates are shown



**Fig. 3a–b** Time series of *D. dentifera* density (dotted lines, triangles, left axis) and **a** infection prevalence (solid line, circles, right axis) and **b** water clarity (Secchi depth; dashed line, squares, right axis). For clarity, the means ( $\pm 1$  SE) of the data from all nine enclosures are shown. The statistical analysis included the full time series from each of the nine enclosures

bags declined to  $<0.1 \text{ L}^{-1}$ , much lower than carrying capacity in this highly productive lake (Mittelbach et al. 2006; Threlkeld 1979). This negative effect of infection prevalence on changes in host density was significant (CLS estimate:  $-0.33$ ,  $P < 0.03$ ; Fig. 3a). Interestingly, *D. dentifera* density recovered in two of the nine enclosures.

The parasite-driven decline in *D. dentifera* density appears to have cascaded down to the phytoplankton level. There was a significant positive effect of *D. dentifera* density on changes in Secchi depth (Fig. 3b; CLS estimate:  $0.17$ ,  $P = 0.01$ ). Because Secchi depth was strongly negatively correlated with chlorophyll *a* ( $r = -0.67$ ,  $P < 0.001$ ), this indicates a negative effect of *D. dentifera* density on phytoplankton.

## Discussion

In this study, predation did not protect the host population from the effects of parasitism. Instead, there were large *Metschnikowia* epidemics in all enclosures, followed by large reductions in *Daphnia* density. These decreases in *Daphnia* density were mirrored by increased phytoplankton abundance and decreased water clarity. Together, these suggest a parasite-driven trophic cascade.

All of the enclosures had large epidemics, regardless of predation rate. This result is somewhat surprising, given that several observational studies on *Daphnia*-parasite systems suggest that fish predation can reduce parasitism in *Daphnia* (Duffy 2006; Duffy et al. 2005; Johnson et al. 2006). Why, then, wasn't there an effect of fish predation on parasitism in this study? It is not that fish were ineffective predators in this experiment—they had a significant effect on host density early in the experiment, before the parasite began driving host dynamics. Given this evidence for effective fish predation in this experiment, as well as significant and large effects of fish in other experiments with similar designs (i.e., bluegill in cages in bag enclosures; González and Tessier 1997), it seems unlikely that these results are simply an artifact of the experimental set-up. However, the possibility that the experimental setup altered bluegill foraging behavior (or selectivity) cannot be excluded.

There are at least two other possible explanations for the lack of an effect of predation on infection prevalence. One possible reason is that the parasite can survive predation, minimizing the effect of predation on infection prevalence. Indeed, I have found that about half of *Metschnikowia* spores survive fish predation (Duffy 2006). However, theoretical results suggest that this cannot explain the data—even with half the spores surviving predation, there should still be a strong negative correlation between predation and infection prevalence (M.A. Duffy, unpubl. data). A more likely explanation for the lack of an effect of predation on infection prevalence is very high transmission rates. Very high transmission rates can swamp a predator effect (Duffy et al. 2005; Hall et al. 2006), allowing the disease to persist in the system for all reasonable predation rates. Very high transmission rates seem likely in this case. Even though the spore concentration used to inoculate the enclosures was less than half that typically seen at the beginning of epidemics (1,900 spores/L vs. 5,000 spores/L; S.R. Hall, unpubl. data), the epidemics were as large as those typically seen in lake populations (Cáceres et al. 2006). Part of this increased transmission may be due to the extremely warm lake temperatures during this experiment (epilimnion temperatures were as high as  $28^\circ\text{C}$ ; M.A. Duffy, unpubl. data), since parasite transmission rate increases with temperature (Hall et al. 2006). However, predation rate should

also increase with temperature, which would increase selective culling by fish (Hall et al. 2006), so it is likely that factors other than temperature were also driving the high transmission rates. One of these factors may have been mixing associated with sampling, which likely increased contact between *Daphnia* and *Metschnikowia* spores.

This study suggests that there were parasite-driven trophic cascades in the enclosures. Because there is no parasite-free treatment, this finding is instead based on strong correlations between infection prevalence, *Daphnia* density and water clarity. Epidemics led to large decreases in *Daphnia* density—during the epidemic, density decreased to  $<0.1\text{ L}^{-1}$  in most enclosures; this is several orders of magnitude less than typical summer densities in this lake (Mittelbach et al. 2006; Threlkeld 1979). This, in turn, led to increased phytoplankton and decreased water clarity. Previously, I found indirect evidence of parasite-driven changes in phytoplankton in an observational study of *D. dentifera* and a bacterial parasite, *Spirobacillus cienkowski*, in three lakes (Duffy 2006). In that case, the fecundity of uninfected adults increased during *Spirobacillus* epidemics, which can indicate increases in food availability (Ghilarov 1985). Together, these results suggest that parasite-driven trophic cascades may play an important, yet overlooked, role in food web dynamics in lakes.

It is interesting that the densities recovered in two of the enclosures. We have found that *Daphnia dentifera* evolve rapidly in response to *Metschnikowia* epidemics and that this evolution can be important to ecological host–parasite dynamics (Duffy 2006; Duffy and Sivars-Becker 2007). *Daphnia* clones collected from the two enclosures in which density recovered lived significantly longer after being infected than *Daphnia* clones collected from Warner Lake (the source population for the enclosures; M.A. Duffy, unpubl. data). The possibility that this increase in tolerance may have helped these populations recover is intriguing and warrants further study.

Overall, this study suggests that parasites have the potential to drastically reduce host densities, even in the presence of selective predators. Thus, selective predation cannot necessarily be counted upon to “keep the herds healthy,” and caution should be used before using this healthy herds approach as a conservation strategy.

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